Handbook of Clinical Audiology

SEVENTH EDITION

Jack Katz
Marshall Chasin
Kristina English
Linda J. Hood
Kim L. Tillery

Wolters Kluwer

Thank you

for purchasing this e-book.

To receive special offers and news about our latest products, sign up below.

Or visit LWW.com

Wolters Kluwer

HANDBOOK OF
CLINICAL AUDIOLOGY

SEVENTH EDITION

www.cambodiamed.blogspot.com | Best Medical Books | Chy Yong | Credit STORM

HANDBOOK OF CLINICAL AUDIOLOGY

SEVENTH EDITION

EDITOR-IN-CHIEF

JACK KATZ, Ph.D.

Director
Auditory Processing Service
Prairie Village, Kansas
and Research Professor
University of Kansas Medical Center
Kansas City, Kansas and Professor Emeritus
University at Buffalo
State University of New York
Buffalo, New York

EDITORS

MARSHALL CHASIN, Au.D.

Director of Auditory Research
Musician's Clinics of Canada
Toronto, Ontario, Canada

KRISTINA ENGLISH, Ph.D.

Professor and Interim School Director
School of Speech Pathology and Audiology
University of Akron/NOAC
Akron, Ohio

LINDA J. HOOD, Ph.D.

Professor
Department of Hearing and Speech Sciences
Vanderbilt Bill Wilkerson Center
Vanderbilt University
Nashville, Tennessee, USA
Honorary Professor
University of Queensland
Brisbane, Australia

KIM L. TILLERY, Ph.D.

Professor and Chair
Department of Communication Disorders & Sciences
State University of New York at Fredonia
Fredonia, New York

Wolters Kluwer

Philadelphia • Baltimore • New York • London
Buenos Aires • Hong Kong • Sydney • Tokyo

Acquisitions Editor: Michael Nobel
Product Development Editor: Linda G. Francis
Marketing Manager: Leah Thomson
Editorial Assistant: Tish Rogers
Production Project Manager: Priscilla Crater
Design Coordinator: Stephen Druding
Illustration Coordinator: Jennifer Clements
Manufacturing Coordinator: Margie Orzech
Prepress Vendor: Aptara, Inc.

7th edition

Copyright © 2015 Wolters Kluwer Health

Copyright © 2009, 2001 Lippincott Williams & Wilkins. Copyright © 1993, 1985, 1978, 1972 Williams & Wilkins.
Two Commerce Square
2001 Market Street
Philadelphia, PA 19103 USA
LWW.com

Not authorised for sale in United States, Canada, Australia, New Zealand, Puerto Rico, or U.S. Virgin Islands.

All rights reserved. This book is protected by copyright. No part of this book may be reproduced or transmitted in any form or by any means, including as photocopies or scanned-in or other electronic copies, or utilized by any information storage and retrieval system without written permission from the copyright owner, except for brief quotations embodied in critical articles and reviews. Materials appearing in this book prepared by individuals as part of their official duties as U.S. government employees
are not covered by the above-mentioned copyright. To request permission, please contact Wolters Kluwer Health at Two Commerce Square, 2001 Market Street, Philadelphia, PA 19103, via email at permissions@lww.com, or via our website at lww.com (products and services).

9 8 7 6 5 4 3 2 1

Printed in China

Library of Congress Cataloging-in-Publication Data

Handbook of clinical audiology / editor-in-chief, Jack Katz ; editors, Marshall Chasin, Kristina English, Linda J. Hood, Kim L. Tillery. – Seventh edition.
p. ; cm.
Includes bibliographical references.
ISBN 978-1-4511-9163-9
I. Katz, Jack, editor. II. Chasin, Marshall, editor. III. English, Kristina M., 1951- editor. IV. Hood, Linda J., editor. V. Tillery, Kim L., editor.
[DNLM: 1. Hearing Disorders. 2. Hearing–physiology. WV 270]
RF291
617.8–dc23

2014014240

Care has been taken to confirm the accuracy of the information presented and to describe generally accepted practices. However, the authors, editors, and publisher are not responsible for errors or omissions or for any consequences from application of the information in this book and make no warranty, expressed or implied, with respect to the currency, completeness, or accuracy of the contents of the publication. Application of this information in a particular situation remains the professional responsibility of the practitioner; the clinical treatments described and recommended may not be considered absolute and universal recommendations.

The authors, editors, and publisher have exerted every effort to ensure that drug selection and dosage set forth in this text are in accordance with the current recommendations and practice at the time of publication. However, in view of ongoing research, changes in government regulations, and the constant flow of information relating to drug therapy and drug reactions, the reader is urged to check the package insert for each drug for any change in indications and dosage and for added warnings and precautions. This is particularly important when the recommended agent is a new or infrequently employed drug.

Some drugs and medical devices presented in this publication have Food and Drug Administration (FDA) clearance for limited use in restricted research settings. It is the responsibility of the health care provider to ascertain the FDA status of each drug or device planned for use in his or her clinical practice.

CONTRIBUTORS

DANIEL ARTHUR ABRAMS, Ph.D.
Research Associate, Department of Psychology and Behavioral Sciences
Stanford University
Palo Alto, California

ANGELA LOUCKS ALEXANDER, Au.D.
Director, Taupo Audiology and Auditory Processing Network
Taupo, New Zealand

EDOARDO ARSLAN, M.D.*
Department of Neuroscience, University of Padova, Padova, Italy
Service of Audiology and Phoniatrics, Treviso Regional Hospital, Piazza Ospedale,Treviso, Italy

A.U. BANKAITIS, Ph.D.
Vice President, Oaktree Products, Inc.
St. Louis, Missouri

JANE A. BARAN, Ph.D.
Professor and Chair
Department of Communication Disorders
University of Massachusetts Amherst
Amherst, Massachusetts

DOUGLAS L. BECK, Au.D.
Director of Professional Relations, Oticon, Inc.
Somerset, New Jersey
Web Content Editor, American Academy of Audiology
Reston, Virginia

LINDSAY BONDURANT, Ph.D.
Assistant Professor of Audiology, Communication Sciences and Disorders
Illinois State University
Normal, Illinois

CARMEN BREWER, Ph.D.
Chief Research Audiologist, Audiology Unit, Otolaryngology Branch
National Institute on Deafness and other Communication Disorders
National Institutes of Health
Bethesda, Maryland

ROBERT BURKARD, Ph.D.
Professor and Chair Department of Rehabilitation Science
University at Buffalo
State University of New York
Buffalo, New York

ANTHONY T. CACACE, Ph.D.
Professor
Communication Sciences and Disorders
Wayne State University
Detroit, Michigan

*Deceased

MARSHALL CHASIN, Au.D.
Director of Research
Musicians' Clinics of Canada
Toronto, Ontario, Canada

LAUREL A. CHRISTENSEN, Ph.D.
Chief Audiology Officer
Vice President, Research and Development
GN ReSound Group
Glenview, Illinois

JOHN GREER CLARK, Ph.D.
Associate Professor, Department of Communication Sciences and Disorders
University of Cincinnati
Cincinnati, Ohio
President, Clark Audiology, LLC
Middletown, Ohio

CHRISTOPHER GRAY CLINARD, Ph.D.
Assistant Professor
Department of Communication Science & Disorders
James Madison University
Harrisonburg, Virginia

CLAUDIA BARROS COELHO, M.D., Ph.D.
Research Scientist
Department of Otolaryngology
University of Iowa
Iowa City, Iowa

WILLIAM COLE, B.a.Sc., P.Eng.
President
Audioscan Division of Etymonic Design, Inc.
Dorchester and
Adjunct Associate Professor
School of Communication Science and Disorders
Western University
London, Ontario, Canada

BARBARA CONE, Ph.D.
Professor Speech, Language and Hearing Sciences
The University of Arizona
Tucson, Arizona

ALLAN O. DIEFENDORF, Ph.D.
Professor, Department of Otolaryngology, Head and Neck Surgery
Indiana University School of Medicine
Director, Audiology and Speech/Language Pathology
Indiana University Health
Indianapolis, Indiana

ANDREW DIMITRIJEVIC, Ph.D.
Assistant Professor
Communication Sciences Research Center
Cincinnati Children's Hospital
Department of Otolaryngology, University of Cincinnati
Cincinnati, Ohio

RACHEL N. DINGLE, Ph.D.
Student, School of Communication Sciences and Disorders
Western University
London, Ontario, Canada

MANUEL DON, Ph.D.
Head, Electrophysiology Department, Scientist III (retired) House Research Institute Los Angeles, California

M. PATRICK FEENEY, Ph.D.
Professor, Department of Otolaryngology, Head and Neck Surgery
Oregon Health and Science University
Director, Veterans Affairs National Center for Rehabilitative Auditory Research
Portland Veterans Affairs Medical Center
Portland, Oregon

JEANANE FERRE, Ph.D.
Adjunct Faculty
Communication Sciences & Disorders
Northwestern University
Evanston, and
Audiologist, Central Auditory Evaluation and Treatment
Oak Park, Illinois

TRACY S. FITZGERALD, Ph.D.
Staff Scientist/Director, Mouse Auditory Testing Core Facility
National Institute on Deafness and Other Communication Disorders
National Institutes of Health
Bethesda, Maryland

BRIAN J. FLIGOR, Sc.D.
Chief Audiology Officer
Lantos Technologies, Inc.
Wakefield, Massachusetts

RICHARD E. GANS, Ph.D.
Founder & CEO
The American Institute of Balance (ABI)
Largo, Florida

DOUGLAS B. GARRISON, Au.D.
Director, Duke Vestibular Lab
Department of Otolaryngology—Head and Neck Surgery
Duke University Health System
Durham, North Carolina

JENNIFER E. GONZALEZ, B.A.Au.D./Ph.D.
Candidate, Department of Speech, Language and Hearing Sciences
University of Connecticut
Storrs, Connecticut

JENNIFER GROTH, M.A.
Director, Audiology Communications Research and Developement
GN ReSound Group
Glenview, Illinois

SAMANTHA GUSTAFSON, Au.D.
Ph.D. Student, Department of Hearing and Speech Sciences
Vanderbilt University
Nashville, Tennessee

TROY HALE, Au.D.
Assistant Professor, Audiology
AT Still University
Director, AFA Balance and Hearing Institute
Mesa, Arizona

MELANIE HERZFELD, Au.D.
Practice Owner Hearing and Tinnitus Center
Woodbury, New York

THERESA HNATH-CHISOLM, Ph.D.
Professor and Chair, Communication Sciences and Disorders
University of South Florida
Tampa, Florida

LINDA J. HOOD, Ph.D.
Professor
Department of Hearing and Speech Sciences
Vanderbilt Bill Wilkerson Center
Vanderbilt University
Nashville, Tennessee, USA
Honorary Professor
University of Queensland
Brisbane, Australia

LISA L. HUNTER, Ph.D.
Associate Professor
Department of Otolaryngology and Communication Sciences and Disorders
University of Cincinnati
Scientific Director, Department of Audiology
Cincinnati Children's Hospital Medical Center
Cincinnati, Ohio

ANDREW B. JOHN, Ph.D.
Assistant Professor
Department of Communication Sciences and Disorders
College of Allied Health
University of Oklahoma Health Sciences Center
Oklahoma City, Oklahoma

ANDREW B. JOHN, Ph.D.
Assistant Professor, Communication Sciences and Disorders
University of Oklahoma Health Sciences Center
Oklahoma City, Oklahoma

CHERYL DeCONDE JOHNSON, Ed.D.
Private Consulting Practice
The ADVantage
Auditory-Deaf Education Consulting
Leadville, Colorado

HYUNG JIN JUN, M.D., Ph.D.
Department of Otolaryngology-Head and Neck Surgery
Guro Hospital, Korea University College of Medicine
Seoul, South Korea

JACK KATZ, Ph.D.
Director
Auditory Processing Service
Prairie Village, Kansas
and Research Professor
University of Kansas Medical Center
Kansas City, Kansas and Professor Emeritus
University at Buffalo
State University of New York
Buffalo, New York

WILLIAM JOSEPH KEITH, Ph.D.
Director, SoundSkills Auditory Processing Clinic
Auckland, New Zealand

PAUL KILENY, Ph.D.
Professor and Academic Program Director, Audiology
Otolaryngology, Head-and-Neck Surgery
University of Michigan
Ann Arbor, Michigan

KELLY KING, Ph.D.
Research Audiologist
Audiology Unit, Otolaryngology Branch National Institute on Deafness and Other Communication Disorders
National Institutes of Health
Bethesda, Maryland

NINA KRAUS, Ph.D.
Hugh Knowles Professor, Communication Sciences and Disorders
Northwestern University
Evanston, Illinois

BRIAN KREISMAN, M.D., Ph.D
Department of Speech Pathology and Audiology
Calvin College
Grand Rapids, Michigan

FREDERICK N. MARTIN, Ph.D.
Lillie Hage Jamail Centennial Professor Emeritus
Department of Communication Sciences and Disorders
The University of Texas at Austin
Austin, Texas

RACHEL McARDLE, Ph.D.
Associate Professor, Communication Sciences and Disorders
University of South Florida
Tampa, Florida
Chief, Audiology and Speech Pathology
Bay Pines Veterans Affairs Healthcare System
Bay Pines, Florida

JOSEPH J. MONTANO, Ed.D.
Associate Professor of Audiology
Department of Otolaryngology
Weill Cornell Medical College
New York, New York

FRANK E. MUSIEK, Ph.D.
Professor, Speech Language and Hearing Sciences
University of Connecticut
Storrs, Connecticut

RICK NEITZEL, Ph.D.
Assistant Professor, Department of Environmental Health Sciences
University of Michigan
Ann Arbor, Michigan

PEGGY NELSON, Ph.D.
Professor, Department of Speech-Language-Hearing Sciences
University of Minnesota
Minneapolis, Minnesota

WILLIAM NOBLE, Ph.D.
Psychology
School of Behavioural, Cognitive and Social Sciences
University of New England
Armidale, Australia

TABITHA PARENT-BUCK, Au.D.
Chair, Audiology Department
AT Still University
Audiologist, AFA Balance and Hearing Institute
Mesa, Arizona

DENNIS P. PHILLIPS, Ph.D.
Professor, Department of Psychology and Neuroscience
Dalhousie University
Halifax, Nova Scotia, Canada

ERIN G. PIKER, Ph.D.
Assistant Professor, Department of Surgery-Division of Otolaryngology
Duke University
Durham, North Carolina

BETH A. PRIEVE, Ph.D.
Professor, Communication Sciences and Disorders
Syracuse University
Syracuse, New York

EVELING ROJAS RONCANCIO, M.D.
Department of Otolaryngology
University of Iowa
Iowa City, Iowa

CHRIS SANFORD, Ph.D.
Assistant Professor
Communication Sciences and Disorders
Idaho State University
Pocatello, Idaho

ROSAMARIA SANTARELLI, Ph.D., M.D.
Department of Neuroscience
University of Padova
Padova, and
Deputy Director of Audiology and Phoniatrics
Treviso Regional Hospital
Treviso, Italy

KIM SUZETTE SCHAIRER, Ph.D.
Adjunct Faculty, Department of Audiology and Speech-Language Pathology
East Tennessee State University
Johnson City
Audiologist, Department of Audiology
James H. Quillen Veterans Affairs Medical Center
Mountain Home, Tennessee

ROBERT S. SCHLAUCH, Ph.D.
Professor
Department of Speech-Language-Hearing Sciences
University of Minnesota
Minneapolis, MN

SUSAN SCOLLIE, Ph.D.
Associate Professor School of Communication Sciences and Disorders
Western University,
London, Ontario, Canada

JOSEPH SMALDINO, Ph.D.
Professor, Communication Sciences and Disorders
Illinois State University
Normal, Illinois

JENNIFER L. SMART, Ph.D.
Associate Professor, Audiology, Speech-Language Pathology and Deaf Studies
Towson University
Towson, Maryland

CARRIE SPANGLER, Au.D.
Educational Audiology Clinician
School of Speech Language Pathology and Audiology
The University of Akron
Akron, Ohio

JAMES R. STEIGER, Ph.D.
Professor
School of Speech-Language Pathology and Audiology
The University of Akron; Northeast Ohio AuD Consortium
Aakron, Ohio

DE WET SWANEPOEL, Ph.D.
Professor, Speech-Language Pathology and Audiology
University of Pretoria
Pretoria, South Africa
Adjunct Professor, Ear Sciences Centre, School of Surgery
The University of Western Australia
Perth, Australia

ANNE MARIE THARPE, Ph.D.
Professor and Chair, Department of Hearing and Speech Sciences
Vanderbilt University
Nashville, Tennessee

KIM L. TILLERY, Ph.D.
Professor and Chair
Department of Communication Disorders & Sciences
State University of New York at Fredonia
Fredonia, New York

HENRY P. TRAHAN, Au.D.
Assistant Professor, Audiology
AT Still University
Mesa, Arizona

KELLY TREMBLAY, Ph.D.
Professor, Speech and Hearing Sciences
University of Washington

RICHARD S. TYLER, Ph.D.
Professor, Department of Otolaryngology
University of Iowa
Iowa City, Iowa

KRISTIN M. UHLER, Ph.D.
Assistant Professor
School of Medicine
Department of Otolaryngology
University of Colorado Denver

MICHAEL VALENTE, Ph.D.
Director of Adult Audiology
Department of Otolaryngology
Washington University School of Medicine
St. Louis, Missouri

MAUREEN VALENTE, Ph.D.
Director of Audiology Studies
Program in Audiology and Communication Sciences
Associate Professor
Department of Otolaryngology
Washington University School of Medicine
St. Louis, Missouri

BARBARA E. WEINSTEIN, Ph.D.
Professor and Founding Executive Officer
Health Sciences Doctoral Programs, Au.D. Program
Graduate Center, City University of New York
New York, New York

KARL R. WHITE, Ph.D.
Director, National Center for Hearing Assessment and Management
Emma Eccles Jones Endowed Chair in Early Childhood Education
Professor of Psychology
Utah State University
Logan, Utah

LAURA ANN WILBER, Ph.D.
Professor Emeritus, Communication Sciences and Disorders
Northwestern University
Evanston, Illinois

WILLIAM S. YACULLO, Ph.D.
Professor, Communication Disorders
Governors State University
University Park, Illinois

CHRISTINE YOSHINAGA-ITANO, Ph.D.
Professor, Department of Speech, Language and Hearing Sciences
University of Colorado, Boulder
Boulder, Colorado

TERESA A. ZWOLAN, Ph.D.
Professor, Otolaryngology
University of Michigan
Ann Arbor, Michigan

DEDICATION

The Seventh Edition of the Handbook of Clinical Audiology is Dedicated to Raymond Carhart (192?–1975), who is recognized as the "Father of Audiology." He talked about the shared responsibility of the clinician and the hearing scientists (both of whom he considered as audiologists) to learn about the hearing process and develop ways to help persons living with hearing loss.

FOREWORD

It is fitting that this book is dedicated to Dr. Raymond Carhart. He stated in a 1976 interview that he conceived of an audiologist "as someone who has a prime commitment to learning about hearing and its processes as well as a commitment to understanding and coping with its problems." He talked about the shared responsibility of the clinician and the hearing scientists (both of whom he considered as audiologists) to learn about the hearing process and ways to help the persons with hearing impairment. The seventh edition of *Handbook of Clinical Audiology* book strives to do that, as have the previous editions.

Carhart has been referred to as the "Father of Audiology"—or sometimes the "Grandfather of Audiology." Perhaps it would be most appropriate to call him the "Grand Father of Audiology."

Although he came to the field somewhat indirectly, his contributions were enormous.

Dr. Carhart was born in Mexico City. He received his Bachelor's degree from Dakota Wesleyan University in 1932 in speech pathology and psychology; his Master's and Ph.D. degrees from Northwestern in 1934 and 1936, respectively, in Speech Pathology, Experimental Phonetics, and Psychology. He was an instructor in speech reeducation at Northwestern from 1936 to 1940 and then an assistant, and associate professor in 1943 in speech science.

Although Carhart initially worked in speech science, he was asked to replace C.C. Bunch following Bunch's untimely death in June, 1942. Carhart then began to teach Bunch's courses in hearing and became so interested in the problems that, as he said, "I've been working with them ever since."

In 1943, Carhart joined the Medical Administrative Corps, U S Army, as a captain, he was assigned to DeShon Hospital in Butler, Pennsylvania as Director of the Acoustic Clinic and as Acoustic Physicist where he was asked to develop a program for veterans who had lost their hearing during the war. In that capacity he contacted the scientists at the Psycho-Acoustic Laboratory (PAL) at Harvard, who, among other things, had come up with word lists that might be used in evaluating a person's ability to understand speech. He also developed a fairly comprehensive rehabilitation program that involved selecting and fitting hearing aids (which were not so complex or elaborate as they are today), and teaching the soldiers and veterans how to use them.

When Carhart returned to Northwestern in 1946, he convinced the dean to establish an academic program in Audiology, which was the name that Dr. Norton Canfield chose for the department at DeShon. He became the first professor of Audiology at Northwestern.

Carhart later said (in class if not in print) that sound—and especially speech—was comprised of many dimensions. Four of those dimensions were (1) sensitivity (how faintly can one hear); (2) clarity of sound (speech or otherwise) in quiet; (3) clarity in noise; and (4) tolerance (how loud can sound be without becoming a problem). Two of these dimensions (sensitivity and clarity), which were detailed by Carhart in a 1951 paper, became the basis of Plomp's (1978) two-component model of hearing loss involving audibility and distortion. Carhart said there were many more dimensions to hearing, but those four should always be measured when fitting hearing aids and working with patients with hearing loss.

Although Carhart worked as a speech scientist, a clinician (in speech and in hearing), a researcher in speech and especially in hearing, his primary contribution is probably as a teacher and educator. An educator can be described as one who conveys learning in which the knowledge, skills, and habits of a group of people are transferred from one generation to the next through teaching, training, or research, and that certainly describes Dr. Carhart.

In his capacity as an educator, Carhart directed some 35 dissertations, beginning in 1946 with a study by John Keys entitled "Comparative Threshold Acuity of Monaural and Binaural Hearing for Pure Tone and Speech as Exhibited by Normal and Hard of Hearing." Although his primary interest was in speech and speech understanding, the dissertations he directed covered a range of hearing problems from difference limens, to effects of surgery and specific diseases on hearing, to auditory fatigue, loudness and many more topic areas. In addition, as an educator he taught some of the leaders in the field of audiology like James Jerger, Donald Dirks, Cornelius Goetzinger, Jack Willeford, and many more. Many of those went on to teach, and to educate other students in audiology programs at our most prestigious universities.

In 1949, he directed the dissertation of Miriam Pauls Hardy, who may have been the first female to graduate with a Ph.D. in audiology. Unlike some of the professors of the time, Dr. Carhart did not discriminate on the basis of gender. He believed that it was the mind—not the gender—that was important. He did, however, believe that one should do the work, not just talk the talk. He set an example in that one often found him in his office or laboratory in the evening and on weekends.

His early research interests at Northwestern were in conductive hearing loss (the "Carhart notch," which can be an indicator of possible otosclerosis was named for him), including a method of checking the accuracy of bone conduction measurements before there was an artificial mastoid, let alone an ANSI standard. He was interested in masking (forward, backward, and perceptual, which we now call informational

masking), and did much to enhance our understanding of the way speech is processed by the damaged ear.

Before there were computers in our clinics and most academic research laboratories, he developed a key-sort system as a way of classifying audiograms so that one could better interpret the puretone audiogram.

Finally, Carhart believed that audiology was more than a clinical field and that the clinicians who practiced it should continue to explore and research the ways in which we hear and how to improve the lives of those who do not hear normally.

Raymond Carhart died at his desk in October 1975, leaving behind a legacy to the academic discipline of audiology, the numerous leaders in the field of audiology whom he had educated, and the greater understanding of audiologic assessment and hearing aid rehabilitation for which he is known as the father—or Grand Father of Audiology.

We dedicate this seventh edition to Dr. Carhart, because like him the *Handbook of Clinical Audiology* has educated so many leaders of our field and has inspired countless audiologists throughout the world. This edition of the Handbook will provide a broad perspective of the field of audiology by nearly 100 contributing experts in the field. They offer their knowledge, wisdom, and enthusiasm to help another generation of audiologists to fulfill their mission.

REFERENCES

Carhart R. (1951). Basic principles of speech audiometry. *Acta Otolaryngol.* 40:62–71.

Plomp R. (1978). Auditory handicap of hearing impairment and the limited benefit of hearing aids. *J Acoust Soc Am.* 63:533–549.

Laura Ann Wilber

PREFACE

For more than 40 years, the *Handbook of Clinical Audiology* (HOCA) has maintained an important role in the education of graduate students in audiology, both in North America and throughout the world. It also serves as a useful reference for audiologists, otologists, and speech–language pathologists who wish to have a comprehensive and practical guide to the current practices in audiology.

Each edition of the HOCA has been an update of the previous one, but we have also striven to make the newest edition better than the one that came before. For this edition, there are four highly skilled and knowledgeable editors plus one senior editor. We have worked together to select highly qualified contributors on topics that are both core and current for students and professionals in audiology. Online case studies and references have been added to this edition to enable the reader to go beyond the basic scope of this book.

THE FOREWORDS

In the previous edition of the Handbook the foreword was written by Moe Bergman, a distinguished gentleman with many years of audiology behind him. Moe Bergman, Ed.D., was in the very first group of audiologists who began this discipline more than 70 years ago. Starting prior to World War II and for decades following, Dr. Bergman was a clinician, administrator, professor, researcher, and writer, and after he retired from Hunter College in New York City, he went to Israel to establish audiology as a profession there. He is considered as the Father of Audiology in Israel. For many years, Dr. Bergman has continued to be active as an advisor and an officer in international professional organizations. His clarity about the events and developments so many years ago (see Bergman, 2002) makes him a treasured link to our roots.

This edition is dedicated to Raymond Carhart, "The Father of Audiology." We are delighted to have the book's dedication and foreword discussing Dr. Carhart; written by Laura Ann Wilber a former student of his and a distinguished audiologist in her own right. Dr. Carhart was her dissertation advisor and she worked with him and Tom Tillman to develop what later became the NU-6 word recognition test. When Laura Wilber earned her Ph.D. there were few women who were educated at that level in audiology and many people felt that it was a male profession. So Dr. Carhart's acceptance of her and clearing a path for her was especially important.

It is worth noting that Laura Wilber has contributed to each edition of the Handbook since the first edition in 1972. She herself was honored by the editors of the HOCA in the previous edition by dedicating the book to her and to three other audiologists.

SECTIONS, CHAPTERS, AND CONTRIBUTORS

The strength of HOCA has always been the knowledge and expertise of the contributors in the many aspects of audiology. They have both clinical and research credentials in the topics they write about and most are also professors who are proficient in communicating with students. Audiologists looking down the list of contributors will recognize familiar and highly respected colleagues. They have contributed much to the field in the past and now contribute again by providing important and readable materials for both colleagues and students. We have made every effort to provide up-to-date, accurate, and clinically applicable information.

Each of the four main editors of this book has a distinguished record of teaching, research, writing, and clinical work. Each one took responsibility for significant portions of the book. Linda Hood helped to edit the Sixth Edition and decided to go "another round." Her chapters deal primarily with physiological methods for audiologic diagnosis. Marshall Chasin, our first Canadian editor, edited the chapters dealing with amplification and other technical aspects. Kim Tillery edited the chapters dealing with central auditory processing disorders and shared in the final editing of all chapters. Kristina English edited the basic chapters and those dealing with re/habilitation. Jack Katz reviewed all of the chapters and saw to the overall manuscript issues.

The Handbook is divided into four sections. There are eight chapters dealing with Introduction, Basic Tests, and Principles. A chapter that summarizes diagnostic audiology and brings together the various contributions has been added in this edition. Other top-notch audiologists wrote on Puretone Air Conduction, Bone Conduction, and Speech Audiometry, as well as Masking and Case History.

The second section is made up of 14 chapters dealing with Physiologic Principles and Measures. This section of the book includes auditory measures starting with the conductive mechanism up to the brain and vestibular measures that assess from the inner ear to the brain. Some chapters include specialty areas such as intraoperative monitoring and therapy for vestibular disorders. Some of the most pronounced advances in recent years have been made in these areas.

The third section is devoted to a wide variety of Special Populations. It contains 14 chapters beginning with Newborn Hearing Screening, Assessment of Hearing Loss in Children and Educational Audiology and ends with Hearing Loss in the Elderly, Tinnitus/Hyperacusis, and Tele-Audiology. Four chapters deal with Central Auditory Processing Disorders and Central Auditory Functions.

The final section, Management of Hearing Disorders, is made up of 10 chapters. Five of the chapters deal with hearing aids and cochlear implants, two focus on management, and two more are on Room Acoustics and Assistive Technologies as well as Building a Successful Audiologic Practice. In addition, for the first time we will have a chapter dealing with infection control in audiology practice that was written by A.U. Bankaitis. This important topic relates to all aspects of audiology that deal with clinical patients and/or research subjects.

Sadly, during the writing of the chapter on electrocochleography with Rosamaria Santarelli, contributor Edoardo Arslan passed away. He was both her co-author and mentor.

NEW FEATURES

Six new chapters have been introduced in the seventh edition. They are Diagnostic Audiology, The Dizzy Patient and Vestibular Rehabilitation, Newborn Hearing Screening, Genetic Hearing Loss, Tele-Audiology, and Infection Control. At the close of every chapter is a new section called Food for Thought, which encourages readers to interact more deeply with the text.

thePoint

In this edition of this Handbook we have added supplemental materials (e.g., extended references and case studies) on thePoint companion website at http://thepoint.lww.com/Katz7e.

TERMINOLOGY

The following is an explanation of some of the spelling conventions used in the HOCA and briefly why we chose them. Further discussion may be found in Chapter 1.

Compound Words

In clinical audiology, as well as in English generally, compound words (two words written as one) are common. Compound words are simplifications of words that are frequently used together. For example, *brain* and *stem* are combined in the term *auditory brainstem response.* When two words are frequently used together to express a certain meaning, in time, they may be connected by a hyphen and eventually joined together into a single word (base ball, base-ball, baseball).

PURETONE

The terms *pure tone* and *pure-tone* are constantly used in audiology with or without a hyphen. This has encouraged us to combine them into a compound word, *puretone.* By choosing a single word it eliminates the inconstancies that we see when they are used or misused with or without a hyphen.

SENSORY/NEURAL

On the one hand, while there is good reason to use *puretone* as a compound word, on the other hand, it would be beneficial for the term *sensorineural* to be separated into sensory or neural using a slash as we often use for "or" (i.e., *sensory/neural*). This makes it clear that the test or result it did not distinguish sensory from neural. From the term sensorineural it is often not clear what is intended as many professionals assume that sensorineural means sensory. This problem has led to important confusions and errors that can be easily remedied by the use of sensory/neural if it is unclear which is indicated (e.g., with no air-bone gap for puretone thresholds we do not know if it is sensory, neural, or both). If the specific region is identified (e.g., present Otoacoustic Emissions but absent Middle Latency Response) we indicate that it was specifically neural or retrocochlear. If it is both we state "both sensory and neural" or just "sensory and neural."

EPILOGUE

We are pleased that the *Handbook of Clinical Audiology* (HOCA) is used widely by audiologists around the world. Interestingly when the HOCA first came out in 1972, we were living in the Turkish Republic. There the word *hoca* means a religious leader or a revered teacher. While HOCA is certainly not a religious leader, we do hope it will be a revered teacher for the many students and colleagues that read this book.

ACKNOWLEDGMENTS

We would like to thank the editors of Wolters Kluwer, especially Linda Francis and Meredith Brittain, not only for their fine editing of this book but also for advising us and keeping us to our deadlines. We would like to mention the following colleagues and other individuals who also helped us in many ways to make the Handbook as high quality a text and reference book that we could. They are Mark Chertoff, Robin Gashler, Jay Hall, Amy Lane, Larry Medwetsky, Marcello Peppi, Lynden Ronsh, and Natalie Turek. In addition, I appreciate the tremendous support from my family and would like to highlight those who made special contributions to the completion of this edition. They are Eric Kaseff, Lainie Kaseff, Mark Katz, and Miriam Kaseff. Another member of my family deserves very special mention. My wife Irma Laufer Katz has been heavily involved as reader, advisor, and organizer of most of my projects over the past 58 years. For the Handbook she was also the secretary who kept track of the 46 chapters as they came and went from authors to editors and back again in the various stages of completion. We must certainly mention the many authors who contributed their knowledge and skills to make this Handbook an important contribution to the education and practice of those in the field of Audiology. To all of them and, of course, to my fellow editors my thanks and gratitude.

CONTENTS

Contributors v
Dedication ix
Foreword xi
Preface xiii

SECTION I:
BASIC TESTS AND PROCEDURES 1

1 **A Brief Introduction to Clinical Audiology and This Handbook . . . 3**
Jack Katz

2 **Calibration . . . 9**
Laura Ann Wilber and Robert Burkard

3 **Puretone Evaluation . . . 29**
Robert S. Schlauch and Peggy Nelson

4 **Bone Conduction Evaluation . . . 49**
James R. Steiger

5 **Speech Audiometry . . . 61**
Rachel McArdle and Theresa Hnath-Chisolm

6 **Clinical Masking . . . 77**
William S. Yacullo

7 **Case History . . . 113**
Douglas L. Beck

8 **Diagnostic Audiology . . . 119**
Brian M. Kreisman, Jennifer L. Smart, and Andrew B. John

SECTION II:
PHYSIOLOGICAL PRINCIPLES AND MEASURES 135

9 **Tympanometry and Wideband Acoustic Immittance . . . 137**
Lisa L. Hunter and Chris A. Sanford

10 **Acoustic Stapedius Reflex Measurements . . . 165**
M. Patrick Feeney and Kim S. Schairer

11 **Introduction to Auditory Evoked Potentials . . . 187**
Robert Burkard and Manuel Don

12 **Electrocochleography . . . 207**
Rosamaria Santarelli and Edoardo Arslan

13 **Auditory Brainstem Response: Differential Diagnosis . . . 231**
Frank E. Musiek, Jennifer E. Gonzalez, and Jane A. Baran

14 **Auditory Brainstem Response: Estimation of Hearing Sensitivity . . . 249**
Linda J. Hood

15 **Auditory Steady-State Response . . . 267**
Andrew Dimitrijevic and Barbara Cone

16 **Intraoperative Neurophysiological Monitoring . . . 295**
Paul R. Kileny and Bruce M. Edwards

17 **Middle-Latency Auditory-Evoked Potentials . . . 315**
Anthony T. Cacace and Dennis J. McFarland

18 **Cortical Auditory-Evoked Potentials . . . 337**
Kelly Tremblay and Christopher Clinard

19 **Otoacoustic Emissions . . . 357**
Beth Prieve and Tracy Fitzgerald

20 **Clinical Neurophysiology of the Vestibular System . . . 381**
Erin G. Piker and Douglas B. Garrison

21 **Evaluation of the Patient with Dizziness and Balance Disorders . . . 399**
Troy Hale, Henry Trahan, and Tabitha Parent-Buck

22 **Vestibular Rehabilitative Therapy . . . 425**
Richard Gans

SECTION III:
SPECIAL POPULATIONS 435

23 **Newborn Hearing Screening . . . 437**
Karl R. White

24 **Assessment of Hearing Loss in Children . . . 459**
Allan O. Diefendorf

25 **Genetic Hearing Loss . . . 477**
Carmen Brewer and Kelly King

26 **Educational Audiology . . . 501**
Cheryl DeConde Johnson and Carrie Spangler

27 **Central Auditory Processing: A Functional Perspective from Neuroscience . . . 513**
Dennis P. Phillips and Rachel N. Dingle

28 **Auditory Pathway Representations of Speech Sounds in Humans . . . 527**
Daniel A. Abrams and Nina Kraus

29 Central Auditory Processing Evaluation: A Test Battery Approach 545
Kim L. Tillery

30 Central Auditory Processing Disorder: Therapy and Management 561
Jack Katz, Jeanane Ferre, William Keith, and Angela Loucks Alexander

31 Individuals with Multiple Disabilities 583
Anne Marie Tharpe and Samantha Gustafson

32 Noise Exposure 595
Brian Fligor, Marshall Chasin, and Rick Neitzel

33 Nonorganic Hearing Loss 617
Frederick N. Martin and John Greer Clark

34 Hearing Loss in the Elderly: A New Look at an Old Problem 631
Barbara E. Weinstein

35 Tinnitus and Hyperacusis 647
Richard S. Tyler, William Noble, Claudia Coelho, Eveling Rojas Roncancio, and Hyung Jin Jun

36 Tele-audiology 659
De Wet Swanepoel

SECTION IV:
MANAGEMENT OF HEARING DISORDERS 673

37 Room Acoustics and Auditory Rehabilitation Technology 675
Joseph Smaldino, Brian Kreisman, Andrew John, and Lindsay Bondurant

38 Hearing Aid Technology 703
Jennifer Groth and Laurel A. Christensen

39 Troubleshooting and Testing Hearing Aids 727
William Cole and Marshall Chasin

40 Hearing Aid Fitting for Children: Selection, Fitting, Verification, and Validation 759
Susan Scollie

41 Hearing Aid Fitting for Adults: Selection, Fitting, Verification, and Validation 777
Michael Valente and Maureen Valente

42 Building and Growing an Audiologic Practice 805
Melanie Herzfeld

43 Implantable Hearing Devices 817
Teresa A. Zwolan

44 Intervention, Education, and Therapy for Children with Hearing Loss 835
Christine Yoshinaga-Itano and Kristin M. Uhler

45 Audiologic Rehabilitation 849
Joseph Montano

46 Infection Control 861
A.U. Bankaitis

SECTION V:
APPENDICES 869

Author Index 895

Subject Index 907

SECTION I

Basic Tests and Procedures

CHAPTER 1

A Brief Introduction to Clinical Audiology and This Handbook

Jack Katz

Audiology is the study of hearing and hearing disorders, a field devoted to helping those with auditory and vestibular dysfunctions. This work may involve evaluation, re/habilitation, counseling, education, research, and/or screening/prevention.

Audiology combines aspects of science and art with techniques that are based on both basic and clinical research. We use sophisticated equipment to provide precision in determining the type and extent of the problems. But audiology is also an art. It involves the ability to perform the various tasks precisely and to provide information and maximum support to the individuals affected and their families. Because of these intellectually and emotionally gratifying aspects, it makes audiology an exciting career.

In my more than 50 years in this field, audiology has continued to be interesting and rewarding work. It is a comparatively new field that emerged in the aftermath of World War II (WWII) to aid service members who suffered hearing impairments. It brought together speech-language pathologists, deaf educators, psychologists, and ear, nose, and throat (ENT) physicians. This interdisciplinary cooperation was responsible for the excellent services that were provided to the injured military personnel. At the same time these multidisciplinary activities helped to lay the groundwork for the field of audiology. Indeed this interdisciplinary aspect of the field of audiology remains one of its great strengths even today. Initially, audiologic work was carried out in military hospitals and then spread to universities and university clinics, afterward to hospitals and community clinics.

Presently there are about 12,000 members of the American Academy of Audiology and approximately 2,000 members of the International Society of Audiology. Also memberships continue to grow in local, state, and national associations around the world. Audiology has several peer-reviewed journals and other publications, both printed and digital, that report on research and clinical developments. The field of audiology is constantly expanding its horizons and developing deeper understandings of both normal and abnormal processes.

AUDIOLOGY FROM 1940s TO TODAY

As mentioned above, the field of audiology was founded during WWII. Prior to that time hearing testing was carried out using tuning forks and whispered speech by medical doctors, although some puretone audiometers that provided repeatable stimuli were also in use. The combined efforts of the different disciplines fostered the variety of procedures we have to address the problems caused by hearing impairment. Bone-conduction testing and speech audiometry were soon added to the clinical tools. Aspects such as lip reading/speech reading, auditory training, and counseling were borrowed from deaf education, psychology, and speech-language pathology. An important adjunct for the service members was the fitting of hearing aids which were quite limited by today's standards. Nevertheless for years after the war these veterans were still using and benefiting from the amplification and training that they had received from those early audiologists when the profession was in its infancy.

After leaving military service, the early audiologists began to train others at colleges and universities. Audiologists began to research the clinical problems that they faced and many of these approaches and solutions are still in use today. These procedures also led the way to important innovations. Because it was clear that we did not have enough diagnostic information to accurately measure and categorize hearing disorders, early on, there was a heavy emphasis on developing new diagnostic procedures. For a number of years the area of diagnosis was the primary focus in audiologic research and practice.

When audiologists began dispensing hearing aids, this caused an expansion of attention, from just evaluation and identification of hearing loss to include providing means of managing hearing difficulties and therapy to address the communication problems. Hearing aid fitting was also a major impetus for audiologists to go into private practice. At the same time there were major breakthroughs in physiological measurements. This began with what we now refer to as cortical responses, but after a few years, earlier responses were identified from the auditory nerve and even

the cochlea. The field of audiology has expanded to include the assessment of more complex functions at all levels of the peripheral and central auditory nervous system. Immittance measurements enabled audiologists to assess mechanical properties of the auditory system of the outer and middle ears, as well as middle-ear muscle responses that rely on auditory nerve and brainstem activity. Specialties such as auditory processing disorders, educational audiology, vestibular function, and interoperative monitoring have added to the breadth and depth of the field.

The growing sophistication and understanding of auditory functions and development can be seen in the lowering of the target ages for various services. In the mid-1950s it was taught that we should wait until deaf children are perhaps 10 years of age before testing them and presumably provide amplification after that. Given our current state of knowledge, in retrospect, this seems absurd and counterproductive. At that time we did not understand that developmental problems should be identified at the earliest possible time. Otherwise, the person could miss critical periods and lose plasticity, as well as fall further behind with reduced learning and more acquired misconceptions. Now, neonatal hearing screening is widespread and we strive to begin habilitation by 6 months of age. In fact, in the past, one audiologist was ridiculed when she advocated that audiologists fit hearing aids for children as young as 1 year of age. Once we realized the critical importance of the early years for later development, early identification and assessment procedures, as well as training procedures were targeted and developed.

As the field of audiology expanded so did the academic demands on the practitioners. Initially, a bachelor's degree was required to practice and then a master's degree was the entry level along with basic clinical certification. As in the past a Ph.D. was generally desired for university teaching and research. In more recent years (in the United States) the Doctorate of Audiology (Au.D.) degree was introduced to provide even broader clinical teaching and training experiences. Also, higher levels of competency and certification are generally required today to practice audiology. Students interested in a career that includes independent research continue to pursue a Ph.D. in audiology, hearing science, or related areas. Now many of the top university programs in audiology have both Ph.D. and Au.D. trained professors to provide the student the best of both worlds. We also see combined Au.D./Ph.D. programs that offer students excellent ground for both clinical and research endeavors.

We owe a debt of gratitude to those early audiologists who helped to form this vibrant and vital health profession. Although we cannot mention the many important contributors, it is perhaps appropriate to mention Raymond Carhart (1912 to 1975) who is generally recognized as "The Father of Audiology." He was an important contributor to the developing field of audiology and an excellent teacher. Many of his students from Northwestern University in Evanston, Illinois went on to contribute significantly to the field of audiology in their own right.

ABOUT THIS HANDBOOK

The first edition of the *Handbook of Clinical Audiology* was published in 1972 and subsequent editions have served several generations of audiologists in the United States and increasingly throughout the world. It is used widely as both a text and reference book by students and professionals in various fields.

Currently, for this edition, we have five editors who have diverse areas of specialization in clinical, research, and teaching aspects for which they are responsible. To broaden our horizons and to be as inclusive as possible, in this edition we have tried to include more international voices and procedures.

We have squeezed in as much information in 1,000 pages as we could. The more than 90 contributors are highly regarded audiologists who also have clinical, research, and teaching experience. This makes the chapters authoritative, well organized, and geared for sharing our knowledge in a field that we love. We have always considered readability an important feature of this book and especially now that it is used by many people whose first language is not English.

The 46 chapters are divided into four sections. Section I deals with basic tests and procedures that are used by most audiologists for most of the people with whom they work. This involves puretone air and bone conduction, as well as standard speech audiometry. Calibration and case history chapters are also important components for any audiologic assessment. The Diagnostic Audiology chapter helps the reader to combine all of the previous information into a coherent diagnosis.

Section II introduces the various physiological and electrophysiological procedures used by audiologists at this time. These include immittance measures that primarily reveal the status of the middle ear. Electrocochleography and Otoacoustic Emissions provide detailed information about the responses from the cochlea, the end organ of hearing. Five chapters in this section discuss the electrophysiological responses from the auditory nerve, brainstem, and various areas of the brain. The chapter on intraoperative monitoring describes the analysis of the auditory system during surgery that informs the surgeons about the status and possible adverse effects of their manipulations of the auditory system. The final three chapters in this section deal with the vestibular system. They begin with the study of vestibular neurophysiology and end with vestibular rehabilitation.

Section III is called Special Populations. This recognizes that certain groups often require modifications in audiometric procedures or accommodations. Evaluation of young children offers a special challenge to the audiologist because they do not have the auditory or cognitive development needed for some of the tests, and it is sometimes

difficult to have them perform in the expected fashion. This chapter describes ways to obtain the desired results. Hearing, screening, and educational audiology generally involve the work carried out in schools with those who have normal hearing as well as those with auditory impairments. This section also includes chapters that deal with those who have multiple disabilities, hereditary hearing loss, and the elderly. Other special groups are those with noise-induced hearing loss, those who have tinnitus, and individuals who have "nonorganic" hearing loss. Four of the other chapters involve auditory processing disorders, which include the bases of central auditory problems, diagnostic procedures, and subsequent remediation. This section concludes with a chapter on tele-practice in which audiologists can work with people at far distances via communication systems.

Section IV deals with the management of hearing disorders. It begins with acoustical environments and technologies that are used to aid the hard-of-hearing person in reducing the influence of noise and other factors that can compromise communication. This is followed by four chapters related to various aspects of hearing aids and hearing aid fittings. Another chapter, which deals with implantable hearing devices, is a rapidly expanding area. The chapter discusses cochlear implants and other devices that are surgically imbedded into the person with a hearing loss. Two other chapters deal with management of those with hearing problems in the classroom and with rehabilitation of adults. There is also a chapter in this section that advises audiologists on how to start an effective audiologic practice.

New Chapters in This Edition

a. *Diagnostic Audiology* serves an important purpose in bringing together the information from the basic evaluation procedures in this book to form an audiologic interpretation and an understanding of the patient's needs. This chapter will also discuss some procedures that are not covered in the preceding chapters, as well as mentioning what our tests do not tell us.
b. *The Dizzy Patient and Vestibular Rehabilitation* chapter is an extension of a former Handbook chapter, applying diagnostic information to enable appropriate treatment decisions for patients with vestibular problems. It will describe audiologic procedures designed to relieve patient's symptoms, as well as the role of physical therapy and the necessity of collaboration among healthcare professionals.
c. *Hearing Screening* discusses newborn hearing screenings, school screenings, and other screening procedures using universal approaches and targeted population approaches. The specific procedures, their value, and outcomes of screening programs will be discussed.
d. *Hereditary Hearing Loss* describes much-needed information for audiologists related to genetic aspects of hearing loss that may be nonsyndromic or part of a known syndrome. Since audiologists are often the first professionals to suspect a genetic basis for a hearing loss, it is important to have current information available as well as the knowledge of resources.
e. *Audiology Tele-practice* follows the global trend to provide appropriate services at a distance from the professional. Using a range of communication technologies and appropriate training of para-professionals, audiologists can treat individuals in remote places who might otherwise not receive care. Tele-practice also provides convenience to patients who live relatively close by, but nonetheless find it challenging to visit the clinic for routine problems. By making oneself available using tele-technology, the audiologist helps patients conserve their physical energy, time, and travel expenses, while keeping abreast of the patient's challenges as they develop.
f. The topic of *Infection Control* relates to every aspect of audiology, because it is important not to harm the people whom we are here to help. Infection control is part of every aspect of our work and for this reason it is the first of many chapters, in the future, that will be available from the Point on internet.

Other New Features in This Handbook

In this edition of the Handbook we have reduced the number of references provided in each chapter, but there are extensive lists of references for the interested students, professors, and researchers on the Point. In this way the reader is not encumbered with reading through or skipping over many references when trying to understand the concepts and to remember the facts in this book. At the same time there are thousands of references organized by chapters online for those who are interested in research or for greater depth on the topics covered in this book.

Another new feature is the thought questions at the end of each chapter. They will ask how and what you would do in dealing with, or solving, problems associated with the information in the chapter. This is not another hoop to jump through but a valuable exercise. The student must take what they have learned from the chapter and combine it with their other knowledge to figure out a good solution to a problem/question. In this way they take what was on page and internalize it, while it is fresh in their minds, and put the information to a practical use. This will help you to internalize the information and make the material your own.

Terminology

Most of the terms used in this edition are standard in the field at this time. However, when a change is made it should be for a worthwhile purpose and not one that creates important problems. For example, this writer was pleased to

see a recent change back to a previous term. What was once called *Central Auditory Processing* was changed to *Auditory Processing* and recently was changed back to the clearer and more specific *Central Auditory Processing* again (American Academy of Audiology, 2010).

Sensory/Neural

A conductive loss is a mechanical impairment of hearing, associated with the outer and/or middle ears. For many years a nonconductive loss had been called a "nerve loss." After WWII it was changed to "sensory-neural loss" when ENT doctors and audiologists were then able to separate sensory (cochlear) from neural (acoustic nerve or brainstem) disorders. For example, cochlear problems (such as Meniere's disease) were demonstrated by a rapid growth of loudness when a sound was presented above the person's threshold of hearing. On the other hand with retrocochlear losses (e.g., auditory nerve or brainstem) there was no accelerated growth of loudness with sounds above the neural hearing level (as with a person who had an auditory nerve tumor). However, after a number of years the term sensory-neural was changed to "sensorineural." There was little reaction to this minor change.

I was shocked, however, to receive an angry phone call from a doctor who claimed that I made a mistake which caused him to delay surgery for his patient's auditory nerve tumor. From a review of my report it was abundantly clear that the patient had retrocochlear characteristics that are consistent with an "eighth nerve or brainstem involvement" and not cochlear involvement. How could that have been misinterpreted? The physician only had read up to the first test result, that puretone testing showed a "sensorineural loss in the right ear." On seeing the term "sensorineural" he incorrectly concluded that it was a cochlear problem and not a very dangerous auditory nerve tumor. He did not know that the term *sensorineural* could represent two importantly different types of hearing loss. Puretone thresholds distinguish conductive from both sensory and neural disorders.

Later on similar mistakes, with the term *sensorineural,* were made by knowledgeable audiologists in two separate publications. This convinced me that the term *sensorineural* can create serious problems that should be less problematic with the original term *sensory-neural.*

Since the second edition of the Handbook we have used the term *sensory-neural* to avoid the errors caused by sensorineural (Katz, 1978). If those who coined the term *sensorineural* originally did not try to combine two auditory components that we try hard to distinguish from one another, it is likely that fewer problems would have occurred. Other authors have recognized the problem with the term *sensorineural.* Jacobson and Northern (1991) suggest using just sensory or neural, when it is clearly one or the other. Martin and Clark (2012) avoid the confusion by using the term *sensory/neural* which is also a good way to clarify the term. For this edition we will combine both the Jacobson and Northern and the Martin and Clark approached as this seems to be better than sensory-neural and avoids the problems that 'sensorineural' has caused.

Puretone

The reader might infer that the writer does not like compound words (two words that are combined to form a composite of the two, e.g., flashlight, textbook). We rarely combine opposites (e.g., dogcat, daynight, or even sensorineural). But when two words are frequently spoken together (e.g., *base* and *ball*) often the first step is to hyphenate them (base-ball) and when people get used to this expression, they are often combined and made a compound word (*baseball*).

The term "pure tone" is shown one or more times on every audiogram and appears in almost every report and is a very common type of audiometer (but in that case it might be hyphenated because it is followed by a noun, e.g., pure-tone audiometer). Because (1) we have to explain this to students and often have to decide if it needs a hyphen when we are writing, and (2) it is surely time to graduate from pure-tone to puretone, this change seems appropriate. In this case there is no compelling reason for doing so (as it would be in the case of sensorineural) but it seems that it is time for "pure" and "tone" to be officially married and to be a compound word forever more.

ESPECIALLY FOR STUDENTS– SOME SUGGESTIONS

As a student, it is most helpful to educate yourself broadly in your profession and related subjects. You may benefit from speech, psychology, and many other courses as much as from some of your audiology courses. The ability to take a broader view is certainly an advantage no matter how you plan to practice audiology.

When you have a choice in taking your first job, it is well to take one that covers a wider area of professional activity over one that is narrow. You may find that an area that previously did not seem too interesting is one that you realize is very interesting or gratifying. Also, if you have a broad experience you can qualify for more opportunities later on.

As you get deeper into your areas of major interest you will necessarily reduce how broadly you can practice. But having a prior background or learning can help you in what you are doing and perhaps provide variety in your professional activities. Later on, if you have specialized in one area then an exciting and enriching aspect is to carry out research to improve your success or simply to obtain a better understanding. One way to repay your profession for training you is to supervise students in your external practicum site. Mentoring students and sharing what you have learned can be most rewarding, but in addition you may learn some

new concepts from the students that you may have missed or learn from having to answer their questions.

It is our pleasure to provide you with this book full of knowledge that was written by dozens of audiologists who have enjoyed sharing with you their hundreds of years of experience in this wonderful field. Finally, as professionals we should be committed to helping those we serve. We also need to follow the rules. Of course, in addition your work needs to provide you with the necessities of life. Despite these constraints, to a great extent, your profession is pretty much what you make of it.

FOOD FOR THOUGHT

1. What personal characteristics and experiences do you have that you think will be helpful to you as an audiologist?
2. You are the Director of an Audiology Department at a medical center. There is a need to establish guidelines for the audiologists to provide a degree of consistency (e.g., in reports). You have seen "sensorineural" spelled like that and also as sensory/neural. Consider the main reason for choosing each of them for your department.
3. If you were the editor of *Handbook of Clinical Audiology* and could only add one chapter to this edition, based on what you know or imagine, which of the six new chapters (see above) would you choose and why?

KEY REFERENCES

thePoint A full list of references for this chapter can be found at http://thePoint.lww.com. Below are the key references for this chapter.

American Academy of Audiology. (2010) Guidelines for diagnosis, treatment and management with children and adults with central auditory processing disorders. Available online at: http://www.audiology.org/resources/documentlibrary/documents/CAPDGuidelines 8–2010.pdf.

Jacobson J, Northern J. (1991) *Diagnostic Audiology.* Austin, TX: Pro-Ed; p 8.

Katz J. (1978) Clinical audiology. In: Katz J, ed. *Handbook of Clinical Audiology.* Baltimore, MD: Williams & Wilkins Co.; p 5.

Martin F, Clark JG. (2012) *Introduction to Audiology.* Boston: Pearson; p 446.

CHAPTER 2

Calibration

Laura Ann Wilber and Robert Burkard

WHY CALIBRATE?

In some ways, calibration can be compared to exercising. We know it is good for us, but some of us would prefer not to participate. However, unlike exercising, if one does not calibrate, it hurts others (our clients) more than it does us. For years, many clinicians felt that calibration was something that researchers did but that such procedures were not necessary in the clinic. Today, that basic attitude has changed dramatically. The Occupational Safety and Health (OSHA) regulations (1983) require that audiometric equipment be regularly checked. Some state regulations for hearing aid dispensers and/or for audiologists also require that equipment calibration (and records of calibration) be maintained. Furthermore, many state health departments concerned with school screening also insist on having calibration checked on a routine basis. Thus, we *must* calibrate if we are to meet the current regulations, and we *should* calibrate to make sure our results are within specified tolerances.

Initial audiometric calibration provided by the manufacturer is insufficient to guarantee that the audiometer will function correctly over time. Although modern digital audiometers are less likely to arrive out of calibration and are less likely to develop problems later than the older vacuum tube machines, even brand new audiometers that have just arrived from the factory, as well as audiometers that were in perfect calibration when they were new, can show variations in sound level, frequency, distortion, to name a few. Problems are often related to the transducers (earphones, bone vibrators, loudspeakers), but the electronic components can also lead to the audiometer failing to remain in calibration. It is the responsibility of the user (i.e., the audiologist) to either check its calibration personally or to arrange for regular calibration of the equipment by an outside service. The audiologist who has demonstrated that the clinic equipment is "in calibration" can then feel confident in reporting the obtained results. Calibration checks can determine if an audiometer meets appropriate standards and also whether the instrument has changed over time.

The purpose of this chapter is to tell the audiologist or student how to check audiometers to determine if they meet current national (or international) standards. Throughout this chapter, we will refer to various standards. In the United States, we (mostly) rely on standards that have been approved by the American National Standards Institute (ANSI). Nonetheless, we will also refer standards written and approved by the International Electrotechnical Commission (IEC) and the International Organization for Standardization (ISO). Since these standards do not have the status of law, it is important to understand how, and perhaps why, they are developed: Standards are developed so that manufacturers of equipment (from all countries) and users of the equipment are all on the same page. According to its website (http://www.ansi.org/about_ansi/overview/overview.aspx?menuid=1), ANSI is "a private, nonprofit organization (501(c) 3) that administers and coordinates the U.S. voluntary standardization and conformity assessment system." Its "mission is to enhance both the global competitiveness of U.S. business and the U.S. quality of life by promoting and facilitating voluntary consensus standards and conformity assessment systems, and safeguarding their integrity" (ANSI, 2004). Some values (e.g., the "0" hearing level [HL]) have both international and national approval. In most cases, ANSI standards and ISO and IEC standards are technically very similar (in current jargon, this is called harmonization). Harmonization of ANSI and international standards enhances commercial interchange between nations. If, for example, the ANSI audiometer standard was radically different from the IEC standard, manufacturers would have to build instruments solely for the American market and solely for the European or World market. In a relatively small-volume industry (such as audiometric instrumentation), this would be impractical at best.

All standards are reviewed periodically. If they are reaffirmed (and not changed), then the standard will read, for example, ANSI S3.39-1987 (R2012). This means the standard was approved in 1987 and was most recently reaffirmed in 2012. If the standard is revised, then the date changes (e.g., ANSI S3.6-2010, which was previously ANSI S3.6-2004). An announcement is made when the standard is going to be voted on so that interested parties can obtain a copy and comment to the person or persons who will be voting. For example, audiologists might contact the American Speech-Language-Hearing Association (ASHA) or the American Academy of Audiology (AAA), both of which are voting

members. This is the basic procedure for development and approval of standards. For more information on the standards process, the reader is referred to Melnick (1973) and Wilber (2004). There are three primary sources of funding for the production of standards in acoustics: financial support from Acoustical Society of America (ASA), fees paid by the voting members of an Accredited Standards Committee, and income from the sales of standards. Through your purchase of standards, you are supporting the efforts of those professionals who donate their time and effort to develop and maintain ANSI standards. Contact information of the secretariat of ANSI S1, S2, S3, and S12 is:

Acoustical Society of America
ASA Secretariat
35 Pinelawn Road, Suite 114E
Melville, NY 11747-3177
E-mail: asastds@aip.org

PARAMETERS OF CALIBRATION

The first step in learning how to check calibration should always be to read the appropriate manual(s) that accompany the audiometric equipment that you have purchased. Additional resources include electronic parts, stores that often have basic manuals on test equipment, ASHA and ASA. A number of books have also discussed procedures for acoustic measurements and equipments that might be used in such measurements (Beranek, 1988; Decker and Carrel, 2004; Silverman, 1999). The United States Government Printing Office is also a good source of information on basic test procedures. The specific parameters that must be checked in an audiometer are outlined in standards provided by the ANSI and the IEC. See Table 2.1 for a listing of standards relevant to calibration of audiometric equipment. It is beyond the scope of this chapter to discuss each area of calibration in detail. For the readers who intend to perform their own calibration of audiometric equipment, they need to purchase copies of the latest standards to verify the exact parameters to be checked and their permissible variability. To better understand the procedures for checking calibration, one must first understand the parameters that need to be checked, as well as the equipment used to perform these calibration checks. For puretone and speech audiometers, the three parameters are (1) frequency, (2) level (sound pressure level [SPL] or force level or [FL]), and (3) time. These parameters apply whether one is using a portable audiometer, a standard diagnostic audiometer, or a computer-based audiometric system.

Some organizations, such as ASHA and OSHA, specify time intervals at which calibration checks should be made. With current solid-state electronic circuitry, frequency, and time, parameters should be checked when the audiometer is first acquired and at yearly intervals thereafter. Older equipment should be checked at least biannually. For newer equipment, if daily listening checks are strictly enforced, transducers should be verified at least annually, unless there is reason to suspect that the output has changed. If daily listening checks are not strictly enforced more complete checks might be necessary. In addition to regularly scheduled checks, audiometers should be tested *whenever* the clinician notices anything unusual in their performance.

Sometimes test results themselves reveal the need for an immediate calibration check (e.g., when the same air–bone gap is obtained for two successive patients). It is always better to check the audiometer first rather than assume the problem lies with the client or clinician. A quick biologic check (described later) can always be performed. If this confirms the probability of an equipment problem, then a more elaborate electroacoustic check should be carried out.

If the audiologist discovers that the frequency or time components of the audiometer are out of calibration, then in most instances the manufacturer or a local representative should be contacted for immediate repair and/or proper calibration of the instrument. However, if there is a stable deviation in output level at a given frequency, calibration corrections can be made by adjusting the trim pots (potentiometers) on the audiometer, by using the audiometer's self-calibrating mechanism, or by posting a note on the front of the audiometer indicating the corrections. If paper corrections must be used, then the adjustment in decibels (plus or minus) that should be made at the various frequencies should be shown for each transducer. Note that if the SPL output is too high (e.g., by 5 dB), then you must increase their audiometric threshold (e.g., by 5 dB HL). Most modern audiometers provide some sort of internal (typically software based) calibration system for earphones, and many also provide this for bone conduction or sound field. If one plans to use bone vibrators for both mastoid and frontal bone testing or two sets of earphones with the same audiometer (e.g., supra-aural earphones and insert receivers), it is probably advisable to use "paper corrections," rather than trying to adjust trim pots between each transducer's use. If frequent level adjustments are required, it is probably wise to check with a qualified technician.

INSTRUMENTATION

As mentioned earlier, the calibration of an audiometer requires the use of various pieces of electroacoustic and electronic instrumentation. Most, if not all, graduate audiology programs will have the instrumentation needed to at least evaluate whether the audiometer meets the reference equivalent threshold sound pressure level (RETSPL), frequency, linearity, and distortion standards specified in ANSI S3.6 Specification for Audiometers. In this section, we will review the use of several basic instruments, including sound level meter (SLM), multimeter, frequency counter, oscilloscope, and digital spectrum analyzer. More details on acoustics and instrumentation can be found in numerous

TABLE 2.1

ANSI, IEC, and ISO Standards for Audiometers and Audiometric Testing

Number	Title
ANSI S3.1-1999 (R 2008)	Maximum Permissible Ambient Noise for Audiometric Test Rooms
ANSI S3.2-2009	Method for Measuring the Intelligibility of Speech Over Communication Systems
ANSI S3.6-2010	Specification for Audiometers
ANSI S3.7-1995 (R 2008)	Coupler Calibration of Earphones, Method for
ANSI S3.13-1987 (R 2012)	Mechanical Coupler for Measurement of Bone Vibrators
ANSI S3.20-1995 (R 2008)	Bioacoustical Terminology
ANSI S3.21-2004 (R 2009)	Method for Manual Pure-Tone Threshold Audiometry
ANSI S3.25-2009	Occluded Ear Simulator
ANSI S3.36-2012	Specification for a Manikin for Simulated In Situ Airborne Acoustic Measurements
ANSI S3.39-1987 (R 2012)	Specifications for Instruments to Measure Aural Acoustic Impedance and Admittance (Aural Acoustic Immittance)
ANSI S1.4-1983 (R 2006)	Specifications for Sound Level Meters
IEC 60318-1:2009	Electroacoustics: Simulators of Human Head and Ear. Part 1–Ear Simulator for the Calibration of Supra-aural and Circumaural Earphones
IEC 60318-4:2010	Electroacoustics: Simulators of the Human Head and Ear. Part 4–Occluded-Ear Simulator for the Measurement of Earphones Coupled to the Ear by Means of Ear Inserts
IEC 60318-6:2007	Electroacoustics: Simulators of Human Head and Ear. Part 6–Mechanical coupler for the measurement on bone vibrators
IEC 60645-3:2007	Electroacoustics: Audiometric equipment. Part 3–Auditory Test Signals of Short Duration for Audiometric and Neuro-otological Purposes
IEC 60645-5:2004	Electroacoustics: Audiometric Equipment. Part 5–Instruments for the Measurement of Aural Acoustic Impedance/Admittance
IEC 60645-6:2009	Electroacoustics: Audiometric Equipment. Part 6–Instruments for the Measurement of Otoacoustic Emissions
IEC 60645-7:2009	Electroacoustics: Audiometric Equipment. Part 7: Instruments for the Measurement of Auditory Brainstem Responses
IEC 60645-6:2009	Electroacoustics: Audiometric Equipment. Part 6: Instruments for the Measurement of Otoacoustic Emissions
IEC 60645-7:2009	Electroacoustics: Audiometric Equipment. Part 7: Instruments for the Measurement of Auditory Brainstem Responses
ISO 8253-1:2010	Acoustics: Audiometric Test Methods. Part 1: Basic Pure-Tone and Bone Conduction Threshold Audiometry
ISO 389-1:l998	Acoustics: Reference Zero for the Calibration of Audiometric Equipment. Part 1: Reference Equivalent Threshold Sound Pressure Levels for Pure Tones and Supra-aural Earphones
ISO 389-2:l994	Acoustics: Reference Zero for the Calibration of Audiometric Equipment. Part 2: Reference Equivalent Threshold Sound Pressure Levels for Pure Tones and Insert Earphones
ISO 389-3:l994	Acoustics: Reference Zero for the Calibration of Audiometric Equipment. Part 3: Reference Equivalent Threshold Force Levels for Pure Tones and Bone Vibrators
ISO 389-4:l994	Acoustics: Reference Zero for the Calibration of Audiometric Equipment. Part 3: Reference Equivalent Levels for Narrow-Band Masking Noise
ISO 389-5:2006	Acoustics: Reference Zero for the Calibration of Audiometric Equipment. Part 5: Reference Equivalent Threshold Sound Pressure Levels for Pure Tones in the Frequency Range 8 kHz to 16 kHz
ISO 389-6:2007	Acoustics: Reference Zero for the Calibration of Audiometric Equipment. Part 6: Reference Threshold of Hearing for Test Signals of Short Duration
ISO 389-7:2005	Acoustics: Reference Zero for the Calibration of Audiometric Equipment: Part 7: Reference Threshold of Hearing under Free-Field and Diffuse-Field Listening Conditions
ISO 389-8:2004	Acoustics: Reference Zero for the Calibration of Audiometric Equipment. Part 8: Reference Equivalent Threshold Sound Pressure Levels for Pure Tones and Circumaural Earphones

ANSI, American National Standards Institute; ASHA, American Speech-Language-Hearing Association; IEC, International Electrotechnical Commission; ISO, International Organization for Standardization.

NOTE: All ANSI, ISO, and IEC Standards referred to in this chapter are listed in this table.

texts (e.g., Decker and Carrell, 2004; Harris, 1998; Rosen and Howell, 1991; Speaks, 1996).

Multimeter

The term "multimeter" indicates that this device can be used to make multiple measurements. In most cases, a multimeter will allow one to make measurements of voltage, current, and resistance. Each of these measurements is made differently, and we will limit our discussion herein to making voltage measurements. To measure voltage, we must make the measurement in parallel to (across) the device of interest. For example, if we are interested in attenuator linearity, we want to place the leads of the multimeter across the earphone leads, with the earphone plugged into the audiometer output. We can replace the earphone with an equivalent impedance (in most cases, a 10-, 50-, or 300-ohm resistor for ER-3 A, TDH-39, TDH-49, or TDH-50 earphones). Simply unplugging the earphones and plugging in the multimeter will likely produce inaccurate results, because this approach in most cases will change the load impedance of the audiometer output. It is important to purchase a true root mean square (RMS) multimeter for accurate RMS voltage readings. It is important to set the meter to AC, or alternating current (vs. DC, or direct current), voltage. The meter is most accurate when set to the lowest voltage range possible. In most cases, the voltage range is set in powers of 10, where the listed voltage is the maximum voltage possible for that voltage range. When this maximum voltage is exceeded, an overload is indicated (see multimeter manual for the overload indicator for your multimeter). You adjust the multimeter range until you have the most sensitive range (lowest maximum voltage) where the output is NOT overloaded.

Frequency Counter

This might be a stand-alone device, or it might be an option on your multimeter. In the case of a stand-alone device, a frequency counter will often have a trigger adjust (the voltage level and direction: positive-going or negative-going) that determines when an event is triggered. The frequency counter combines an event counter with an accurate clock. The ratio of events (i.e., cycles) divided by the time elapsed gives you the frequency (in hertz). Thus, if 20 events are measured in 10 ms (one-hundredth of a second), then the cycles per second (or hertz) = 20 cycles/0.01 s = 2,000 cycles/s (Hz). If the counter does not trigger (no events counted), you need to reduce the trigger level or turn up the signal (e.g., increase the dB HL on the audiometer dial). If the frequency counter reads a number substantially larger than expected, then it is possible that the trigger level is set too low (or the signal presented is set too high) and that multiple triggers per cycle are occurring. In this case, turning the signal level down or increasing the trigger level of the frequency counter should correct this problem. If the frequency counter is an option in a multimeter, there is often no adjustable trigger level, and the signal level must be changed to correctly trigger the counter function.

Sound Level Meter

The SLM is actually multiple instrumentation components provided in a single instrument. You can combine separate instruments into a usable device when an SLM is not available. At a minimum, for checking the calibration of RETSPL (i.e., 0 dB HL values on the audiometer), you need an acoustic calibrator, an appropriate coupler (2 cc and/or 6 cc), a microphone, and the SLM. SLMs used for checking the calibration of audiometers should be Type 1, as should microphones used for such calibrations. The most commonly used Type 1 microphone is a condenser microphone. Condenser microphones come in four standard sizes (referring to their diameter): 1/8″, 1/4″, 1/2″, and 1″. For calibration of a supra-aural earphone, a 1″ microphone is specified in ANSI S3.6-2010 (because of its sensitivity—see the statement that follows). In general, the smaller the microphone is, the higher its upper frequency cutoff and the less its sensitivity. Sensitivity is a measure of its efficiency transferring sound pressure into voltage and is commonly reported as millivolts per pascal, or in dB re: 1 V/Pa. Many condenser microphones require a DC polarization voltage of 200 V. Some condenser microphones are prepolarized and hence do not require an externally applied polarization voltage. Microphones also come as pressure microphones (to be used in a coupler), free-field microphones (to be used in sound field recordings such as when measuring the ambient noise in the sound booth), or random-incidence microphones (for measures in, e.g., reverberant environments). More detailed information about microphones and SLMs can be found in Johnson et al. (1998) and Yeager and Marsh (1998). It is important that your SLM and microphone be compatible (i.e., provide the correct polarization voltage), or equipment damage and/or incorrect SPL measures may result.

The SLM also contains amplifiers (whose gain is changed when you change the SPL range), time-weighting circuits (for fast, slow, and possibly impulse and peak time weightings), various filter settings (e.g., dBA, dBC, and octave and/or third-octave band filters), as well as a display function (this could be a volume unit (VU) meter, an LED indicator, and/or a digital readout). The gain of an amplifier in the SLM must be adjusted to account for the sensitivity of each microphone. For example, a 1″ microphone might have a sensitivity of 50 mV/Pa, whereas a 1/4″ microphone might have a sensitivity of 1 mV/Pa. If the SLM were adjusted appropriately for the 1/4″ microphone, then when 1 Pa of pressure was presented to the microphone diaphragm, the SLM would read 94 dB SPL [20 log(1 Pa/0.0002 Pa) = 94 dB SPL]. If we replaced the 1/4″ microphone with the

1″ microphone but did not change the SLM amplifier gain, the 1″ microphone would read 128 dB SPL [94 dB SPL + 20 log(50 mV/1 mV)]. How, then, do we calibrate the SLM so that it displays the correct SPL? In most instances, we would use a device that presents a known SPL to the diaphragm of the microphone. Two types of calibration devices are commercially available for this purpose: pistonphones and acoustic calibrators. The former produces sound by a mechanical piston, whereas the latter uses an electrical oscillator and a transducer to produce the tone. Each calibrator produces a specified SPL at a specified frequency, and this calibrator should be periodically sent back to the manufacturer to assure it remains within specified tolerances of frequency and SPL. These calibrators can accommodate a variety of microphone sizes by inserting nesting adapters. Using an acoustic calibrator is very simple: turn on the SLM, place the calibrator snugly over the microphone, and turn on the calibrator. Making sure that the frequency response of the SLM is wideband (flat, or dBC if flat weighting is not available), adjust the gain of the SLM (by trimming a calibration potentiometer using a screwdriver or via software) until the specified output of the calibrator (e.g., 114 dB SPL) is displayed on the SLM.

Once the SLM is calibrated, you must remove the acoustic calibrator (or pistonphone) and place an appropriate coupler over the microphone: a 2-cc coupler for insert earphones (e.g., Etymotic ER3 A earphones) or a 6-cc coupler for supra-aural earphones (such as TDH-39, TDH-49, or TDH-50 earphones). ANSI S3.6-2010 has RETSPL values for both insert and supra-aural earphone for several 6-cc (National Bureau of Standards [NBS] 9-A, IEC 318) and 2-cc (HA-1, HA-2, occluded ear simulator) couplers.

Oscilloscope

The oscilloscope, in its most common display mode, presents voltage as a function of time. Oscilloscopes come in analog and digital types. In the analog oscilloscope, the output of an electron gun transiently illuminates the screen of a cathode ray tube. Freezing the display on the oscilloscope screen involves repeated triggering of the oscilloscope on a fixed phase of the stimulus. Specialized analog oscilloscopes that can freeze a display for prolonged periods of time are called storage oscilloscopes. A digital oscilloscope is similar to an analog oscilloscope, except that instead of electron guns and a cathode ray tube, the signal is recorded by an analog-to-digital converter and displayed on a flat panel display. Digital oscilloscopes often have features that are not typically available on analog oscilloscopes (e.g., storage of waveforms, cursor functions, and summary statistics such as peak-to-peak and RMS voltage calculations). Simple amplitude and voltage measurements are easily performed on a signal using an oscilloscope. Manipulations of the time base (in time per division) and amplitude (in volts per division), as well as the appropriate adjustment of the trigger, allow the "freezing" of the signal on the oscilloscope. To measure, for example, peak-to-peak voltage, one counts the number of vertical divisions (usually a division is a centimeter) extending from the positive to the negative extremes and multiplies this number of divisions by the voltage per division to obtain the peak-to-peak voltage. It should be noted that measurements made on an analog oscilloscope are assumed to have an error of 5% or more.

Spectrum Analyzer

Numerous devices can be used to provide a frequency-domain representation of a signal (including the octave or third-octave band filters available on many SLMs). In this section, we will limit our discussion to instruments referred to as digital spectrum analyzers. These instruments may be stand-alone hardware devices or might be part of a computer-based hardware/software application. These devices convert an analog input signal to digital format by use of an analog-to-digital converter. It is important that the reader understand that if the sampling rate used during analog-to-digital conversion is too slow, it can cause the generation of "false frequencies" in a process called aliasing. Many spectrum analyzers preclude aliasing by judicious use of a low-pass filter (called an antialiasing filter). It should also be noted that not all possible signal amplitudes can be encoded following analog-to-digital conversion, but signal level is rounded off ("quantized") and that the magnitude of possible quantization error is related to the voltage range and the resolution (related to the number of bits) of the analog-to-digital converter. The time-domain signal is digitized over a limited time period, called the time window or the time epoch. Once the signal is digitized into a time epoch, it is converted into the frequency domain by Fourier transformation. (See Rosen and Howell, 1991 for a more complete explanation of aliasing, antialiasing, quantizing, and digitization.) The fast Fourier transform (FFT) is one of many algorithms that have been developed to convert a time-domain (voltage over time) signal into a frequency-domain (amplitude across frequency) signal. Another term for the frequency-domain representation is the spectrum. In addition to the possibility of quantization errors and aliasing, you must be aware that signal processing prior to Fourier transformation can have an influence on the results. Because of some underlying assumptions about the periodic nature of the discretely sampled signal, the spectrum of the signal is distorted unless an integer number of cycles of all frequencies is contained in the time epoch over which the signal is digitized. To prevent the distortion (often called leakage) that occurs when a noninteger number of cycles is contained in the time epoch, the digitized time epoch can be shaped. This shaping multiplies the signal by values at or near zero, near the beginning and end of the time window and weights them at or near 1, near the middle of the time window. One popular windowing function is the

Hanning window. A given windowing function trades amplitude uncertainty for frequency resolution. Once the data are converted to the frequency domain, the amplitude of a given Fourier coefficient (e.g., frequency) can be determined using a cursoring function. It should be noted that Fourier transformation produces multiple discrete harmonically related (i.e., integer multiples) spectral components. The lowest frequency (fundamental frequency) and, hence, the frequency interval between components are related to the recorded time-domain signal. If the time-domain signal is, for example, 200 ms (0.2 s), then the lowest frequency is 1/0.2 s, or 5 Hz. The longer the time window is, the better the spectral resolution.

BASIC EQUIPMENT

The basic calibration equipment for checking output levels of an audiometer should include (1) a voltmeter or multimeter; (2) condenser microphones (both pressure and free-field types); (3) acoustic calibrator; (4) a 6-cc coupler (NBS 9-A or IEC 318); (5) a 2-cc coupler (ANSI HA-1 or HA-2 or IEC occluded ear simulator); (6) a 500-g weight; (7) a mechanical coupler for bone vibrator measurements (artificial mastoid); and (8) an SLM (or equivalent). When purchasing any of the above components, it is wise to check with others who use similar types of equipment to find the best specific brands available locally.

Other equipment such as a digital oscilloscope, frequency counter, and/or a spectrum analyzer will also prove to be invaluable in checking the acoustic parameters of audiometers. In many instances, this equipment can be shared by more than one facility. If one has only one or a few audiometers, a service contract is most sensible. If one has a substantial number of pieces of audiometric test equipment, an SLM (with appropriate couplers, microphone(s), and acoustic calibrator) and a multimeter should be purchased and used. If the accuracy of the audiometer is questioned, it necessitates shutting down the equipment or retesting patients at a later date. This translates into time and financial loss, not to mention more serious consequences in surgical or medicolegal cases. In a busy practice, such a loss would surely be equivalent to the cost of one or more pieces of electronic test equipment that would prevent this problem. This of course assumes that someone working in that practice setting is competent to check the calibration of the audiometric equipment.

CHECKING THE CALIBRATION OF PURETONE AUDIOMETERS

Basic Signal

As soon as one obtains a new audiometer, the manual should be read and, if any calibration instructions are provided, they should be followed.

Biologic Check

After the audiometer has been installed, plugged in, turned on, and allowed to warm up, the operator should listen to the signal at different dial settings through each transducer (earphone, loudspeaker, and bone vibrator). With a little practice, one can hear basic faults in the equipment. A vague complaint to the audiometer technician or distributor that it "sounds funny" is as futile as telling an auto-repair person the same thing. However, a specific description of the sound and when it occurs can help determine the source of the trouble. If the technicians are given a detailed description of the problem, then the fault may be found more quickly, without wasting their time and your money.

Much information on the source of the problem may also be obtained by inspecting the audiometer. Following are some areas of potential malfunction that the audiologist should check periodically (normally on a daily basis):

1. Check the power, attenuator, earphone, and vibrator cords for signs of wear or cracking. Listen to the tone through the transducer at a comfortable level while twisting and jiggling the cords. A defective cord will usually produce static or will cause the tone to be intermittent. Tightening the earphone screws and/or resoldering the phone plug connections might fix the problem. If this does not alleviate the problem, it is wise to replace the cord.
2. If the audiometer has dials, check for loose dials or for dials that are out of alignment. If such faults exist, the dial readings will be inaccurate. Defective dials should be repaired immediately (sometimes this just requires tightening the set screws that hold the dial to the audiometer), and the audiometer should be recalibrated to determine outputs at the "new" dial settings. Check to see that incremental changes are correctly reflected in the readout.
3. The audiologist should listen for audible mechanical transients through the earphone when the dials or switches are manipulated. The ANSI S3.6-2010 standard (section 5.4.4) suggests that two normal-hearing listeners should listen at a distance of 1 m from the audiometer with the earphones in place but disconnected and with a proper load resistance (coinciding with the impedance of the earphone at 1,000 Hz) across the circuit while manipulating the presenter/interrupter switch, and so on, to make sure that there are no audible signals that would inform the subject to the presence of the test signal. A mechanical transient can often be detected more easily by listening than through the use of electronic equipment.
4. To determine if electronic transients are audible, it is wise to listen to the output both at a moderate hearing level (e.g., 60 dB) and below the threshold of hearing. Electronic transients will show up on an oscilloscope as an

irregularity when the problem switch or dial is manipulated. The danger of an audible transient, whether mechanical or electronic, is that the patient may respond to the transient rather than the stimulus tone. Sometimes an antistatic or contact-cleaner spray can alleviate the problem of electronic transients.

5. The audiologist should listen for hum or static with the hearing level dial at a high value, both when a stimulus signal is present and when it is absent. One should not hear static or hum at levels below 60 dB HL on the dial.
6. "Cross-talk" may occur between earphones, that is, the signal that is sent to one earphone may be heard in the contralateral earphone. Such a problem could greatly affect the audiometric thresholds obtained on that audiometer, especially for cases with unilateral hearing loss. Cross-talk may be detected by unplugging one earphone, sending a signal to that phone, and listening to the other earphone. As before, when removing the earphone, a proper resistive load must be put in its place. The signal at a suprathreshold dial setting (e.g., 70 dB HL) should not be heard in the opposite earphone when a signal is presented in the normal manner. Cross-talk may be caused by faulty external wiring between the examiner's booth and that of the test subject or within the audiometer itself. Cross-talk must be corrected before any testing is carried out.
7. The clinician should listen to the signal while the attenuation dial is changed from maximum to minimum levels. For instance, a tone may be present at 20 dB HL on the dial, whereas no tone is present at 15 dB HL on the dial. In some cases, the tone stays at the same hearing level from 20 dB HL to −10 dB HL on the dial. These problems are easily detected by listening to the audiometer.
8. Finally, the threshold of the clinician (or a person with known hearing thresholds) should be checked with the earphones and bone vibrators to make sure that the outputs are approximately correct. If the levels are not within 10 dB of the previous threshold values, the output levels should be checked electronically.

Aside from these gross problems, which can be detected by looking or listening (see Figure 2.1 for an example of a form that may be used to aid the clinician in carrying out the listening check), the precise accuracy of the output levels

Audiometer serial #								
Date:								
Time:								
Checked by:								
Earphone cords								
Power cord								
Attenuator cord								
Hum								
Dials								
Frequency								
Attenuation								
Intensity right phone								
Intensity left phone								
Tone interrupter								
Tone pulse rate								
Cross-talk								
Acoustic radiation								
Bone vibrator(s)								
Loudspeakers								
Other comments								

FIGURE 2.1 Form for biologic check of audiometer. [Reprinted from Wilber L. (1972) Calibration: pure tone, speech and noise signals. In: Katz J, ed. *Handbook of Clinical Audiology.* 1st ed. Baltimore, MD: The Williams & Wilkins Company; pp 11–35, with the permission of Lippincott Williams & Wilkins.]

must be evaluated when the audiometer is first purchased and at regular intervals thereafter. Frequency, output level, linearity of attenuation, and percentage of harmonic distortion should all be checked electronically, in addition to the biologic check. Section 5.4 of ANSI S3.6-2010 describes various checks for unwanted sound from the transducer or audiometer.

Frequency Check

The frequency output from the audiometer should be checked by using an electronic frequency counter. This instrument will tell the exact frequency of the output signal. Quite accurate frequency counters are often included in a digital multimeter. The electrical output from the audiometer may be routed directly to the instrument (i.e., unplug the earphone, then plug in the frequency counter input to the audiometer output) because the frequency is determined by an oscillator in the audiometer rather than the transducer. By using an electronic frequency counter, one can easily determine if the output from the audiometer corresponds to the nominal frequency. The standard for audiometers allows a tolerance of ±1% of the indicated frequency value for Type 1 and 2 audiometers; ±2% for Type 3 and 4 audiometers; and ±3% for Type 5 audiometers. For example, if the audiometer dial reads 1,000 Hz, then the actual output must be between 990 and 1,010 Hz for a standard diagnostic (Type 1) audiometer.

Frequency should be checked on initial receipt of the audiometer and at yearly intervals thereafter. Nevertheless, it is appropriate to listen to the audiometer each day to judge whether the frequencies are maintaining reasonably good accuracy.

Harmonic Distortion Check

Linearity measurements may also help detect distortion in a transducer or in the audiometer itself. Distortion may appear as a lack of linear attenuation, especially at high output levels (90 dB HL and above). Harmonic distortion must be checked through the transducer itself. Excessive harmonic distortion is rarely caused by the audiometer but often arises in the various transducers. The maximum permissible total harmonic distortion in the current standard (ANSI S3.6-2010) is 2.5% for earphones and 5.5% for bone vibrators. The standard also shows the maximum permissible distortion for the second, third, fourth, and higher harmonics, as well as the subharmonics, across audiometric frequency.

Rise–Fall Time

The rise–fall time of the tone is a basic parameter of the audiometer, which may be checked by taking the output directly from the audiometer and routing it into a digital or storage oscilloscope. When gating the signal on, rise time is the length of time it takes for the signal to increase from −20 to −1 dB (10% to 90%) of its final steady-state value. The fall time is the length of time between −1 and −20 dB (90% to 10%) relative to its steady-state value. This is usually checked at a hearing level of 60 dB HL or less. ANSI S3.6-2010 specifies a rise time as well as a fall time of not less than 20 ms and not more than 200 ms. A detailed description of the rise and fall characteristics is given in section 7.5.3 of ANSI S3.6-2010.

Linearity Check

Attenuator linearity (the hearing level dial) may be checked electrically, directly from the audiometer, or acoustically through its transducer (earphone or bone vibrator). If measurements are to be made electrically, the earphone should remain in the circuit and the voltage should be measured in parallel to the earphone, or a dummy load that approximates the earphone impedance should replace the transducer. To check linearity, the audiometer should be turned to its maximum output and then attenuated in 5-dB steps until the output can no longer be read. Each attenuator on the audiometer should be checked separately. To meet the ANSI S3.6-2010 standard, the attenuator should be linear within 0.3 of the interval step or by 1 dB, whichever is smaller. That is, if you change the level in 5-dB steps, the audiometer must attenuate between 4 and 6 dB per step. If the attenuation step is 2 dB, then the reading should be between 1.4 and 2.6 dB per step (0.3×2 dB = 0.6 dB, which is less than 1 dB). As noted in section 7.2 (and section 7.3.3) of ANSI S3.6-2010, the SPL or FL of earphones, speakers, or bone vibrators can vary by no more than ±3 dB from 125 to 5,000 Hz and no more than ±5 dB at 6,000 Hz and above, at any dB HL dial setting.

Attenuator linearity should be checked annually. If a "fixed loss pad" (i.e., a device that automatically changes the signal level by a set amount, e.g., 20 dB) is present in the audiometer, its attenuation must also be checked. If the audiometer attenuates in 1- or 2-dB steps, then these smaller attenuation steps should be checked if they are used clinically.

EARPHONE LEVEL CALIBRATION

Real Ear Methods

There are two basic approaches for the calibration of earphones. One is the "real ear" method and the other is the "artificial ear" or coupler method. With the original real ear method, one simply tested the hearing of a group of normal-hearing persons, averaged the results, and checked to see that the average hearing of this group was at zero on the dial for each frequency. Although this is theoretically feasible with a large population sample, it is not a recommended

procedure. ANSI S3.6-2010, Appendix D, describes probe tube, loudness balance, and threshold procedures that may be used for this purpose. Clearly, these procedures are possible but quite unwieldy. For audiometers, this approach is technically incorrect because the ISO 389-1:1998 reference (which is also used in ANSI S3.6-2010) is not tied to normal hearing per se, but simply refers to an arbitrarily accepted SPL (i.e., the RETSPL or FL). If the audiologist wishes to use a new earphone (that is not listed in ANSI S3.6-2010 Standard, its appendix, or any subsequent revision), a real ear procedure might be the only way to check calibration, but if generally accepted earphones are used, it is much easier and more efficient to use an artificial ear/coupler method.

Artificial Ear (Coupler) Methods

The most commonly used procedure today is that of the "artificial ear," which consists of a condenser microphone and a 6-cc coupler (for supra-aural earphones) or 2-cc coupler (for insert earphones). The 6-cc coupler was originally chosen because it was thought that the enclosed volume was approximately the same as the volume under a supra-aural earphone for a human ear (Corliss and Burkhard, l953). However, since volume displacement is only one component of acoustic impedance, it cannot be assumed that the coupler actually represents a human ear. Burkhard and Corliss (1954) pointed out that the impedance characteristics of a 6-cc coupler probably simulates the impedance of the human ear over only a small part of the frequency range. Because the 6-cc coupler does not replicate the impedance of the human ear, it cannot be considered a true artificial ear. Subsequent work by Cox (1986), Hawkins et al. (1990), Killion (1978), and Zwislocki (1970, 1971) has quantified the differences between real ear and coupler values. In an attempt to solve this problem, the IEC 318 coupler was developed. However, there is still some disagreement as to the accuracy of this ear simulator (formerly called an artificial ear) because its impedance characteristics are also not exactly those of a real human ear. However, it is clearly more accurate than the present NBS 9-A coupler.

In addition to the problem of acoustic impedance characteristics, the NBS 9-A coupler is known to have a natural resonance at 6,000 Hz (Rudmose, 1964). This interferes with the measurement of the output of an audiometer earphone around that frequency. Other coupler problems are its size, its shape, and the hard walls that permit the possibility of standing waves at frequencies above 6,000 Hz. Despite these difficulties, the NBS 9-A coupler remains the accepted device (by ANSI S3.6-2010) for measuring the acoustic output from the audiometer through a supra-aural earphone. A coupler developed by Zwislocki (1970, 1971, 1980) appears to very closely approximate the acoustic impedance of the human ear. It is used in KEMAR (a manikin that has a pinna and an ear canal, as well as a coupler and microphone) (Burkhard, 1978; Burkhard and Sachs, 1975). This manikin is described in ANSI S3.25-2009, but RETSPLs are not given for supra-aural or insert receivers using the Zwislocki coupler or the manikin.

When checking the audiometer earphone output, the supra-aural earphone is placed on the coupler and a 500-g weight is placed on top of it. If using an SLM (rather than a microphone preamplifier), the output is read in dB SPL, where SPL = 20 $\log_{10}$ P/P_{ref} (where P is the observed sound pressure and P_{ref} = 20 μPa). After the earphone is placed on the coupler, a low-frequency tone (125 or 250 Hz) is introduced and the earphone is reseated on the coupler until the highest SPL value is read. This helps assure optimal earphone placement on the coupler. The output from the earphone is then compared to the expected values at each frequency. The standard SPL values that are used are given in (1) ISO 389-1:1998, often referred to as ISO-1964 because of its initial publication date, and (2) ANSI S3.6-2010. These values evolved through a "round robin" in which several earphones were measured on various couplers at a group of laboratories throughout the world (Weissler, 1968).

The current ANSI standard includes RETSPLs for the TDH-type earphones, as well as insert earphones. It also provides values for both the IEC and NBS couplers for supra-aural earphones and values for insert phones using an occluded ear simulator, HA-1 or HA-2 coupler. Figure 2.2 shows an audiometer earphone calibration worksheet, which contains the expected values at each frequency with TDH-39 or TDH-49 (or TDH-50) earphones in Telephonics type 51 cushions on an NBS 9-A coupler and insert receivers using an HA-1–type coupler. ANSI S3.6-2010 allows a tolerance from the listed values of ±3 dB from 125 to 5,000 Hz and ±5 dB at 6,000 Hz and higher.

The supra-aural output measurements referred to above are only valid when a supra-aural–type earphone cushion (which touches the pinna) such as the Telephonics 51 is used and not when a circumaural cushion (which encircles the pinna) is used. ANSI S3.6-2010 provides RETSPL values for several circumaural earphones (Sennheiser HDA200 and Koss HV/1 A) with an IEC 60318-2 coupler and a type 1 adapter (Sennheiser earphone) or type 2 adapter (Koss earphone). When the output of the audiometer through the earphone has been established, it is compared to the appropriate standard to determine whether it is in calibration or not. If possible, the audiometer trim pots (or by software adjustments in newer digital audiometers) should be used to bring the audiometer into calibration. However, when this is not possible or when different earphones will be used with the same audiometer, and when corrections are less than 15 dB, a calibration correction card may be placed on the audiometer showing the discrepancy from the established norm. It should be noted that if the output of the audiometer is, for example, 10 dB too low, then the dB HL correction sheet must be decreased by 10 dB. Such corrections must then be taken into consideration when an audiogram is plotted. If an audiometer is off by more than 15 dB at any frequency

AUDIOMETER EARPHONE CALIBRATION SHEET

Audiometer: _____ **S #** _____ **Earphone:** _____ **Channel:** _____ **Room:** _____

Calibrated by: _____ **Date:** _____ **Equipment:** _____

FREQUENCY:	125	250	500	750	1000	1500	2000	3000	4000	6000	8000
1. SPL*											
2. Audiometer dial setting											
3. Nominal ref. SPL (Line 1 – Line 2)											
4. Equipment and mike correction											
5. Corrected ref. SPL (Line 3 – Line 4)											
6a. TDH – 49/50 earphones**	47.5	26.5	13.5	8.5	7.5	7.5	11.0	9.5	10.5	13.5	13.0
TDH - 39	45.0	25.5	11.5	8.0	7.0	6.5	9.0	10.0	9.5	15.5	13.0
6b. ER 3-A earphones***	26.5	14.5	6.0	2.0	0.0	0.0	2.5	2.5	0.0	-2.5	-3.5
7. Calibration error (Line 5 – Line 6)											
8. Corrections @											

* SPL = sound pressure level in dB re: 20 μPA
** TDH-49/50 values from **ANSI S3.6-1996,** p. 18 (see standard for coupler and cushions)
*** ER3-A values from **ANSI S3.6-1996,** p. 20 using HA-1–type coupler (see standard for different coupler values)
@ Correction – Rounded to the neatest 5 dB; – = audiometer weak, make threshold better
+ = audiometer weak, make threshold better

FIGURE 2.2 Earphone calibration worksheet. [Reprinted from Wilber L. [1972] Calibration: pure tone, speech and noise signals. In: Katz J, ed. *Handbook of Clinical Audiology.* 1st ed. Baltimore, MD: The Williams & Wilkins Company; pp 11–35, with the permission of Lippincott Williams & Wilkins.]

or by 10 dB at three or more frequencies, it is advisable to have the audiometer put into calibration by the audiometer manufacturer or their representative. If the audiometer is new, it should meet ANSI S3.6-2010 tolerances. With current digital audiometers, deviations in desired output are usually due to the transducer rather than the audiometer, so sometimes it is easier to bring the audiometer into calibration by replacing the offending transducer(s).

BONE VIBRATOR CALIBRATION

Real Ear Procedures

Checking the calibration of a bone vibrator presents a different problem than that of an earphone. Whereas earphones can be checked easily using a microphone as a pickup, bone vibrators cannot. The original technique for checking bone vibrator calibration was a real ear procedure (American Medical Association, 1951), which was somewhat different than that used for earphones. The method assumes that air- and bone-conduction thresholds are equivalent. If 6 to 10 normal-hearing subjects are tested for both air and bone conduction with an audiometer whose air-conduction system is in proper calibration, bone-conduction corrections for the audiometer can be determined by using the difference obtained between air- and bone-conduction thresholds. This procedure makes a few assumptions that are not always met. For example, it presupposes that true thresholds can be obtained for all the normal-hearing subjects using the given audiometer. Because (1) many audiometers do not go below 0 dB HL and (2) the ambient noise in test booths often does not allow assessment below 0 dB HL, it is not always possible to determine the true threshold. To avoid these problems, Roach and Carhart (1956) suggested using individuals with pure sensory/neural losses for subjects in the real ear procedure. Such an approach eliminates the problems of ambient noise and lack of audiometric sensitivity, thus increasing the probability that one will obtain "true" thresholds. However, it can be problematic to find a group of subjects with "pure sensory/neural" losses (i.e., those who have no conductive component) and who have thresholds that do not extend beyond the bone-conduction limits of the audiometer. A more basic problem with real ear bone vibrator calibration is the supposition that air- and bone-conduction thresholds are equivalent in the absence of conductive pathology. Although this is certainly true, on

average, for a large group of people, it cannot be expected to be true for any individual or for small groups (Studebaker, 1967; Wilber and Goodhill, 1967).

Artificial Mastoid Procedure

The preferred procedure for calibrating bone vibrators involves the use of a mechanical coupler, often referred to as an artificial mastoid. Artificial mastoids were proposed as early as 1939 by Hawley (1939). However, it was not until Weiss (1960) developed his artificial mastoid that they became commercially available. Just as replication of the acoustic impedance of the human ear is difficult with a coupler, replication of the mechanical impedance of the head is difficult with an artificial mastoid. Because no commercially available artificial mastoid met the mechanical impedance requirements of the ANSI (S3.13-1972) or IEC (IEC 60373:1971) standards, both the ANSI and IEC standards were revised to conform more closely to an artificial mastoid that *is* available (ANSI S3.13-1987; IEC 60318-6:2007). ANSI S3.6-2010 gives threshold values in reference equivalent threshold force levels (RETFLs) that are appropriate for a bone vibrator such as the B-71 or B-72, or one meeting the physical requirements described in section 9.4.3 of ANSI S3.6-2010. The ISO standard (ISO 389-3:1994) gives one set of values that are to be used for all bone vibrators having the circular tip described in the ANSI and IEC documents. These values are also used in the ANSI standard. It is important to recognize that both the ANSI and the ISO values are based on unoccluded ears using contralateral masking. Thus, the values presuppose that masking will be used in the contralateral ear when obtaining threshold. One can use the same type of worksheet for bone as for air—substituting the appropriate RETFL values. In both earphone and bone vibrator calibration, it is important to check distortion as well as overall level through the transducer. Distortion may be measured directly with software integrated into the SLM or by routing the output of the artificial mastoid and SLM to a spectrum analyzer. As mentioned earlier, allowable distortion values for bone vibrators are more lenient than for earphones. This is because bone vibrators have more distortion than earphones. In addition to the earlier mentioned physical measurement procedures, the importance of just listening to the audiometer output through the bone vibrator cannot be overstated. The normal ear (with audiologist attached) should be able to perceive gross attenuation and distortion problems. The electroacoustic procedures, however, serve to quantify the problems that the human ear can only identify subjectively.

SPEECH AUDIOMETERS

Because running speech fluctuates in SPL (as well as frequency content) over time, the preferred method is to introduce a puretone (1,000 Hz) into the microphone, tape, or compact disc (CD) input of the speech circuit of the audiometer. The input level should be adjusted so that the monitoring VU meter on the face of the audiometer reflects the appropriate level, usually 0 dB. The output from the transducer is then measured. For most speech stimuli used for audiologic purposes, there is a 1,000 Hz tone on the tape or CD (or in other digital forms) that has an RMS voltage that is similar to the RMS voltage of the speech stimuli. Details concerning the calibration of the speech circuit of an audiometer are given in section 6.2 of ANSI S3.6-2010.

ANSI S3.6-2010 states that the speech output for the 1,000-Hz tone at 0 dB HL should be 12.5 dB above the RETSPL for the earphone at 1,000 Hz. Bone vibrators should be calibrated separately. All subsequent speech testing must be carried out with the monitoring meter peaking at the same point as during the calibration check. If, for example, one prefers −3 dB on the meter rather than 0 dB, then calibration of the 1,000-Hz tone must be peaked at −3 dB, or an appropriate correction must be made in reporting measurements.

The required flatness of the frequency response of the speech audiometer circuit is defined as ±3 dB for the frequencies of 250 to 4,000 Hz and from 0 to −10 dB between 125 and 250 Hz and ±5 dB between 4,000 and 6,000 Hz. ANSI S3.6-2010 gives specific requirements for checking the microphone circuit as well as the other speech input circuits. If the puretone and speech audiometers are separate machines, then the speech audiometer must also be checked for cross-talk, internal noise, and attenuator linearity as described earlier. More specific information on calibration of the speech circuit may be found in section 6.2.10 of ANSI S3.6-2010.

MONITORING METER

Monitoring (or VU) meters are indicators of signal level and are found on the face of most audiometers. The monitoring meter is calibrated relative to the input signal that it monitors and should not be interpreted as yielding any absolute values such as 0 dB SPL. On a speech audiometer, the meter is used to monitor the speech signal or to aid the audiologist in adjusting the input calibration tone that precedes the recorded speech materials. The specifications for the meters may be found in section 6.2.10 of ANSI S3.6-2010. In general, it is important that the meter be stable, that there is minimal undershoot or overshoot of the needle indicator relative to the actual signal, and that any amplitude change is accurately represented on the meter. The audiologist may check the meter and its entire accompanying input system as described below.

A puretone should be fed from an oscillator through an electronic switch to the input of the audiometer. The tone should be monitored by a voltmeter or an oscilloscope. By activating the electronic switch to produce a rapidly interrupted signal, one can watch the meter to ascertain whether

there is any overshoot or undershoot relative to the signal in its steady state. One must also check the response time of the needle on the VU meter. A computer-generated or tape-recorded tone may be used to ensure that the needle reaches its 99% state deflection in 350 ± 10 ms. In addition, the overshoot should be no more than 1.5%. One can insert a linear attenuator in the line between the oscillator and the audiometer input, one may reduce the output from the oscillator and the audiometer input, or one may reduce the output from the oscillator by a known amount (as monitored by a voltmeter or oscilloscope). The change in input should be accurately reflected by a corresponding change on the monitoring meter.

SOUND FIELD TESTING

ANSI S3.6-2010 describes the primary characteristics of sound field testing in section 9.5. This includes the test room, frequency response, method for describing the level of the speech signal, and the location of the speakers. Table 9 of the standard also gives specific RETSPL values for band-limited stimuli (frequency-modulated tones or narrow bands of noise) for binaural and monaural listening. An ASHA working group prepared a tutorial for sound field testing that discusses some of the problems of setting up the test procedure (ASHA, 1991). Characteristics of the frequency-modulated signals are given in section 6.1.3 of ANSI S3.6-2010. In addition, the characteristics of narrowband noise levels are presented in table 4 of the standard. The level for speech in sound field should be comparable to the corrected free-field response for earphones.

When calibrating stimuli is present in the sound field, it is important to place some sort of marker (such as a ring suspended from the ceiling) at the place where the subject's head will be. A free-field microphone should be placed so that the diaphragm is facing toward the direction of the plane-propagated wave (called frontal incidence). If a pressure microphone is used, the microphone diaphragm should be placed facing at a right angle to the direction of the plane-propagated wave (called grazing incidence). In either case, the microphone should be placed at the place where the subject's head will be during testing. There should be nothing between the speaker and the calibration equipment.

The amplifier hum or internal noise of the loudspeaker system should be checked. This may be done by adjusting the attenuator dial to some high setting (between 80 and 90 dB HL) and then measuring the output from the loudspeaker when no signal is present. That is, everything is in normal position for testing except that there is no signal presented to the speaker. The equipment noise (in SPL) should be at least 50 dB below the dial setting (in HL; i.e., if the dial reads 80 dB HL, then the equipment noise should be <30 dB SPL).

CALIBRATION OF ANCILLARY EQUIPMENT

Masking Generator

ANSI S3.6-2010 defines white noise, weighted random noise for masking of speech, and narrowband noise. Instead of HL, masking noise is discussed in terms of effective masking (dB EM), meaning that, for example, a 20-dB EM noise is that noise level that perceptually masks a 20-dB HL signal. The bandwidths for narrow bands of noise are specified by frequency with RETSPL corrections for third-octave and half-octave measurements. Cutoff values are given in the standard (see table 4 of the standard). When checking the bandwidth of the narrowband noise, it is necessary to have a frequency analyzer or spectrum analyzer (or a computer program that allows one to produce a Fourier analysis of the noise) to determine if the noise bandwidths from the audiometer conform to specifications. The same transducer that will be used when delivering the masking sound should be used to make final calibration measurements. However, because the characteristics of various transducers are quite different from one another, it is sensible to first do an electronic check directly from the audiometer to verify that any variation from the bandwidth is due to the transducer rather than the electrical output of the audiometer.

The masking sound should be checked periodically through the transducers used to present it. The examiner should be careful to use a signal that is high enough in level to avoid interference by ambient room noise (generally about 80 dB HL). In the case of narrowband noise, the SPL values measured should be within ±3 dB of the RETSPLs for the geometric center frequency and corrected appropriately for masker bandwidth. If broadband white noise (noise that has equal level across frequency) is the only masking signal on the audiometer, one need only check the output through the earphone with a linear setting (no filter) on the SLM. The overall output and attenuation characteristics should be checked in the same basic manner as described for puretones using an appropriate coupler.

When making noise measurements, the characteristics of the measuring equipment are critical. Since noise is not a "clean" (i.e., uniform and unvarying) signal, it is highly susceptible to errors of overshoot and undershoot on a meter and to damping on a graphic level recorder. A spectrum analyzer that is capable of frequency-domain averaging and with storage capabilities is optimal for checking calibration of noise. Unfortunately, most clinics do not have such sophisticated equipment.

COMPACT DISC AND TAPE PLAYERS

CD or tape players that are used in a clinic for reproducing speech signals, filtered environmental sounds, or other test

stimuli should be checked electroacoustically at least once every 12 months. However, if the CD or tape player is in regular use, weekly maintenance should be carried out (such as cleaning and demagnetizing the heads for tape players). The instruction manuals normally outline the procedures to be used with the particular tape or CD player. If not, any good audio equipment dealer can explain the procedure. In addition, the frequency response and time characteristics of the tape player should be checked.

At present, there are no standards for tape players used with audiometers. However, the frequency response and time characteristics of the tape player may be checked by using a standard commercial tape recording of puretones of various frequencies. If you do not have access to such a tape, it is possible to make one by introducing puretones from an audio oscillator into the machine, recording them, and playing them back. This enables the operator to check both the record and playback sections of the tape recorder. Unfortunately, if both the record and playback are equally reduced (or increased) in frequency, the output will appear at the nominal frequency. The output from the oscillator should be monitored with a voltmeter to make certain that a constant voltage signal is used. Distortion of the puretone from the tape player should also be checked. If none of this is possible, the speed of the tape player can be checked grossly by marking a tape and then, after timing a segment as it goes across the tape head, measuring to see how many inches passed over the heads per second. Also, if the machine is badly out of calibration, it will be audible as a pitch change in the recorded speech (higher if too fast, lower if too slow).

AUTOMATIC (AND COMPUTERIZED) AUDIOMETERS

A calibration check of automatic (or Bekesy) audiometers begins with frequency, level, cross-talk, and other aspects described for manual puretone audiometers. In addition, the attenuation rate and interruption rate for pulsed signals should be checked. ANSI S3.6-2010 requires that a rate of change of 2.5 dB/s be provided for Type 1, 2, and 3 audiometers. Permissible rates for all types of audiometers are given in the ANSI S3.6-2010 standard. As in manual audiometers, the permissible variance in level per step is 1 dB or 0.3 of the indicated step size, whichever is smaller. The attenuation rate may be measured quite easily with a stopwatch. After starting the motor, a pen marking on the chart is started at the same instant as a stopwatch is started. One reads the chart to determine how far the signal was attenuated (or increased) during the measured time interval. By dividing the duration (in seconds) into the decibel change in level, one can find the decibel per second attenuation rate. The audiometer should be checked for signals both increasing and decreasing in level.

To check the pulsed stimulus duration, one may go from the "scope sync" output on the back of the audiometer (if such exists) to an electronic counter, or if that is not available, one can record across the terminals of the timing mechanism inside the audiometer. It is difficult to check the pulse speed on a graphic level recorder because of pen damping, but it is possible to check it on a digital or storage oscilloscope. It is not difficult to estimate whether there is roughly a 50% duty cycle (on half the time and off half the time), but it is quite difficult to judge whether the signal is on for 200 ms versus 210 ms. The characteristics of the pulsed tone are described in section 7.5.4 of ANSI S3.6-2010.

If both pulsed and continuous signals are used, it is important to check the relative level of the pulsed and continuous signals. If they are not equal, this should be corrected. The relative levels can be compared by observing the envelope of the waveform on an oscilloscope or by recording the output with a graphic level recorder if there is no damping problem. The attenuation rate and pulse rate should be checked annually unless there is a reason to suspect a problem earlier.

Computerized audiometers are becoming commercially available. It should be noted that computerized audiometers, just like manual audiometers, must meet all of the technical specifications included in ANSI S3.6-2010.

AUDITORY-EVOKED POTENTIAL INSTRUMENTS

There is an IEC standard for auditory test signals of short duration for audiometric and neuro-otologic purposes (IEC 60645-3:2007). There is also an IEC standard for auditory brainstem response instruments (IEC 60645-7:2009 Electroacoustics—Audiometric Equipment—Part 7: Instruments for the measurement of auditory brainstem responses). ISO 389-6:2007 reports RETSPLs for clicks and standard (2–1–2 cycle) tonebursts. There is currently no ANSI standard that provides RETSPL for clicks and tonebursts.

The basic parameters of the acoustic signals used for auditory-evoked potentials (AEPs) are the same as for conventional audiometry. One must check output level, frequency, and time. When calibrating acoustic transients from an AEP instrument, the instrumentation used to calibrate an audiometer may be inappropriate. It is especially important to check the output from the AEP unit acoustically as well as electrically. It is easy to display the electrical output from the AEP unit on an oscilloscope, but to analyze that display, one needs to repeat it very rapidly or, preferably, use a digital or storage oscilloscope. Determination of the acoustic level of these acoustic transients requires an SLM that can record true peak SPL (pSPL) or that allows routing the output to an oscilloscope to determine pSPL or peak equivalent SPL (peSPL).

Calibration of Acoustic Transients

Acoustic transients must be calibrated utilizing specialized instrumentation and procedures. For clinical procedures,

it is prudent to use earphones that can be coupled to either a 6- or a 2-cc coupler. TDH-39s, TDH-49s, and TDH-50s housed in MX 41/AR or Telephonics type 51 cushions can be coupled to a 6-cc (NBS 9-A) coupler. Etymotic insert earphones (ER-1, ER-2, ER-3 A) can be coupled to a 2-cc coupler. At the base of a 2- or a 6-cc coupler is (typically) a space designed to house a 1″ condenser pressure microphone, but for some couplers, they are designed to house a ½″ microphone. The microphone output can be routed to either an SLM or to a conditioning amplifier that provides the polarization voltage for the condenser microphone and (in some instruments) provides voltage amplification (gain). The measurement of the SPL of an acoustic transient with an SLM is complicated by the time constants used for the SPL measurement. Most SLMs have at least two exponential-time–weighted averaging modes: fast and slow. Fast exponential-time–weighted averaging has a measurement time constant of 125 ms, whereas slow exponential-time–weighted averaging has a measurement time constant of 1,000 ms (Yeager and Marsh, 1998). In either case, clicks or tonebursts have durations that are much shorter than the time constant of even the fast exponential-time weighting, and you will underestimate the true SPL of the toneburst using fast (or worse yet slow) exponential-time–weighted averaging. There are several solutions to this measurement problem. **First**, if you can turn the toneburst on for several seconds, you can record the level of the tone in the fast or slow exponential-time–weighted averaging mode. If you measure over three time constants (375 ms in fast, 3,000 ms in slow), the recorded value will closely approximate the true exponential-time–weighted SPL of the stimulus. This is one method to obtain what is commonly referred to as the peSPL of the toneburst. This approach will not work for a click stimulus, as increasing the duration of the electrical pulse will alter the spectrum of the stimulus. **A second approach** for determining the level of an acoustic transient is to purchase an SLM that records the largest instantaneous pressure (the "peak" pressure) and "holds" this value in the display until the meter is reset. This peak-hold measurement may vary with the specific SLM, as the measurement interval over which this "peak" is evaluated (the time constant) varies with the particular SLM. It is desirable to use a meter with a pSPL time constant of several tens of microseconds, or less. **A third approach** is to use an oscilloscope and an SLM that has an analog (AC) output. This type of output enables you to route the analog voltage output of the microphone to the oscilloscope. This technique can be used with any transient, including a click, and this approach is another method to determine the peSPL. Figure 2.3 diagrams two procedures for determining click peSPL.

In the first procedure, referred to as the baseline-to-peak peSPL procedure, the click (in this case) or other transient is routed through the earphone/coupler/microphone/SLM to the oscilloscope. The click (or other transient) stimulus is presented and the baseline-to-peak voltage is measured on the oscilloscope. Making sure not to change the SPL range on the SLM, a tonal stimulus is routed through the earphone, and the level of the tone is adjusted until the baseline-to-peak voltage on the oscilloscope is identical to that measured for the click. The reading on the SLM is the baseline-to-peak peSPL of the click.

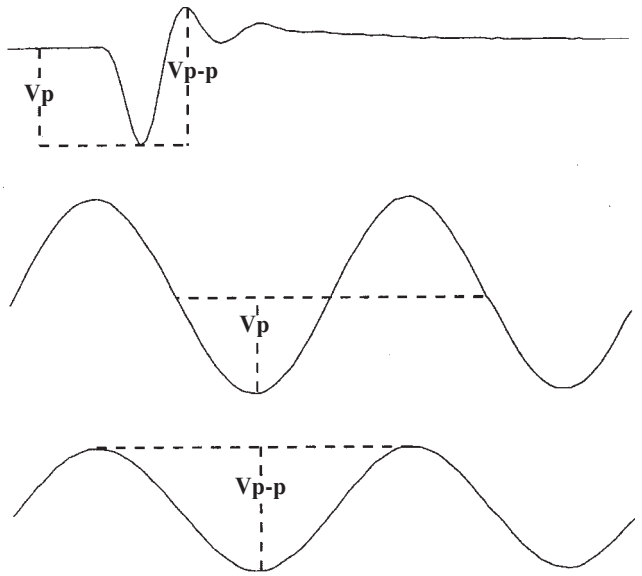


FIGURE 2.3 The procedure for obtaining peSPL (both baseline-to-peak and peak-to-peak measures) is shown. (Reprinted from Burkard R, Secor C. (2002) Overview of auditory evoked potentials. In: Katz J, ed. *Handbook of Clinical Audiology.* 5th ed. Baltimore, MD: Lippincott Williams & Wilkins; pp 233–248, with the permission of Lippincott Williams & Wilkins.)

The second method of determining peSPL involves measuring the peak-to-peak voltage of the click (or other transient) on the oscilloscope, and adjusting the sine wave until its peak-to-peak voltage is equal to the peak-to-peak voltage of the click. The SPL value displayed on the SLM is recorded as the peak-to-peak peSPL of the click (or other transient). The baseline-to-peak peSPL can never be less than the peak-to-peak peSPL. If the voltages of the positive and negative phases of a click (or other transient) are equal, then the baseline-to-peak and peak-to-peak peSPL values will be numerically equal. If the click is critically damped and shows a voltage deflection in only one direction (positive or negative), then the baseline-to-peak peSPL will be 6 dB larger than the peak-to-peak peSPL. As the baseline-to-peak peSPL and the peak-to-peak peSPL values can differ by as much as 6 dB, it is imperative that the measurement technique used when reporting peSPL is reported.

A fourth approach to measuring the level of a transient is to eliminate the SLM and to use a coupler, microphone, microphone conditioning amplifier and oscilloscope. The click (or other transient) stimulus is presented, and the peak

voltage on the oscilloscope is measured. The microphone is calibrated by using a sound source with a known SPL (i.e., a pistonphone or acoustic calibrator), or the microphone sensitivity curve can be used to convert microphone output voltage to input sound pressure (and ultimately to SPL). The acoustic calibrator is coupled to the microphone, and the voltage out of the microphone conditioning amplifier is measured. In this way, a given voltage is produced when a specified SPL is present at the microphone diaphragm. For example, an acoustic calibrator produces 114 dB SPL, and 1 V is measured at the conditioning amplifier output. Then, a 2-cc coupler is placed on the microphone. An Etymotic ER-3 A insert microphone is then mated to the coupler. A sine wave is presented through the earphone, and 50 mv is measured. The SPL is

$$20 \log (50 \text{ mv}/1{,}000 \text{ mv}) + 114 \text{ dB SPL} = 88 \text{ dB SPL}$$

The first part of the formula estimates the dB re: 114 dB SPL, which is −26 dB; 114 is added to convert from "dB re: 114 dB SPL" to "dB SPL."

Using the sensitivity curve of the microphone, the peak voltage on the oscilloscope is converted to peak pressure (e.g., in Pascals). The sensitivity of a microphone relates the voltage out of the microphone to the sound pressure at the microphone diaphragm, in for example, millivolts per pascal. Then the pressure is converted to SPL. For example, a peak voltage of 100 mV is measured. The microphone sensitivity is 50 mV/Pa. If 50 mV represents 1 Pa, then 100 mV represents 2 Pa. Converting to pSPL,

$$\text{pSPL} = 20 \log (2 \text{ Pa}/0.00002 \text{ Pa}) = 20 \log 10{,}000$$
$$= \sim 100 \text{ dB pSPL}$$

This value should correspond to the pSPL produced by an SLM in peak-hold mode, although if the time constant of the SLM is too long (say 100 µs), then the SLM may produce a lower value. For a given stimulus, the baseline-to-peak peSPL value should be 3.01 dB less than the true pSPL value. This is because the peSPL value is actually referenced to a RMS measure of a sine wave, rather than a peak measure. To obtain the true pSPL value, 3.01 dB must be added to the peSPL (using the baseline-to-peak procedure), because the crest factor (ratio of peak to RMS) of a true sine wave is 1.414, or 3.01 dB (3.01 dB = 20 log 1.414).

In addition to the overall level, it is important to determine the frequency characteristics of the signal (i.e., its spectrum) as it is played through the transducer. The acoustic spectrum of a signal is not necessarily identical to the spectrum of the electrical signal. This is because each system has its own transfer function (i.e., filtering characteristics), and the acoustic spectrum of the stimulus will be affected by both the earphone and the coupler used to mate the earphone to the SLM microphone. The spectrum of the signal can be measured by routing the acoustic signal through a coupler, condenser microphone, and microphone conditioning/preamplifier or SLM. Finally, the output of the microphone conditioning/amplifier or SLM is routed to a spectrum analyzer or to an analog-to-digital converter to a computer that is programmed to do a Fourier analysis of the signal. In each case, a display of the spectrum of each signal type can be obtained. NOTE: Most clinics will likely not have the instrumentation or perhaps the expertise to do the calibration of transient stimuli. These calibration guidelines will thus be most useful to the manufacturers and to the technician who periodically calibrates the AEP instrumentation.

Many AEPs are elicited by presentation of brief acoustic transients. The two most commonly used transients are clicks and tonebursts. A click is produced by exciting a transducer with a brief-duration electrical pulse. For use with human subjects, a common click duration is 100 µs. In a pulse with duration *d,* there are spectral zeroes (energy at that frequency dips toward zero) at frequencies that are integer multiples of 1/*d.* Thus, for an electrical pulse with a duration of 100 µs, the first spectral zero occurs at 10,000 Hz, with spectral zeroes occurring at 20,000, 30,000, 40,000 Hz, and so on. The acoustic representation of this 100-µs pulse, as recorded through a TDH-50 earphone in a 6-cc coupler, is shown in Figure 2.4. It is important for the reader to understand that the click has energy over a wide range of frequencies, and thus is a broadband stimulus. It is also important to understand that the spectrum of a transient (indeed, of any signal) can and is influenced by not only the earphone, but also the coupler (and perhaps the microphone) used in the calibration process.

Clicks are often the stimuli of choice when using the auditory brainstem response (ABR) for hearing screening, site-of-lesion testing, and intraoperative monitoring. In contrast, when interested in obtaining an electrophysiological estimate of the behavioral audiogram, then a broadband stimulus is not optimal. Several approaches have been used to elicit responses from a limited cochlear region. The various approaches can be broken down into two strategies: (1) using stimuli with narrow spectra and/or (2) using masking procedures to eliminate the contribution of specific regions of the cochlea. The latter approach (the use of masking procedures) goes beyond the scope of the present chapter, but is considered later in this book (Chapter 11: Introduction to auditory-evoked potentials).

One approach to generating a limited-spectrum stimulus is to present a sine wave for a brief duration. ANSI S3.6-2010, which reviews technical specifications for audiometers, states that for audiometric purposes a tone must be presented for a duration of not less than 200 ms, and have a rise time and fall time ranging between 25 and 100 ms. Figure 2.5 (upper panel) shows a toneburst with a carrier frequency of 2,000 Hz. The time required for the toneburst envelope to increase from zero to its maximal amplitude is termed its rise time. The time that the toneburst envelope remains at this maximal amplitude is called its plateau time. The time required for the toneburst envelope to

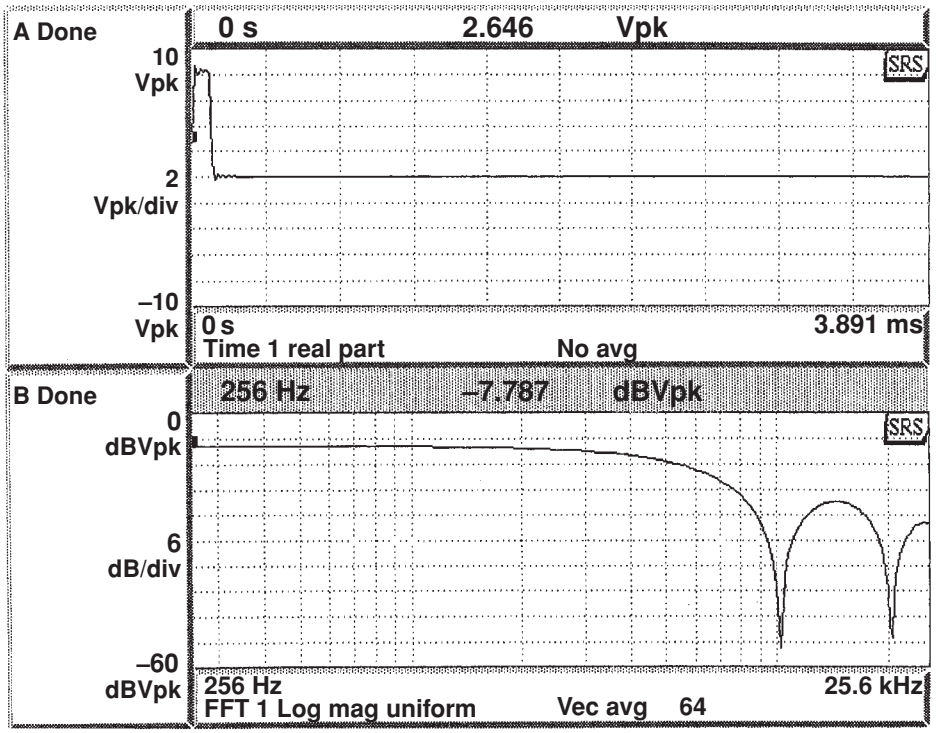


FIGURE 2.4 Time-domain waveform (**upper panel**) and spectrum (**lower panel**) of an electrical pulse with a duration of 100 μs. (Reprinted from Burkard R, Secor C. (2002) Overview of auditory evoked potentials. In: Katz J, ed. *Handbook of Clinical Audiology.* 5th ed. Baltimore, MD: Lippincott Williams & Wilkins; pp 233–248, with the permission of Lippincott Williams & Wilkins.)

decrease from its maximal amplitude to zero amplitude is called its fall time. Figure 2.5 (lower panel) shows the amplitude spectrum (i.e., frequency-domain representation–amplitude across frequency) of this toneburst. A tone that is infinitely long in duration only has energy at the carrier frequency (i.e., the frequency of the sine wave). For the toneburst, there is significant energy over a range of frequencies centered at 2,000 Hz. This spread of energy to frequencies above and below the carrier frequency is referred to as spectral splatter. Acoustic transients are used to elicit

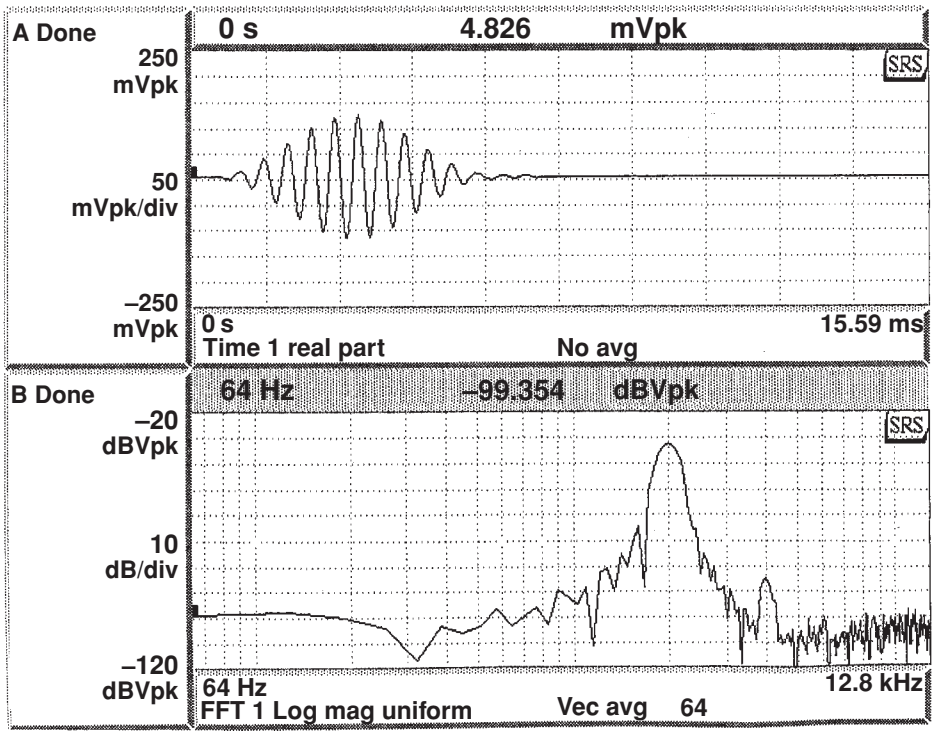


FIGURE 2.5 **Upper panel:** The time-domain representation of a toneburst. **Lower panel:** The frequency-domain representation of the same toneburst. (Reprinted from Burkard R, Secor C. (2002) Overview of auditory evoked potentials. In: Katz J, ed. *Handbook of Clinical Audiology.* 5th ed. Baltimore, MD: Lippincott Williams & Wilkins; pp 233–248, with the permission of Lippincott Williams & Wilkins.)

AEPs, because many of these AEPs, such as the ABR, are onset responses. Onset responses are elicited by the first few milliseconds of the stimulus onset, and hence are primarily affected by the stimulus rise time. When using toneburst stimuli, it is intended to serve as a compromise between an audiometric tone (long duration, long rise/fall times, and a very narrow spectrum) and a click (short duration, very fast rise/fall times, and very broad spectrum). To add even more complexity to this topic, there are many unique gating functions that can be used to shape the onset and offset of the toneburst, including linear, Blackman, and Hanning (cosine^2) functions. As mentioned previously, methods for the calibration of acoustic transients can be found in IEC 60645-3:2007, and RETSPLs for clicks and select toneburst stimuli can be found in ISO 389-6:2007.

OTOACOUSTIC EMISSION DEVICES

There are currently no ANSI standards for otoacoustic emission (OAE) devices. IEC 60645-6 is an international standard that can be used to specify the stimuli used to obtain OAEs. If using a click to obtain transient-evoked otoacoustic emissions (TEOAEs), reporting the level in pSPL or peSPL is appropriate. For distortion product otoacoustic emissions (DPOAEs), verification that the primary signals (as measured in an appropriate coupler/microphone/SLM and frequency counter or spectrum analyzer) are close to the levels and frequencies as specified by the OAE device is desirable. Because the DPOAE response is an intermodulation distortion product (typically the cubic difference tone), it is critical to measure the amplitude of the distortion at this frequency in a hard-walled cavity to know when measured distortion in fact represents distortion in the instrumentation itself, rather than representing nonlinearities generated by the inner ear.

ACOUSTIC IMMITTANCE DEVICES

The standard for acoustic immittance (impedance/admittance) devices is ANSI S3.39-1987. Note that there is also an IEC standard, IEC 60645-5:2004, for measurement of aural acoustic impedance/admittance. ANSI S3.39-1987 describes four types of units for measuring acoustic immittance (listed simply as Types 1, 2, 3, and 4). The specific minimum mandatory requirements are given for Types 1, 2, and 3. There are no minimum requirements for the Type 4 device. Types 1, 2, and 3 must have at least a 226-Hz probe signal, a pneumatic system (manual or automatic), and a way of measuring static acoustic immittance, tympanometry, and the acoustic reflex. Thus, to check the acoustic immittance device, one may begin by using a frequency counter to determine the frequency of the probe signal(s). The frequency should be within 3% of the nominal value. The total harmonic distortion shall not exceed 5% of the fundamental frequency level when measured in an HA-1–type coupler. The probe signal shall not exceed 90 dB SPL as measured in that coupler, in an attempt to minimize the possibility that the probe signal will elicit an acoustic reflex. The range of acoustic admittance and acoustic impedance values that should be measurable varies by instrument type. The accuracy of the acoustic immittance measurements should be within 5% of the indicated value or $\pm 10^{-9}$ cm^3/Pa (0.1 acoustic mmho), whichever is greater. The accuracy of the acoustic immittance measurement can be determined by connecting the probe to the test cavities and checking the accuracy of the output at specified temperatures and ambient barometric pressures. A procedure for checking the temporal characteristics of the acoustic immittance instrument is described by Popelka and Dubno (1978) and by Lilly (1984).

Air pressure may be measured by connecting the probe to a "U tube" manometer and then determining the water displacement as the immittance device air pressure dial is rotated. If the SI unit of decapascals (daPa) is used, then an appropriate manometer or pressure gauge must also be used. The air pressure should not differ from that stated on the device (i.e., 200 daPa) by more than ±10 daPa or ±15% of the reading, whichever is greater. The standard states that the air pressure should be measured in cavities with volumes of 0.5 to 2 cm^3.

Finally, the reflex-activating system should be checked. In checking the activation of a contralateral or ipsilateral reflex, normally an insert receiver will be used that may be measured on a standard HA-1 coupler. The frequency of the activator can be measured electrically directly from the acoustic immittance device. In this case, one uses a frequency counter as described earlier for checking the frequency of puretones in audiometers. Frequency should be ±3% of the stated value, and harmonic distortion should be less than 3% at specified frequencies for earphones and 5% or less for the probe tube transducer or insert receiver. Noise bands should also be checked if they are to be used as activating stimuli. Broadband noises should be uniform within ±5 dB for the range between 250 and 6,000 Hz for supra-aural earphones. This can be checked by sending the output through the transducer connected to a coupler, a microphone, and a graphic level recorder or spectrum analyzer. The SPL of tonal activators should be within ±3 dB of the stated value for frequencies from 250 to 4,000 Hz and within ±5 dB for frequencies of 6,000 to 8,000 Hz and for noise. The rise and fall times should be the same as those described for audiometers and may be measured in the same way. Daily listening checks as well as periodic tests of one or two persons with known acoustic immittance to check tympanograms and acoustic reflex thresholds should be performed to catch any gross problems.

In summary, acoustic immittance devices should be checked as carefully as one's puretone audiometer. Failure

to do so can lead to variability in measurement, which may invalidate the immittance measurement.

TEST ROOM STANDARDS

It is insufficient to limit the periodic calibration checks to the audiometric equipment. The environment in which the test is to be carried out must also be evaluated. ANSI S3.1-1999 provides criteria for permissible ambient noise during audiometric testing. Section 11 of ISO 8253-1:2010 also specifies appropriate ambient noise levels. The ambient level in the test room is checked by using an SLM that is sensitive enough to allow testing to levels as low as 8 dB SPL. Many modern SLMs can measure to levels of 5 dB SPL or less. One should place the SLM (preferably using a free-field microphone) in the place where the subject is to be seated. The doors of the test room should be closed when making the measurements. If one plans to use monitored live voice testing, the ambient levels in the examiner's room should also be checked. However, there are no standards concerning acceptable noise levels in the examiner's room. ANSI S3.1-1999 provides acceptable ambient noise values for threshold estimation at 0 dB HL, for one- and third-octave bandwidths, for use with supra-aural and insert earphones, and for free-field (or bone conduction) testing. These values vary with the range of audiometric frequencies investigated. If the level reported by ANSI S3.1-1999 is exceeded, then the minimum dB HL value that can be recorded is increased from 0 dB HL. This is (more or less) a linear function, so if the accepted ambient noise level in a given band is exceeded by 5 dB, then the minimum dB HL value that you can measure is increased to 5 dB HL.

CONCLUSIONS

This chapter has emphasized that the audiologist must, on a daily basis, listen to the output of the equipment. There are many problems that can be detected by a trained human ear. However, the listener is simply not good enough to check the auditory equipment with the precision that is needed to ensure that it is working properly. Thus, it has also been stressed that, to determine the precise characteristics of the equipment, routine electroacoustic checks must be carried out. Even when there are no current standards (i.e., there is, at the time this chapter is being written, no ANSI standard specifying the RETSPL of transients), one should at least check the stability of one's equipment. Because the test results that one obtains are no more accurate than the equipment on which they are performed, both clinical and calibration equipment must be chosen and maintained with care. The ultimate responsibility for the accuracy of the test results lies with the audiologist. Therefore, the audiologist must make sure that the equipment is working properly by carrying out routine calibration checks.

FOOD FOR THOUGHT

1. Do you think it is more important for standards to tell us (a) how to *optimally* characterize our stimuli or (b) how to *be consistent* in how we characterize our stimuli? What are your reasons? If you believe that they are equally important, please state your reasons for that point of view.
2. Describe the advantages and disadvantages of having a formal electroacoustic characterization of your audiometric equipment every 3 months versus annually.
3. State why you agree or disagree with the following statement: All Au.D. students should learn (both in lecture and in hands-on laboratories) how to calibrate all audiometric equipment that they use in the clinic.

REFERENCES

American Medical Association. (1951) Specifications of the Council of Physical Medicine and Rehabilitation of the American Medical Association. *J Am Med Assoc.* 146, 255–257.

American National Standards Institute. (2004) About ANSI overview. Available at: http://www.ansi.org/about_ansi/overview/overview.aspx?menuid=1 (accessed July 13, 2013).

American Speech-Language-Hearing Association. (1991) *Sound Field Measurement Tutorial.* Rockville, MD: American Speech-Language-Hearing Association.

Beranek LL. (1988) *Acoustical Measurements.* New York, NY: American Institute of Physics.

Burkhard MD. (1978) *Manikin Measurements–Conference Proceedings.* Elk Grove Village, IL: Industrial Research Products.

Burkhard MD, Corliss ELR. (1954) The response of earphones in ears and couplers. *J Acoust Soc Am.* 26, 679–685.

Burkhard MD, Sachs RM. (1975) Anthropometric manikin for acoustic research. *J Acoustic Soc Am.* 58, 214–222.

Corliss ELR, Burkhard MD. (1953) A probe tube method for the transfer of threshold standard between audiometer earphones. *J Acoust Soc Am.* 25, 990–993.

Cox R. (1986) NBS-9 A coupler-to-eardrum transformation: TDH-39 and TDH-49 earphones. *J Acoust Soc Am.* 79, 120–123.

Decker TN, Carrell TD. (2004) *Instrumentation: An Introduction for Students in the Speech and Hearing Sciences.* Mahwah, NJ: Lawrence Erlbaum Associates.

Harris C. (1998) *Handbook of Acoustical Measurements and Noise Control.* 3rd ed. Woodbury, NY: Acoustical Society of America.

Hawkins DB, Cooper WA, Thompson DJ. (1990) Comparisons among SPLs in real ears, 2 cm^3 and 6 cm^3 couplers. *J Am Acad Audiol.* 1, 154–161.

Hawley MS. (1939) An artificial mastoid for audiophone measurements. *Bell Lab Rec.* 18, 73–75.

Johnson D, Marsh A, Harris C. (1998) Acoustic measurement instruments. In: Harris C, ed. *Handbook of Acoustical Measurements and Noise Control.* 3rd ed. Woodbury, NY: Acoustical Society of America; pp 5.1–5.21.

Killion MD. (1978) Revised estimate of minimum audible pressure: where is the "missing 6 dB?" *J Acoust Soc Am.* 63, 1501–1508.

Lilly DJ. (1984) Evaluation of the response time of acoustic-immittance instruments. In: Silman S, ed. *The Acoustic Reflex.* New York, NY: Academic Press.

Melnick W. (1973) What is the American National Standards Institute? *ASHA.* 10, 418–421.

Occupational Safety and Health Administration. (1983) *Occupational Noise Exposure, Hearing Conservation Amendment. Rule and Proposed Regulation.* Washington, DC: Federal Register, United States Government Printing Office.

Popelka GR, Dubno JR. (1978) Comments on the acoustic-reflex response for bone-conducted signals. *Acta Otolaryngol (Stockh).* 86, 64–70.

Roach R, Carhart R. (1956) A clinical method for calibrating the bone-conduction audiometer. *Arch Otolaryngol.* 63, 270–278.

Rosen S, Howell P. (1991) *Signals and Systems for Speech and Hearing.* London: Academic Press.

Rudmose W. (1964) Concerning the problem of calibrating TDH-39 earphones at 6 kHz with a 9 A coupler. *J Acoust Soc Am.* 36, 1049.

Silverman FH. (1999) *Fundamentals of Electronics for Speech-Language Pathologists and Audiologists.* New York, NY: Allyn and Bacon.

Speaks C. (1996) *Introduction to Sound. Acoustics for the Hearing and Speech Sciences.* 2nd ed. San Diego, CA: Singular Publishing.

Studebaker G. (1967) Interest variability and the air-bone gap. *J Speech Hear Disord.* 32, 82–86.

Weiss E. (1960) An air-damped artificial mastoid. *J Acoust Soc Am.* 32, 1582–1588.

Weissler P. (1968) International standard reference zero for audiometers. *J Acoust Soc Am.* 44, 264–275.

Wilber LA. (2004) *What Are Standards and Why Do I care? Seminars in Hearing—Current Topics in Audiology: A Tribute to Tom Tillman.* Stuttgart, Germany: Thieme; pp 81–92.

Wilber LA, Goodhill V. (1967) Real ear versus "artificial mastoid" methods of calibration of bone-conduction vibrators. *J Speech Hear Res.* 10, 405–416.

Yeager D, Marsh A. (1998) Sound levels and their measurement. In: Harris C, ed. *Handbook of Acoustical Measurements and Noise Control.* 3rd ed. Woodbury, NY: Acoustical Society of America; pp 11.1–11.18.

Zwislocki JJ. (1970) *An Acoustic Coupler for Earphone Calibration.* Rep. LSC-S-7, Lab Sensory Commun. Syracuse, NY: Syracuse University.

Zwislocki JJ. (1971) *An Ear-Like Coupler for Earphone Calibration.* Rep. LSC-S-9, Lab Sensory Commun. Syracuse, NY: Syracuse University.

Zwislocki JJ. (1980) An ear simulator for acoustic measurements. Rationale, principles, and limitations. In: Studebaker G, Hochberg I, eds. *Acoustical Factors Affecting Hearing Aid Performance.* Baltimore, MD: University Park Press.

CHAPTER 3

Puretone Evaluation

Robert S. Schlauch and Peggy Nelson

INTRODUCTION

Most people who attend primary school in the United States and in other industrialized nations experience puretone* testing firsthand as a method to screen for hearing loss. Puretone threshold testing is seen in films, such as Woody Allen's award-winning movie *Hannah and Her Sisters* or the film *Wind Talkers.* These casual experiences with audiology may give lay people the false impression that audiology is a narrow profession.

Most audiologists would likely agree that puretone (PT) thresholds represent a key component of the assessment battery. Proper administration and interpretation of PT threshold tests require considerable knowledge, as it is not always simple and straightforward. The goal of this chapter is to introduce readers to the complexity of PT threshold testing, as well as to provide clinicians with a reference for clinical applications.

WHAT ARE PURETONES AND HOW ARE THEY SPECIFIED?

PT thresholds represent the lowest level of response to a tonal stimulus. Puretones are the simplest of sounds described by their frequency, amplitude, phase, and duration. The most important of these characteristics for puretone audiometry are frequency and amplitude (or intensity level).

Puretone frequency is perceived as pitch, the characteristic of sound that determines its position on a musical scale. Young people with normal hearing are able to perceive frequencies between 20 and 20,000 Hz. Human hearing is more sensitive (better) in the range of frequencies between 500 and 8,000 Hz than it is at either extreme of the audible range of frequencies. Conventional puretone audiometry typically assesses thresholds for frequencies between 250 (or 125) and 8,000 Hz. The frequency range for conventional audiometry is very similar to the range of frequencies (100 to 6,000 Hz) that is important for speech understanding (French and Steinberg, 1947).

Puretone amplitude or level is usually quantified in decibels. Decibels (dB) represent the logarithm of a ratio of two values; the term is meaningless without a reference. Two commonly used decibel scales are sound pressure level (SPL) and hearing level (HL). The reference level for dB SPL is 20 μPa, a pressure value. This reference value for SPL was selected to correspond to the faintest pressure that is audible in the frequency region where hearing is most sensitive. The frequency is not specified in the reference level for dB SPL; all sounds expressed in units of dB SPL share the same reference of 20 μPa. The SPL scale is frequently used in audiology to compare the level of speech or other sounds at different frequencies. Such comparisons are critical for prescribing and evaluating hearing aids. HL, a second decibel scale, is used to plot an audiogram, the accepted clinical representation of puretone thresholds as a function of frequency. The reference for dB HL is the median threshold for a particular frequency for young adults with no history of ear problems. Unlike dB SPL, the zero reference level for dB HL varies with frequency, because humans have more sensitive hearing at some frequencies than others. Because the reference is normal human hearing, thresholds that deviate from 0 dB HL at any frequency show how much one's hearing deviates from this normal value.

Figure 3.1 illustrates thresholds displayed in dB SPL and dB HL. The left panel shows hearing thresholds plotted in dB SPL as a function of frequency. Thresholds plotted in this way constitute a minimum audibility curve. The right panel shows a conventional audiogram plotted in dB HL. Note that on the dB SPL scale, larger decibel values are plotted higher on the graph. By contrast, larger values in dB HL are plotted lower on the audiogram. To illustrate the relationship between dB SPL and dB HL, the reference values for 0 dB HL (average normal hearing) for a specific earphone are plotted in dB SPL as a solid line. Illustrated with a dashed line on these same two figures are the thresholds for a person with a high-frequency hearing loss. Note in the figure on the left that the separation between the solid line and the dashed line represents values for dB HL on the audiogram.

*The use of the compound noun "puretone" is the editor's choice for consistency purposes.

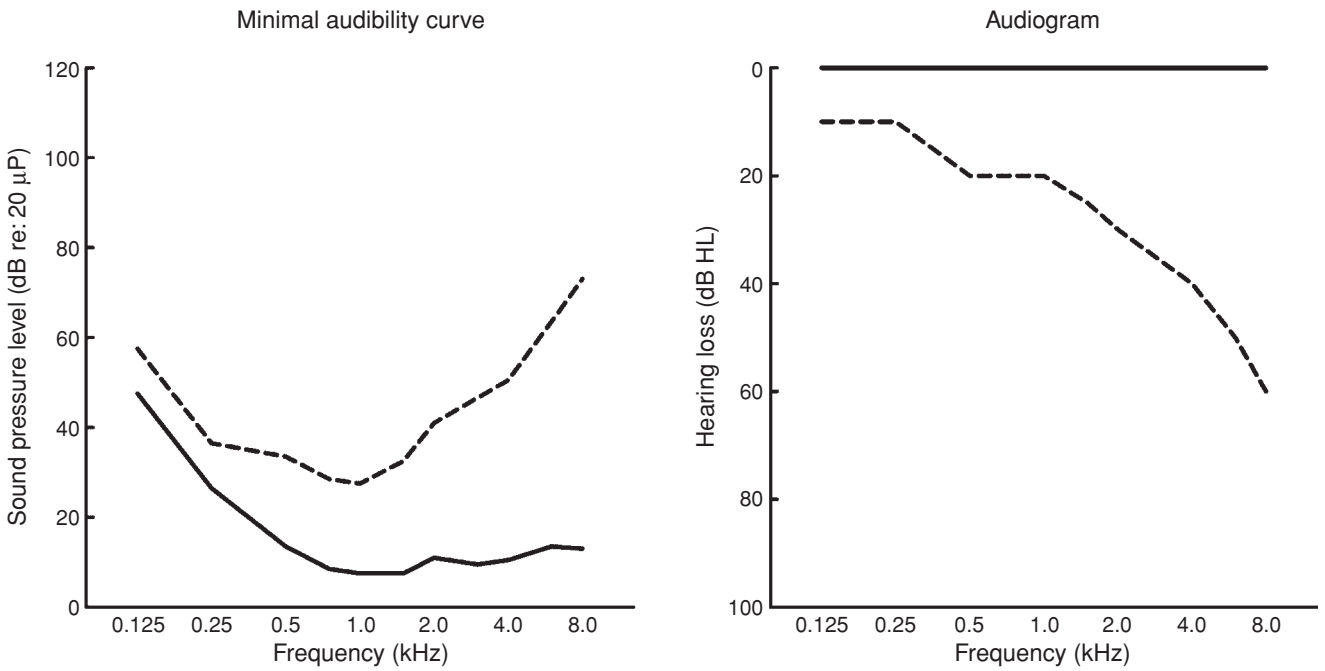


FIGURE 3.1 Thresholds in dB sound pressure level (SPL; **left panel**) and dB hearing level (HL; **right panel**) as a function of frequency. The *solid line* represents average normal hearing; the *dashed line* represents a person's threshold who has a high-frequency hearing loss.

WHY PURETONE THRESHOLDS?

The reader might be wondering why audiologists use puretones at specific frequencies when the most meaningful stimulus is speech. Two important reasons are that PT thresholds provide information about the type of hearing loss, as well as quantify frequency-specific threshold elevations that result from damage to the auditory system.

PT thresholds provide quantification of amount of loss due to problems with the outer and middle ear (the conductive system) separately from the cochlea and the auditory nerve (the sensory/neural system). This distinction helps in the diagnosis and guides audiologists and physicians with important details for providing treatment strategies.

Damage to the auditory system often results in a loss of sensitivity that is frequency specific. For instance, changes in the stiffness and mass properties of the middle ear affect the relative amount of loss in the low and high frequencies (Johanson, 1948). For air-conduction thresholds, an increase in stiffness results in a greater low-frequency loss, whereas an increase in mass results in a greater loss in the high frequencies. Thresholds for puretones (or other narrowband sounds) also provide us with diagnostic information about the integrity of different channels in the sensory/neural pathway. The auditory system is organized tonotopically (i.e., a frequency-to-place mapping) from the cochlea to the cortex. The tonotopic organization of the cochlea is a result of the frequency tuning of the basilar membrane, with high frequencies represented at the basal end and low frequencies at the apical end. Damage to sensory cells of the cochlea at a specific place along the basilar membrane can result in a loss of hearing that corresponds to the frequencies coded by that place. For this reason, PT threshold tests provide details that would otherwise remain unknown if a broadband stimulus such as speech were used.

In addition to providing audiologists with critical diagnostic information about the amount and type of loss, PT thresholds find applications (1) for estimating the degree of handicap, (2) as a baseline measure for hearing conservation programs, (3) for monitoring changes in hearing following treatment or progression of a disease process, (4) for screening for hearing loss, (5) for determining candidacy for a hearing aid or a cochlear implant, and (6) for selecting the frequency-gain characteristics of a hearing aid. PT thresholds also provide a reference level for presentation of suprathreshold speech testing and for the meaningful interpretation of other audiologic tests, such as evoked otoacoustic emissions and acoustic reflex thresholds. PT thresholds are also used to assess the functional attenuation of hearing protection devices.

TUNING FORK TESTS

A struck tuning fork produces a sustained puretone that decays in level over time. Unlike an audiometer, tuning forks cannot present a calibrated signal level to a listener's ear. Despite this shortcoming, tuning fork tests provide qualitative information that can help determine whether a

hearing loss is conductive or sensory/neural. Tuning fork tests are promoted by some as an important supplement to puretone audiometry. In a recently published book, otologists are advised to include tuning fork tests as an integral part of the physical examination for conductive hearing loss (Torres and Backous, 2010).

The two best known tuning fork tests are the Weber and Rinne. Judgments about the type of hearing loss are made by comparing the pattern of results on both tests. Air conduction (AC) is tested by holding the tuning fork at the opening of the ear canal, and bone conduction (BC) is tested by placing the tuning fork on the mastoid process (the bony area behind the pinna) or on the forehead or incisors (British Society of Audiology, 1987). For the Weber test, a client judges whether sound is perceived in one or both ears when the tuning fork is placed on the forehead. For the Rinne test, the client judges whether sound is louder when presented by AC or by BC. Ideally, conductive hearing losses produce a pattern of responses that is uniquely different from the one for sensory/neural hearing losses. In the Weber, the sound is lateralized to the poorer ear with a conductive loss and to the better ear for a sensory/neural loss. In the Rinne, the sound is louder by BC in a conductive loss and by AC with a sensory/neural loss.

Some recommend tuning fork tests to check the validity of audiograms (Gabbard and Uhler, 2005) or to confirm the audiogram before conducting ear surgery (Sheehy et al., 1971). However, it is important to recognize that tuning fork tests administered to people with known conductive losses have shown that these procedures are often inaccurate (Browning, 1987; Snyder, 1989). Although only about 5% of people with normal hearing or sensory/neural losses are falsely identified as having conductive losses with the Rinne test, this test misses many people with significant conductive losses (Browning, 1987), including 50% of losses that have 20-dB air–bone gaps. The Weber test fares equally poorly. Browning (1987) reports that a majority of children with conductive losses give inappropriate responses on the Weber test. From these and other studies, one must conclude that tuning fork tests are not a replacement or even a supplement to audiometry. Audiometry is capable of identifying nearly 100% of air–bone gaps, as small as 15 dB.

PURETONE AUDIOMETRY

Audiometers are used to make quantitative measures of AC and BC PT thresholds. AC thresholds assess the entire auditory pathway and are usually measured using earphones. When sound is delivered by an earphone, the hearing sensitivity can be assessed in each ear separately. BC thresholds are measured by placing a vibrator on the skull, with each ear assessed separately, usually by applying masking noise to the nontest ear. The goal of BC testing is to stimulate the cochlea directly, thus bypassing the outer and middle ears. A comparison of AC and BC thresholds provides separate estimates of the status of the conductive and sensory/neural systems. If thresholds are elevated equally for sounds presented by AC and BC, then the outer and middle ear are not contributing to a hearing loss. By contrast, if thresholds are poorer by AC than by BC, then the source of at least some of the loss is the outer or middle ear. Figure 3.2 illustrates the AC and BC pathways and how hearing thresholds are typically affected by damage to these structures. See Chapter 4 for a complete review of BC assessment.

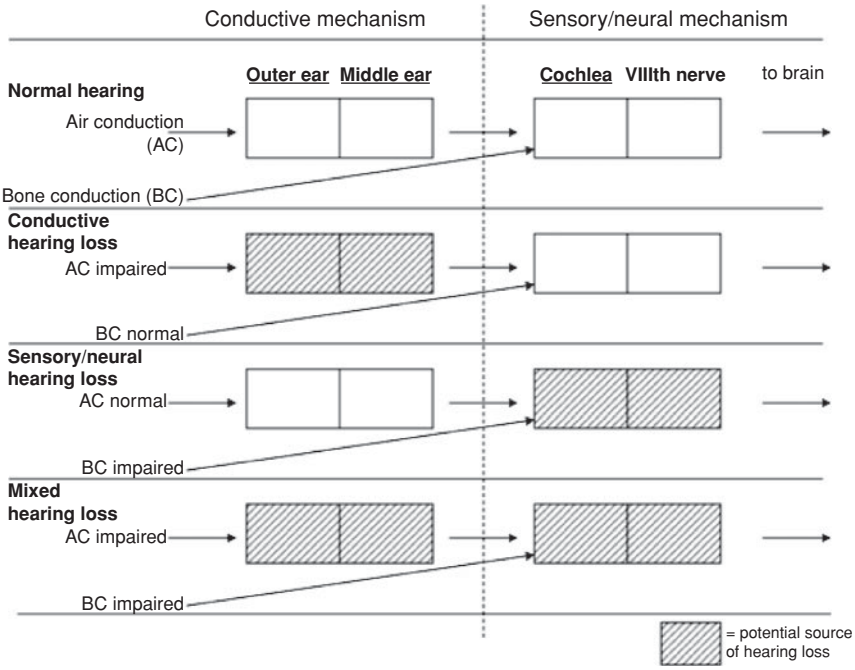


FIGURE 3.2 Conductive and sensory/neural pathways. [Adapted from Martin (1994)]

Equipment

AUDIOMETERS

Puretones are generated within an audiometer. Audiometers have the ability to select tonal frequency and intensity level and to route tones to the left or right earphone. All audiometers also have an interrupter switch that presents the stimulus to the examinee. The American National Standards Institute (ANSI) Specification for Audiometers (ANSI, 2010) describes four types of audiometers, with Type 1 having the most features and Type 4 having the fewest features. A Type 1 audiometer is a full-featured diagnostic audiometer. A Type 1 audiometer has earphones, bone vibrator, loud speakers, masking noise, and other features. A Type 4 audiometer is simply a screening device with earphones, but none of the other special features.

Type 1 (full-featured, diagnostic audiometer) has the ability to assess puretone AC thresholds for frequencies ranging from 125 to 8,000 Hz and BC thresholds for frequencies ranging from 250 to 6,000 Hz. If an audiometer has extended high-frequency capability, air-conduction thresholds can be extended to 16,000 Hz. Maximum output levels for AC testing are as high as 120 dB HL for frequencies where hearing thresholds are most sensitive. By contrast, distortion produced by bone oscillators at high intensities limits maximum output levels for BC thresholds to values nearly 50 dB lower than those for AC thresholds for the same frequency.

TRANSDUCERS

Earphones

Earphones are generally used to test puretone AC thresholds. A pair of supra-aural earphones is illustrated in Figure 3.3. For decades, supra-aural earphones, ones in which the cushion rests on the pinna, were the only choice for clinical audiology. The popularity of supra-aural phones was mainly due to their ease of calibration and the lack of other types of commercially available earphones. In the past few years, insert earphones and circumaural earphones have become available and provide some useful applications for puretone assessment.

FIGURE 3.3 Telephonics model TDH-49, an example of supra-aural earphones.

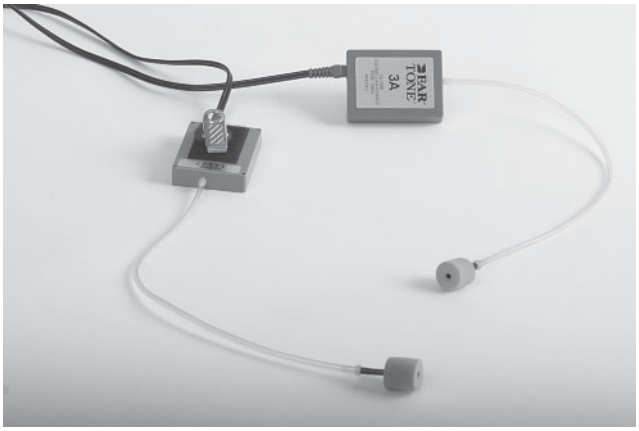


FIGURE 3.4 Etymotic model ER-3A insert earphones.

Insert earphones are coupled to the ear by placing a probe tip, typically a foam plug, into the ear canal. The commercially available model that has a standardized calibration method for audiology is the Etymotic model ER-3A, which is illustrated in Figure 3.4. These earphones have gained popularity in the past few years because they offer distinct advantages over supra-aural earphones. One major advantage is that insert earphones yield higher levels of interaural attenuation than supra-aural earphones (Killion and Villchur, 1989). Interaural attenuation represents the decibel reduction of a sound as it crosses the head from the test ear to the nontest ear. The average increase in interaural attenuation is roughly 20 dB. This reduces the need for masking the nontest ear and decreases the number of masking dilemmas, situations for which thresholds cannot be assessed, because the presentation level of the masking noise is possibly too high. (See Chapter 6 for a comprehensive review of masking.) Another important advantage of insert earphones over supra-aural earphones is lower test–retest variability for thresholds obtained at 6 and 8 kHz; variability for other frequencies is comparable. Given that thresholds for 6 and 8 kHz are important for documenting changes in hearing due to noise exposure and for identifying acoustic tumors, lower variability should increase the diagnostic precision. A third advantage that insert earphones offer is elimination of collapsed ear canals (Killion and Villchur, 1989). In about 4% of clients, supra-aural earphones cause the ear canal to narrow or be closed off entirely when the cushion presses against the pinna, collapsing the ear canal (Lynne, 1969), resulting in false hearing thresholds, usually in the high frequencies (Figure 3.5) (Ventry et al., 1961). Because insert earphones keep the ear canal open, collapsed canals are eliminated. A fourth advantage of insert earphones is that they can be easily used with infants and toddlers who

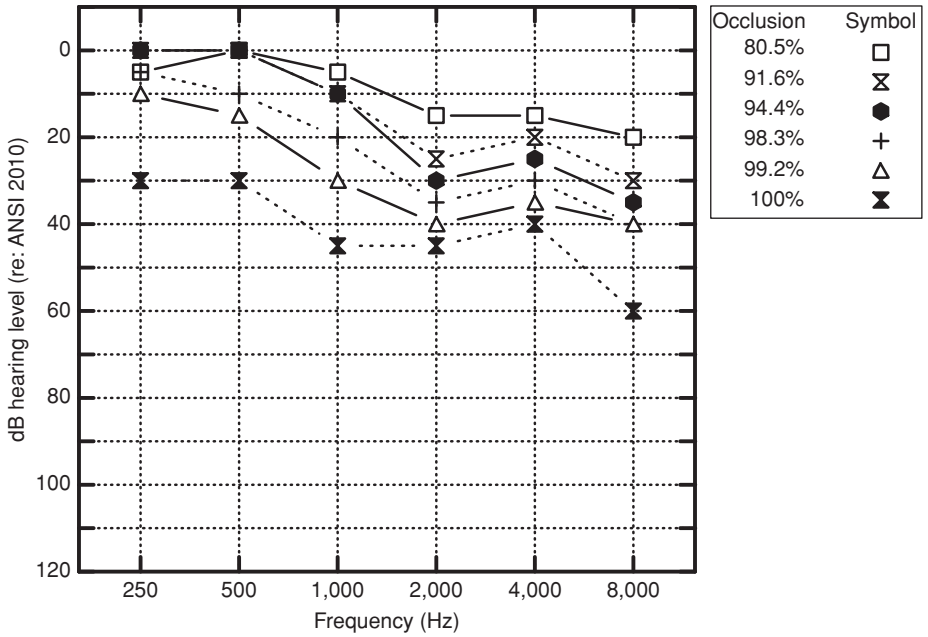


FIGURE 3.5 Air-conduction (AC) thresholds (in dB hearing level [HL]) for different percentages of ear canal occlusion. One hundred percent indicates that the ear canal is completely occluded. Deviations from 0 dB HL represent the loss due to occlusion. (Adapted from Chandler (1964))

cannot or will not tolerate supra-aural earphones. A fifth advantage of insert earphones is the option of conducting middle-ear testing and otoacoustic emission testing without changing the earphones; some recently introduced diagnostic instruments use this approach. Although insert earphones offer a hygienic advantage over supra-aural earphones, because the foam tips that are placed into a client's ear canal are disposable, the replacement cost of those tips is prohibitive for many applications. In addition to higher costs, insert earphones also yield errant thresholds in persons with eardrum perforations, including pressure-equalization tubes (Voss et al., 2000). (See Figure 3.12 for additional information about perforations.) Insert earphones also have maximum output levels that are lower than those produced by supra-aural earphones for some frequencies. Because of these differences, many diagnostic clinics keep both earphone types on hand and switch between them depending on the application.

Circumaural earphones, a third type, have cushions that encircle the pinna. ANSI (2010) describes reference equivalent threshold SPL values (SPL values corresponding to 0 dB HL) for Sennheiser model HDA200 and Koss model HV/1A earphones. These earphones and the Etymotic ER-2 insert earphones are the only ones in the current standard that have reference values covering the extended high frequencies (8 to 20 kHz).

Current standards for earphone calibration specify the level based on measures obtained with the earphone attached to an acoustic coupler or artificial ear. These couplers are designed to approximate the ear canal volume of an average person. Given that some clients (e.g., infants) have very small or very large ear canals (e.g., some postsurgical clients and persons with perforated eardrums), coupler measures may produce erroneous results, regardless of the earphone type (Voss et al., 2000; Voss and Herman, 2005). For these cases, measuring the SPL at the eardrum to specify the level presented to an individual patient would improve the accuracy of hearing thresholds. The probe-tube microphones necessary for these types of measures already exist, and hopefully, this technology will become routinely available for use in diagnostic audiometers (see Scheperle et al., 2011 for a discussion of calibration in the ear canal).

Speakers

AC thresholds can be measured using speakers as the transducer. Thresholds so obtained are known as sound-field thresholds. Sound-field thresholds are unable to provide ear-specific sensitivity estimates. In cases of unilateral hearing losses, the listener's better ear determines threshold. This limitation and others dealing with control over stimulus level greatly limit clinical applications involving sound-field thresholds. Applications for sound-field thresholds are screening infant hearing or demonstrating to the parents their child's hearing ability. Sound-field thresholds may also be desirable for a person wearing a hearing aid or cochlear implant.

In sound-field threshold measures, the orientation of the listener to the speaker has a large effect on stimulus level presented at the eardrum. A person's head and torso as well as the external ear (e.g., pinna, ear canal, concha) affect sound levels (Shaw, 1974). Differences in SPL at the eardrum are substantial for speaker locations at different distances and different angles relative to the listener. For this reason, sound-field calibration takes into consideration these factors. A mark is usually made on the ceiling (or floor) of the room to indicate the location of the listener during testing. Even at the desired location, stimulus level at the eardrum

for some frequencies can vary as much as 20 dB or more by simply having the listener move his or her head (Shaw, 1974). Calibration assumes the listener will always be facing the same direction relative to the sound source (ANSI, 2010). Furniture and other persons in the sound field also affect the stimulus level at a listener's eardrum (Morgan et al., 1979). All of these factors add to the challenge of obtaining accurate sound-field thresholds.

Another important consideration in sound-field threshold measures is the stimulus type. Thresholds corresponding to different frequencies are desired for plotting an audiogram, but puretones can exhibit large differences in level at different positions in a testing suite as a result of standing waves. Standing waves occur when direct sound from the speaker interacts with reflections, resulting in regions of cancellation and summation. Differences in stimulus level due to standing waves are minimized by using narrowband noise or frequency-modulated (FM) tones as the stimulus (Morgan et al., 1979). FM tones, also known as warbled tones, are tones that vary in frequency over a range that is within a few percent of the nominal frequency. This variation occurs several times per second. Under earphones, thresholds obtained with these narrowband stimuli are nearly identical to thresholds obtained with puretones, with some exceptions in persons with steeply sloping hearing loss configurations. FM tones and narrowband noise are the preferred stimuli for sound-field threshold measures.

Bone Vibrators

A bone vibrator is a transducer that is designed to apply force to the skull when placed in contact with the head. Puretone BC thresholds are measured with a bone vibrator like the one illustrated in Figure 3.6. A separation of 15 dB or more between masked AC and BC thresholds, with BC thresholds being lower than AC thresholds, is often evidence of a conductive hearing loss. Other possible explanations for air–bone gaps and bone–air gaps are equipment miscalibration, test–retest variability, and individual differences in anatomy that cause thresholds to deviate from the group-mean data used to derive normative values for relating AC and BC thresholds.

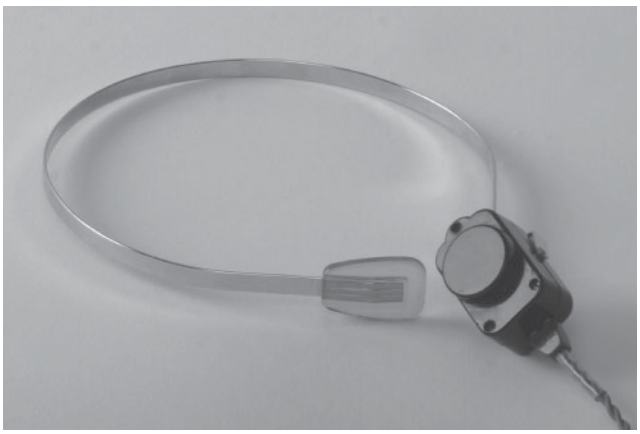


FIGURE 3.6 A clinical bone-conduction vibrator [Radioear Model B-72].

For threshold measurements bone vibrators are typically placed behind the pinna on the mastoid process or on the forehead. Although forehead placement produces slightly lower intrasubject and intersubject threshold differences (Dirks, 1994), placement on the mastoid process is preferred by 92% of audiologists (Martin et al., 1998). Mastoid placement is preferred mainly because it produces between 8 and 14 dB lower thresholds than forehead placement for the same power applied to the vibrator, depending on the frequency (ANSI, 2010). The median difference is 11.5 dB. Given that the maximum output limits for bone vibrators with mastoid placement are as much as 50 dB lower than that for AC thresholds, forehead placement yields an even larger difference. The inability to measure BC thresholds for higher levels means that a comparison of AC and BC thresholds is ambiguous in some cases. That is, when BC thresholds indicate no response at the limits of the equipment (e.g., 70 dB HL) and AC thresholds are poorer than the levels where no response was obtained (e.g., 100 dB HL), the audiologist cannot establish from these thresholds whether the loss is purely sensory/neural or whether it has a conductive component.

Test Environment

Hearing tests ideally are performed in specially constructed sound-treated chambers with very low background noise. A sound-treated room is not a sound-proof room. High-level external sounds can penetrate the walls of a sound-treated room and may interfere with test results. Because test tones near threshold can be easily masked by extraneous, external noise, test chambers have strict guidelines for maximum permissible ambient noise levels. Low background noise levels are particularly important for BC testing, when the ears remain uncovered. When testing is done in a room that meets the ANSI guidelines, the audiogram reflects that by citing ANSI S3.1 (1999), the standard governing permissible ambient noise levels. Table 3.1 shows the minimum levels of ambient noise measured in octave bands encompassing the test frequency that enable valid hearing threshold measurements at 0 dB HL.

At times, audiologists must estimate hearing thresholds in rooms that do not meet the guidelines for minimal ambient noise. Some patients in hospital rooms or nursing homes must be tested at bedside. In those cases, test results should be clearly marked so that others know the conditions under which the test was done. When possible, these bedside tests should be performed using insert earphones, which provide a greater amount of attenuation in low frequencies where ambient noise is typically more of a problem. In these

TABLE 3.1

Maximum Permissible Ambient Noise Levels for Puretone Threshold Testing

Octave Band Center Frequency (Hz)	Max dB SPL with Ears Covered	Max dB SPL with Ears Uncovered
125	39	35
250	25	21
500	21	16
1,000	26	13
2,000	34	14
4,000	37	11
8,000	37	14

Adapted from American National Standards Institute. (1999) *Maximum Permissible Ambient Noise for Audiometric Test Rooms. ANSI S3.1–1999.* New York, NY: American National Standards Institute, Inc. Octave band levels cannot exceed the tabled values to measure valid thresholds at 0 dB HL or lower.

environments, BC testing, particularly in the low frequencies, may not be valid.

Measuring Puretone Thresholds

Psychophysics is the field of study that relates the physical world with perception. PT thresholds are an example of a psychophysical measure relating the physical characteristics of a tone to a behavioral threshold.

A psychophysical procedure describes the specific method used to obtain behavioral thresholds. The most common one used in puretone audiometry is a modified method of limits. In the method of limits, the tester has control over the stimulus. A threshold search begins with the presentation of a tone at a particular frequency and intensity that is often specified by the procedure. After each presentation of the tone (or a short sequence of pulsed tones), the tester judges whether or not the listener heard it based upon the listener's response or lack of response. Each response determines the subsequent dB-level presentation. If a tone on a given presentation is not heard, the tone level is raised. If a tone is heard, the level is lowered. The rules of the psychophysical procedure govern the amount of the level change following each response, when to stop the threshold search, and the definition of threshold. The procedure, which is described in detail in subsequent sections, may be modified slightly based on the clinical population (e.g., the age of the listener).

Cooperative Listeners Age 5 Years to Adult (Earphones)

Guidelines for Manual Pure Tone Audiometry is a publication that describes a uniform method for measuring thresholds (American Speech-Language-Hearing Association [ASHA], 2005). The goal of the guideline is to minimize differences across clinics by standardizing procedures. The committee that drafted this consensus document understood that its recommendations represent general guidelines and that clinical populations may require variations of the procedure.

Instructions

Puretone audiometry begins with instructing the individual being tested. The instructions are a critical part of the puretone test, because thresholds measured using this clinical procedure are biased by the willingness of a person to respond. Some listeners wait for a tone to be distinct before they respond, which leads to higher thresholds than for someone who responds whenever they hear any sound that could be the tone. This bias is controlled in the instructions by informing listeners to respond any time they hear the tone no matter how faint it may be. A study by Marshall and Jesteadt (1986) shows that response bias controlled for in this manner plays only a small role (a few dB at most) in PT thresholds obtained using the ASHA guideline. Marshall and Jesteadt (1986) also reported that the response bias of elderly listeners was not different than that of a group of younger persons. Before the study by these authors, it was believed that elderly persons might adopt an extremely conservative response criterion, resulting in artificially elevated thresholds compared to those of younger persons.

According to the ASHA (2005) guideline, the instructions should also include the response task (e.g., raise your hand or finger, or press a button), the need to respond when the tone begins and to stop responding when it ends, and that the two ears are tested separately. Although not in the ASHA guideline, instructions asking the examinee to indicate which ear the sound is heard in may be useful. This is especially important in cases of unilateral or asymmetrical hearing losses where cross-hearing is possible.

The examiner should present the instructions prior to placement of earphones. Earphones attenuate external sounds making speech understanding more difficult, particularly for persons with hearing loss. Listeners should also be queried after the instructions are presented to determine if they understood what was said. Sample instructions are given below:

> You are going to hear a series of beeps, first, in one ear and then in the other ear. Respond to the beeps by pressing the button [switch] when one comes on and release it as soon as it goes off. Some of the beeps will be very faint, so listen carefully and respond each time you hear one. Do you have any questions?

Earphone Placement

The earphones should be placed by the examiner. For convenience, earphones are color coded; red and blue

correspond to the right and left ears, respectively. Prior to placement of earphones, clients are asked to remove jewelry such as earrings and glasses if they will interfere with the placement of the earphone. This is particularly relevant for supra-aural earphones.

For circumaural and supra-aural earphones, the diaphragm of the earphone should be centered over the ear canal. The examiner should view each ear while the phone is being placed. Immediately after placement, the headband is tightened enough to make the earphone perpendicular to the floor when the examinee is sitting upright.

The first step in placement of insert earphones is to attach a spring-loaded clip that holds the transducer in place to the examinee's clothing. The clip can be attached to clothing near the shoulder (or behind a child's neck) to keep the plug from being pulled out of the ear. In some newer implementations that combine middle-ear and otoacoustic emission measurements, the earphone is attached to a headband. For both types of support, the audiologist compresses the foam plug and inserts it into the ear canal so that its outer edge lines up with the tragus.

Placement of the Bone-conduction Vibrator

Although some recommend forehead placement (Dirks, 1994), typically audiologists place the BC oscillator on the most prominent part of the mastoid process. While holding the oscillator against the mastoid process with one hand, the headband is fit over the head to hold the oscillator in place using the other hand. The oscillator surface should be set directly against the skin, not touching the pinna, and with no hair or as little hair as possible between the oscillator and the skin. Some audiologists play a continuous low-frequency tone while moving the oscillator slightly side to side, asking the listener to report the location at which the tone is the strongest.

Audiometric Procedure for Threshold Measurement

The ASHA Guideline (2005) recommends starting a threshold search from either well below threshold or using a suprathreshold tone that familiarizes the participant with the stimulus. Most clinicians prefer the familiarization method. For the familiarization approach, testing usually begins at 1,000 Hz at 30 dB HL unless prior knowledge of the examinee's hearing suggests otherwise (ASHA, 2005). At 1,000 Hz, an examinee is more likely to have residual hearing than at a higher frequency, and test–retest reliability is excellent. Testing begins with an examinee's self-reported better ear. If the examinee believes both ears are identical, testing begins by convention with the right ear. The better ear is tested first to provide a reference to know whether masking needs to be delivered to obtain a valid estimate of threshold for the poorer ear.

Tonal duration is an important factor in a puretone test. On most audiometers, the option exists to select either pulsed or manual presentation. A 1- to 2-second duration tone is recommended for manual presentation (ASHA, 2005). The duration is determined by the amount of time the interrupter switch is held down. Pulsed tones are achieved by selecting this option on the audiometer's front panel. If pulsed tones are selected, then the audiometer alternately presents the tone followed by a short silent interval (typically 225 ms on followed by 225 ms off) for as long as the interrupter switch is depressed. The minimum duration for a single pulse of the tone is critical. Numerous psychoacoustic studies have shown that tonal durations between roughly 200 ms and 1 second or more yield nearly identical thresholds (Watson and Gengel, 1969). By contrast, the same studies show that durations less than 200 ms result in higher thresholds. For this reason, audiometers are designed to have a nominal pulse duration of 225 ms (ANSI, 2010). Pulsed and manually presented tones presented from audiometers that maintain tonal durations between 200 ms and 2 seconds yield nearly identical thresholds, as the psychoacoustic studies suggest. However, pulsed tones are preferred for two reasons. Most patients prefer pulsed tones (Burk and Wiley, 2004), and pulsed tones also reduce the number of presentations required to find threshold in persons with cochlear hearing loss who have tinnitus (Mineau and Schlauch, 1997). Apparently, pulsed tones help patients to distinguish the puretone signal from the continuous or slowly fluctuating noises generated from within their auditory system (tinnitus), thereby reducing false-positive responses. False-positive responses can lengthen test time (Mineau and Schlauch, 1997), which is costly to an audiology practice.

Thresholds typically are obtained using a modified Hughson–Westlake down-up procedure, which is a specific implementation of a method-of-limits procedure (Carhart and Jerger, 1959; Hughson and Westlake, 1944). The examiner begins the threshold-finding procedure by presenting a tone at 30 dB HL (ASHA, 2005). If the listener responds, the level of the tone is decreased in 10-dB steps until the listener no longer responds. If the listener does not respond to this initial 30-dB tone, the examiner raises the tone in 20-dB steps until a response is obtained. After every response to a tone, the level of the tone is decreased in 10-dB steps until there is no response. For subsequent presentations when there is no response, the examiner raises the level of the tone in 5-dB steps until a response is obtained. Following this "down-10/up-5" rule, the tester continues until the threshold is bracketed a few times, and a threshold estimate is obtained. ASHA (2005) recommends that threshold should correspond to the level at which responses were obtained for two ascending runs, which is what most clinicians based their thresholds on even when the ASHA (1978)Guideline recommended that thresholds be based on three ascending runs. Research based on computer simulations of clinical procedures (Marshall and Hanna, 1989) supports the clinician's position and that of the ASHA (2005) guideline. The computer simulations of thresholds based on three

ascending runs showed only a minimal reduction of the variability when compared to thresholds based on two ascending runs. Listeners who produce inconsistent responses are an exception, and for these listeners, additional measurements can be made to confirm the threshold estimate.

After a threshold is measured at 1,000 Hz, the next frequencies that are examined depend on the goal, but the higher frequencies are typically tested prior to the lower frequencies. For diagnostic audiometry, thresholds are measured at octave intervals between 250 and 8,000 Hz, along with 3,000 and 6,000 Hz. Intra-octave thresholds between 500 and 2,000 Hz should be measured when thresholds differ by 20 dB or more between two adjacent octaves. ASHA (2005) also recommends that 1,000 Hz be tested twice as a reliability check. Refer to the ASHA (2005) guidelines for specifics about the recommended protocol and Chapter 6 for details about the use of masking noise to eliminate the participation of the nontest ear. Masking noise is needed whenever the threshold difference between ears is equal to or exceeds the lowest possible values for interaural attenuation. For BC testing, masking is needed to verify results anytime an air–bone gap in the test ear of greater than 10 dB is observed. For AC testing, masking is needed when the difference between the AC threshold in the test ear and the BC threshold of the nontest ear is greater than or equal to 40 dB for supra-aural earphones, and considerably more for insert earphones, especially in the low frequencies (Killion and Villchur, 1989). Specific recommendations for insert earphones cannot be made until a study with a larger sample size is completed.

Testing Children Younger than Age 5 Years and Persons with Special Needs

For most children younger than age 5 years, audiologists have special procedures that they employ to measure PT thresholds. Some of these same procedures are also appropriate for persons older than 5 years who have cognitive deficits. Chapter 24 on pediatric hearing assessment describes these procedures and their interpretation.

Audiometric Interpretation

PT thresholds are displayed in tabular or graphical formats. The tabular format is useful for recording the results of serial monitoring of thresholds, as in a hearing conservation program, but in many applications, thresholds are plotted on an audiogram. ASHA (1990), in a publication entitled *Guidelines for Audiometric Symbols,* suggests a standardized form for the audiogram. Although other formats for plotting audiograms are acceptable, it is helpful to use a standardized format for ease of interpretation across clinics. The audiogram consistent with that recommended in the ASHA guidelines (1990) is shown in Figure 3.7 along with recommended symbols. This audiogram only covers the conventional frequencies. Thresholds for extended high frequencies are plotted often in units of dB SPL, because average extended high-frequency thresholds vary over a wide range with the age of the listener, making dB SPL a better reference than dB HL for comparing thresholds to norms for listeners of different ages. Conversion between

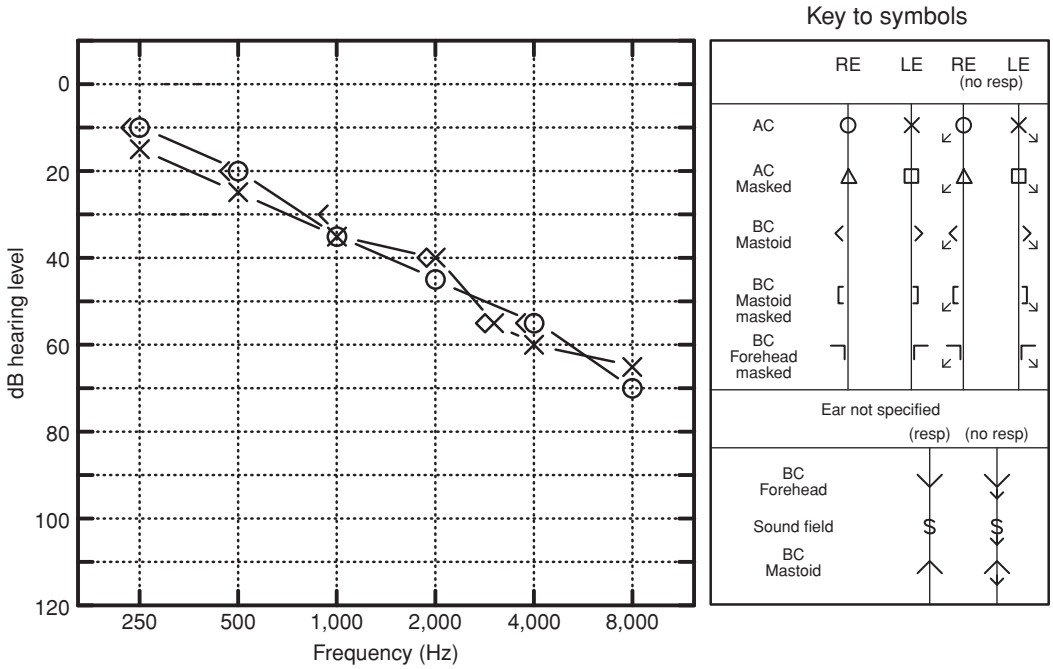


FIGURE 3.7 Recommended audiogram and symbols [ASHA, 1990] with a sensory/neural hearing loss. RE and LE represent the right ear and left ear, respectively. The word "response" is abbreviated "resp."

TABLE 3.2

Classification of Degree of Hearing Loss Calculated from the Average of Thresholds for 500, 1,000, and 2,000 Hz[a]

Degree of Loss	Northern and Downs (2002)	Goodman (1965)	Jerger and Jerger (1980)
None	<16	<26	<21
Slight	16–25		
Mild	26–30	26–40	21–40
Moderate	30–50	41–55	41–60
Moderately severe		56–70	
Severe	51–70	71–90	61–80
Profound	>70	>90	>80

[a]Although all three references cited differ in the value accepted as a profound loss, a loss of 90 dB HL or more is widely accepted as representing a qualitative as well as a quantitative boundary between hearing and deafness.

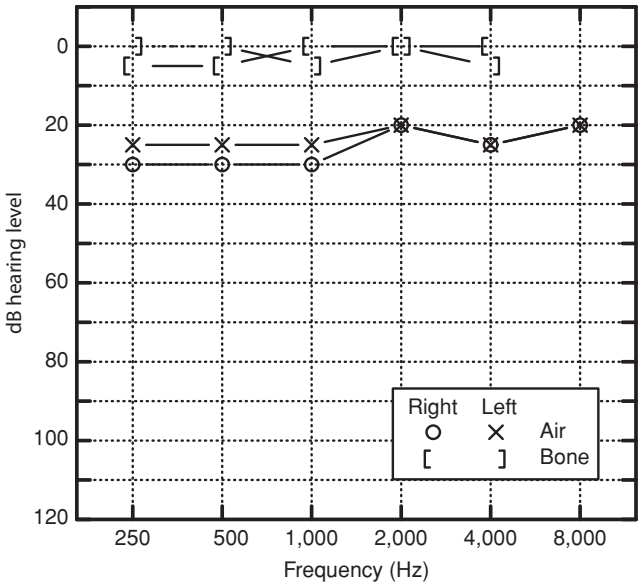


FIGURE 3.8 A bilateral conductive hearing loss. The plotted values represent the average loss reported by Fria et al. (1985) in a group of children with otitis media.

units of dB SPL and dB HL can be accomplished for three different earphone models by consulting reference levels published in ANSI (2010).

Audiograms are often classified by categories based on the degree of hearing loss. A number of authors have published systems for classifying hearing loss based on the average AC thresholds for three frequencies. The frequencies used for this purpose are usually 500, 1,000, and 2,000 Hz, often referred to as the three-frequency puretone average (PTA). Table 3.2 shows the categories for the degree of loss based on this PTA for three different authors (Goodman, 1965; Jerger and Jerger, 1980; Northern and Downs, 2002). The first category is normal hearing. Note that none of the three authors agree on the upper limit for normal, which ranges from 15 to 25 dB HL. Northern and Downs (2002) suggest using 15 dB HL as the upper limit for normal hearing for the three-frequency PTA for children between 2 and 18 years of age and a higher limit for adults. A 15 dB HL upper limit for normal hearing may produce a significant number of false positives when applied to thresholds for individual audiometric frequencies, even in children (Schlauch and Carney, 2012). Regardless of the value used as an upper limit for normal hearing, keep in mind that an ear-related medical problem can still exist even though all thresholds fall within the defined normal range. For example, the presence of a significant air–bone gap might indicate the presence of middle-ear pathology even though all AC thresholds fall within normal limits.

The original intent of classification system for severity of loss based on a three-frequency PTA was to express, in a general way, the degree of handicap associated with the magnitude of the loss. These categories are only somewhat successful at achieving this goal, because (1) handicap is dependent on many factors related to an individual's needs and abilities, (2) only some of the speech frequencies are assessed using this three-frequency average (speech frequencies range from 125 to 6,000 Hz), and (3) identical amounts of hearing loss sometimes result in large differences in the ability to understand speech and, as a consequence, the degree of disability associated with the loss. Despite these limitations, many audiologists use these categories routinely to summarize the amount of loss in different frequency regions of an audiogram when describing results to other professionals or to a client during counseling.

Another factor in audiometric classification is the type of hearing loss. The type of hearing loss is determined by comparing the amount of hearing loss for AC and BC thresholds at the same frequency. A sensory/neural hearing loss has an equal amount of loss for AC and BC thresholds (as shown in Figure 3.7). By contrast, a conductive loss has lower BC thresholds than AC thresholds (as shown in Figure 3.8). Conductive-loss magnitude is described by the decibel difference between AC and BC thresholds. This difference is known as the air–bone gap, a value that has a maximum of about 65 dB[†] (Rosowski and Relkin, 2001). Due to test–retest differences, an air–bone gap needs to exceed 10 dB before it is considered significant. A mixed hearing loss shows a conductive component and a sensory/neural component. In other words, a mixed loss has an air–bone

[†]Physiologic models suggest that the maximum air–bone gap occurs when there is an intact tympanic membrane and a disarticulated ossicular chain (Rosowski and Relkin, 2001).

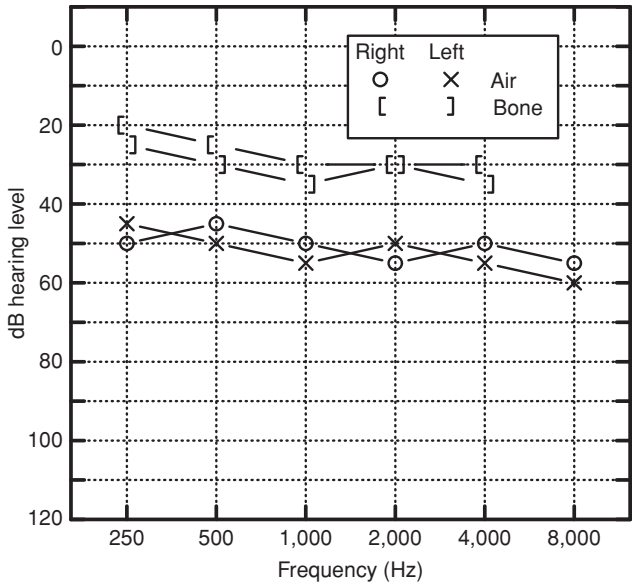


FIGURE 3.9 A mixed hearing loss.

gap, and the thresholds for BC fall outside the range of normal hearing (Figure 3.9).

Yet another way that audiograms are described is by the hearing-loss configuration. The configuration takes into account the shape of the hearing loss. A description of the configuration of the loss helps in summarizing the loss to patients and to other professionals and often provides insight into the etiology or cause of the loss. Some typical shapes and the criteria used to describe them are shown in Table 3.3.

An audiogram is summarized verbally by the degree, type, and configuration of the hearing loss for both ears. If a person has normal thresholds in one ear and a hearing loss in the other ear, this is known as a unilateral hearing loss. A loss in both ears is described as a bilateral hearing loss. Bilateral losses are described as symmetric (nearly equal thresholds in both ears) or asymmetric.

TABLE 3.3

Criteria for Classifying Audiometric Configurations

Term	Description
Flat	<5 dB rise or fall per octave
Gradually falling	5–12 dB increase per octave
Sharply falling	15–20 dB increase per octave
Precipitously falling	Flat or gradually sloping, then threshold increasing at 25 dB or more per octave
Rising	>5 dB decrease in threshold per octave
Peaked or saucer	20 dB or greater loss at the extreme frequencies, but not at the mid frequencies
Trough	20 dB or greater loss in the mid frequencies (1,000–2,000 Hz), but not at the extreme frequencies (500 or 4,000 Hz)
Notched	20 dB or greater loss at one frequency with complete or near-complete recovery at adjacent octave frequencies

Modified from Carhart R. (1945) An improved method of classifying audiograms. *Laryngoscope*. 5, 1–15 and Lloyd LL, Kaplan H. (1978) *Audiometric Interpretation: A Manual for Basic Audiometry*. Baltimore, MD: University Park Press.

Some Limitations of Puretone Testing

Test–Retest Reliability

PT thresholds are not entirely precise. Consider a cooperative adult whose AC thresholds are measured twice at octave intervals between 250 and 8,000 Hz. For these two measures, assume too that the earphones are removed and replaced between tests. For this situation, the probability of obtaining identical thresholds at each frequency is small. This is due to test–retest variability. Test–retest variability is also responsible for BC thresholds not always lining up with AC thresholds in persons with pure sensory/neural losses. As reported by Studebaker (1967), test–retest variability causes false air–bone gaps and false bone–air gaps (BC thresholds poorer than AC thresholds). The source of this variability is a combination of variations in the person's decision process, physiologic or bodily noise, a shift in the response criterion, and differences in transducer placement. It is assumed that the equipment is calibrated correctly for successive tests and that the standard is not in error (Margolis et al., 2013).

The inherent variability of PT thresholds poses a problem for audiologists who are faced with making clinical decisions based on these responses. Audiologists frequently need to assess whether hearing has changed significantly since the last test, whether hearing is significantly better in one ear than the other, and whether an air–bone gap is significant.

A good place to begin with understanding test–retest variability is to consider the standard deviation (SD) of test–retest differences at a single frequency. When a 5-dB SD is assumed, threshold differences on retest of 15 dB or more are rarely expected if only a single threshold measurement is retested. By contrast, when complete audiograms are assessed, the likelihood of obtaining a large threshold difference at one frequency on retest increases. For example, 15 dB or greater differences on retest are expected only 1.24% of the time when the threshold for a single frequency is assessed. When thresholds for six frequencies are assessed in each ear (octave intervals between 0.25 and 8 kHz), 14%

of the persons tested would be expected to have at least one threshold differing by 15 dB or more (Schlauch and Carney, 2007). Thus, differences of 15 dB or more in these applications would be much more commonplace than those predicted by the SD of inter-test differences for a single frequency.

Several methods have been proposed to assess the significance of threshold differences on retest for complete audiograms (Schlauch and Carney, 2007). These methods usually require that thresholds for more than one frequency contribute to the decision process, although some accept a large change for a single frequency, such as 20 dB or more, as a significant difference. One of these methods defines a significant threshold shift by a minimal change in a PTA. For instance, the Occupational Safety and Health Administration (1983) defines a notable threshold shift (in their terminology, a standard threshold shift) as a 10-dB or greater change in the PTA based on thresholds for 2, 3, and 4 kHz in either ear. These frequencies were selected because they include those that are susceptible to damage by occupational noise and have stable test–retest reliability. A second commonly used approach requires threshold differences to occur at adjacent frequencies. One rule that is applicable to many situations defines a significant threshold shift as one for which two adjacent thresholds differ by 10 dB or more on retest. This criterion has been applied widely in audiometric studies and is sometimes combined with other criteria to arrive at a decision (ASHA, 1994). A third approach recommends repeating threshold measurements during a single session to improve audiometric reliability (National Institute for Occupational Safety and Health, 1998). This method is paired with a rule or rules defining the criterion for a significant threshold shift. The notable difference between this method and the others described earlier is that the criterion defining a threshold shift must be repeatable to be accepted as significant.

The examples in this section on the variability of PT thresholds have assumed a fixed SD of test–retest differences of ±5 dB for all audiometric frequencies. Although 5 dB is a reasonable average value for many situations, studies show that the SD varies with type of earphone, the time between tests, and even with audiometric frequency (Schlauch and Carney, 2007).

Vibrotactile Thresholds

In persons with significant hearing losses, sound vibrations produced by earphones and bone vibrators may be perceived through the sense of touch. Such thresholds are known as vibrotactile thresholds.

Figure 3.10 illustrates the range of levels found to yield vibrotactile thresholds for a supra-aural earphone and a bone vibrator. A threshold occurring within the range of possible vibrotactile thresholds is ambiguous; it could be a hearing threshold or a vibrotactile threshold. Because relatively low vibrotactile thresholds are observed for BC at 250 and 500 Hz, a false air–bone gap is likely to occur in persons with significant sensory/neural losses at these frequencies.

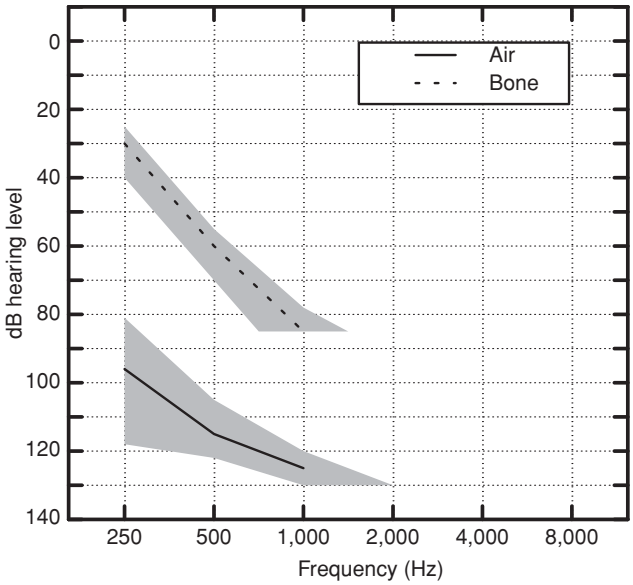


FIGURE 3.10 Mean vibrotactile thresholds for bone conduction (*dashed line*) and air conduction (*solid line*). The range of responses is indicated by the *shaded region.* [Adapted from Boothroyd and Cawkwell (1970)].

Boothroyd and Cawkwell (1970) recommend asking the client if they "feel" the stimulus or "hear" the stimulus as a means to differentiate between these two outcomes. Persons with experience with auditory sensations can usually make this distinction.

The values for vibrotactile thresholds illustrated in Figure 3.10 are based on only nine listeners. A more detailed study needs to be conducted to specify these ranges more precisely for the transducers in current use.

Bone-conduction Thresholds: Not a Pure Estimate of Sensory/Neural Reserve

The goal of BC testing is to obtain an estimate of sensory/neural reserve, but BC thresholds sometimes are influenced by the physiologic properties of the external, middle, and inner ears. The BC vibrator sets the skull into vibration, which stimulates the cochlea, but this does not happen in isolation. When the skull is vibrated, the middle-ear ossicles are also set into motion, and this inertial response of the ossicular chain contributes to BC thresholds. Changes in the external and middle ear can modify the contribution of the inertial response, which may result in significant changes in BC thresholds (Dirks, 1994).

A classic example of a middle-ear problem that influences BC thresholds is otosclerosis. Otosclerosis frequently

causes the footplate of the stapes to become ankylosed or fixed in the oval window. This disease process and some other types of conductive losses (e.g., glue ear) (Kumar et al., 2003) reduce the normal inertial response of the ossicles to BC hearing. The result is poorer thresholds that form a depressed region of BC hearing known as Carhart's notch (Carhart, 1950). This notch, which typically shows poorer BC thresholds between 500 and 4,000 Hz with a maximum usually at 2,000 Hz of 15 dB, disappears following successful middle-ear surgery. The finding that BC thresholds improve following middle-ear surgery is strong evidence that these poorer BC thresholds observed in stapes immobilization are due to a middle-ear phenomenon rather than a change in the integrity of the cochlea.

A frequently observed example of middle-ear problems affecting BC thresholds occurs in persons with otitis media with effusion. In this group, falsely enhanced BC thresholds in the low frequencies (1,000 Hz and below) are seen often. The magnitude of the enhancement can be as much as 25 dB (Snyder, 1989). Upon resolution of the middle-ear problem, these previously enhanced BC thresholds become poorer and return to their premorbid values.

Similarly, enhancement in BC thresholds occurs for low frequencies with occlusion of the external ear canal by a supra-aural ear phone. This low-frequency BC enhancement, known as the occlusion effect, must be considered when occluding the nontest ear to present masking noise during BC testing. However, when the masking noise is presented using an insert earphone with the foam plug inserted deeply into the ear canal, the amount of the low-frequency enhancement is smaller than it is when supra-aural earphones are used to deliver the masking noise (Dean and Martin, 2000). Further, apparent enhancement of BC thresholds can occur in cases of superior canal dehiscence (see Chapter 4).

Special Populations

Tinnitus

Many people who come for hearing testing experience tinnitus, the sensation of hearing internal sounds when no sound is present (see Chapter 35). Tinnitus can interfere with the perception of test tones, which can lead to a large number of false-positive responses, and false-positive responses can produce an inaccurate (too sensitive) threshold estimation. Some listeners simply require additional instruction and encouragement to wait until they are more certain they have heard a test tone. In some cases, the audiologist can present a clearly audible tone at the test frequency to remind the listener of the test tone. For more intractable cases, the examiner can present a series of pulsed tones and ask the listener to count the number of tones. It is important with listeners who are giving false-positive responses to avoid a fixed presentation rhythm and to provide irregular intervals of "no trial" silence to confirm that their responses are, in fact, responses to test tones.

In rare cases, patients have tinnitus resulting from blood flowing nearby auditory structures. Blood flowing through a vein or artery sometimes produces masking noise or "bruit" that can elevate thresholds for low-frequency tones (Champlin et al., 1990). On the audiogram, this form of tinnitus may produce an apparent sensory/neural loss. The loss occurs because the tinnitus masks AC and BC thresholds. Bruit, a recordable form of tinnitus resulting from vibrations in the head or neck, is documented by audiologists by measuring sound levels in the ear canal (Champlin et al., 1990). This problem is treatable when the problem is caused by a vein. In a case study reported by Champlin et al. (1990), the patient received some reduction in tinnitus loudness before surgery by applying pressure to her neck. Surgical ligation of the vein responsible for the tinnitus was shown to be an effective treatment. Surgery reduced tinnitus loudness, SPLs of the bruit measured in the ear canal were lower, and the audiogram showed significantly improved thresholds.

Pseudohypacusis

Pseudohypacusis, also known as functional hearing loss and nonorganic hearing loss, is the name applied to intra-test and inter-test inconsistencies that cannot be explained by medical examinations or a known physiologic condition (Ventry and Chaiklin, 1965). Most persons who present with this condition are feigning a hearing loss for monetary or psychological gain, but a very small percentage of persons have subconscious motivations related to psychological problems (see Chapter 33).

Persons presenting with pseudohypacusis are often identified from inconsistencies in their responses to the puretones. In addition to general poor reliability during threshold searches, there is a tendency for the threshold to become poorer as more presentations are made (Green, 1978). Methods of identifying the pseudohypacusis by comparing PT thresholds with other measures and the use of special tests are covered in Chapter 33.

Auditory Neuropathy

Auditory neuropathy (or auditory dys-synchrony) is a condition that may account for 11% of hearing losses found in children at risk for hearing loss (Rance et al., 1999). Information about this disorder may be found in Chapters 13 and 19. Many of these children appear to be severely hard of hearing because of very poor speech recognition; however, PT thresholds do not follow any specific pattern. Puretone hearing thresholds for these children range from minimal to profound losses. Individuals with auditory neuropathy classically show very inconsistent audiometric responses during a test and between tests.

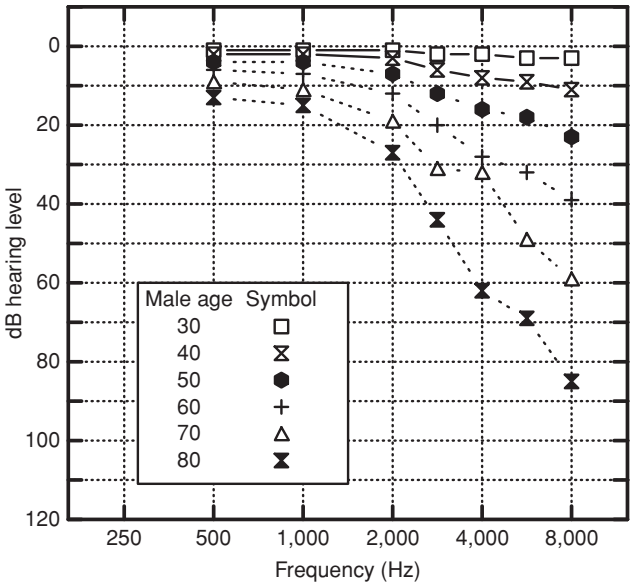


FIGURE 3.11 Average audiograms for adult males for different decades of life. Data from National Institute on Deafness and Other Communication Disorders (2005).

Aging

Presbycusis is a term that describes the gradual loss of hearing sensitivity that occurs in most individuals as they grow older. Studies suggest (Schuknecht, 1974; Dubno et al., 2013) that several different types of damage can occur to the auditory system because of aging. Hearing loss due to aging typically causes a gently sloping, high-frequency sensory/neural hearing loss that tends to be slightly greater in men than in women. Figure 3.11 shows the average amount of threshold elevation expected based on aging in men who have had limited exposure to intense sounds. Even among this select group of participants, large individual differences are often observed.

Acoustic Tumors

An acoustic tumor (acoustic neuroma/neurinoma or vestibular schwannoma) is a rare disorder. Once identified, these tumors are usually removed surgically, because they can compress the brainstem and threaten life. Early diagnosis and removal lessen the risk of complications during surgery and increase the opportunity to preserve hearing if that approach is pursued.

Magnetic resonance imaging (MRI) is the definitive test for acoustic tumors. Unfortunately, it is expensive and only becomes cost effective when a screening test is used to assess which patients should receive an MRI. Puretone audiometry should be considered as part of that screening procedure. When the auditory nerve is compressed by the tumor, it often, but not always (Magdziarz et al., 2000), results in a unilateral or asymmetrical hearing loss. Because the fibers on the outside of the auditory nerve code high frequencies, the hearing loss is associated with the high frequencies (Schlauch et al., 1995). Studies have shown that a screening test that compares the average threshold difference between ears for 1, 2, 4, and 8 kHz is most effective (Schlauch et al., 1995). Threshold differences between ears for this PTA that exceed 15 dB or 20 dB maximize identification of persons with these tumors while minimizing false-positive diagnoses of persons with cochlear losses. The pass–fail criterion (e.g., requiring a 20-dB difference between ears) may differ depending on the money available for follow-up tests. A pass–fail criterion requiring 15-dB or greater differences between ears identifies more tumors than one requiring 20-dB or larger differences, but the smaller difference also yields more false-positive responses. False-positive responses (in this case, persons with cochlear losses identified incorrectly as having tumors) place a burden on the healthcare system, because follow-up tests such as MRI or auditory-evoked potentials are expensive.

The effectiveness of a screening test based on the threshold asymmetries between ears is dependent on the clinical population. This test was found to be ineffective in a Veterans Administration hospital where many patients are males who have presbycusis and noise-induced hearing loss (NIHL) (Schlauch et al., 1995). By contrast, preliminary data from young women with normal hearing in their better ear suggest that true-positive rates and false-positive rates for this test are comparable to those for auditory brainstem response (Schlauch et al., 1995). It should also be noted that a small percentage of persons (<3%) with acoustic tumors have no hearing loss or hearing threshold asymmetry (Magdziarz et al., 2000).

Ménière's Disease

Ménière's disease is diagnosed based on the symptoms of sensory/neural hearing loss, vertigo, tinnitus, and aural fullness (Committee on Hearing and Equilibrium, 1995) as well as the exclusion of other known diseases. Adding to the diagnostic challenge, the four symptoms do not occur all at once, and some of them may occur only during the intermittent attacks that characterize this disease. It takes, on average, 1 year after the first symptom occurs before all of the symptoms are experienced by a person stricken with this disease. Ménière's disease rarely occurs before age 20 and is most likely to begin between the fourth and sixth decades (Pfaltz and Matefi, 1981).

Ménière's disease usually begins as a unilateral sensory/neural hearing loss, but the frequency of bilateral involvement increases with disease duration (Stahle and Klockhoff, 1986). Although audiometric configuration is not too helpful in diagnosing Ménière's disease, a peaked audiogram (described in Table 3.3) is most common (roughly 60% of involved ears), and a rising audiogram is also seen quite frequently, especially in the earliest stages of the disease.

However, the peaked audiogram is also seen in 13% of ears with acoustic tumors (Ries et al., 1998).

Noise-induced Hearing Loss and Acoustic Trauma

Exposure to intense sound levels can cause permanent or temporary hearing loss due to hair cell damage. When a narrowband sound is presented at a level high enough to result in damage, a loss occurs at a frequency roughly one-half octave above the frequency of exposure (Henderson and Hamernik, 1995). Most people who are exposed to damaging noise levels in their work or recreational endeavors are exposed to broadband sounds, but their losses, especially during early stages of NIHL, are characterized by a "notch" (a drop in hearing) on the audiogram. The greatest hearing loss typically occurs in the region of 3,000 to 6,000 Hz. The susceptibility of these frequencies is a result of sound amplification by the external ear (Gerhardt et al., 1987). The amplification is mainly a result of the ear canal resonance, which increases the level of sound by 20 dB or more. Temporary hearing loss is referred to as temporary threshold shift (TTS), and permanent changes are referred to as permanent threshold shifts (PTS).

The greater variability of thresholds at 6 and 8 kHz than at other frequencies makes small noise notches associated with early NIHL difficult to identify. Some frequently used rules for quantifying noise notches can produce high false-positive rates when decisions are based on a single audiogram (Schlauch and Carney, 2011). Averaging multiple audiograms improves diagnostic accuracy as does clearing the ear canals of all earwax, which can result in the appearance of a high-frequency loss (Jin et al., 2013; Schlauch and Carney, 2012). NIHL can be slowly progressive, as listeners are exposed to high sound levels over months and years (Ward et al., 2000), or it can rapidly change, such as noise trauma after a sudden explosion or impulsive sound (Kerr and Byrne, 1975; Orchik et al., 1987). The shooting of a rifle can result in a greater loss in the ear closest to the muzzle of the gun. In right-handed persons, the left ear is exposed directly to the muzzle, and the right ear is protected from the direct blast by the head. New evidence (Kujawa and Liberman, 2009) suggests that PT thresholds may return to near normal following noise exposure, whereas functional auditory abilities may remain compromised due to the noise exposure. (See Chapter 32 for NIHL.)

Ototoxicity

Regular monitoring of PT thresholds is particularly important for patients who take drugs known to be ototoxic. For example, certain powerful antibiotics and cancer-fighting drugs are known to cause cochlear and vestibular damage in many patients. Monitoring hearing sensitivity during treatment could allow a physician to consider alternative treatments that might preserve hearing. Ototoxic drugs typically cause reduction in high-frequency hearing before having any adverse effect on hearing for the speech range. For this reason, extended high-frequency hearing testing is recommended for ototoxic monitoring test protocols. Several studies have demonstrated the effectiveness of early identification of ototoxic hearing loss by monitoring thresholds for frequencies higher than 8,000 Hz (Fausti et al., 1992). However, for ototoxic drugs that selectively damage inner hair cells in the cochlea (e.g., carboplatin), PT thresholds may be unaffected even though extensive damage has occurred (Lobaranis et al., 2013).

Otitis Media

Young children are susceptible to temporary, recurring middle-ear inflammations (otitis media) that are often accompanied by fluid in the middle ear (effusion). Otitis media, often referred to as a middle-ear "infection," may be viral or bacterial but is most often serous (noninfected fluid). Otitis media is the most common medical diagnosis for children, accounting for 6 million office visits in 1990 for children between the ages of 5 and 15 years (Stoll and Fink, 1996). Adults, too, may have otitis media with effusion, although the prevalence decreases significantly with age (Fria et al., 1985). During the active infection, often lasting a month or more, a patient's hearing loss may fluctuate, usually varying between 0 and 40 dB. The average degree of hearing loss is approximately 25 dB. Figure 3.8, which was used earlier in this chapter to illustrate an audiogram for a conductive loss, shows an audiogram derived from the average thresholds from a group of children diagnosed with otitis media.

Tympanic Membrane Perforations

Tympanic membrane perforations are caused by trauma, disease, or surgery. The diameter and location of perforation and the involvement of other middle-ear structures determine the amount of conductive hearing loss, if any. For instance, a myringotomy and the placement of pressure-equalization tubes represent a physician-induced perforation that results in a minimal air–bone gap in successful surgeries.

The measurement of AC thresholds in the presence of tympanic membrane perforations requires special consideration. Figure 3.12 shows an audiogram obtained in a single session from a school-age child who has a tympanic membrane perforation in the left ear and a pressure-equalization tube in the right ear. Thresholds were measured twice in each ear, once with supra-aural earphones and again with insert earphones. Note that the low-frequency thresholds obtained from insert earphones were as much as 15 to 25 dB poorer than the ones obtained with supra-aural earphones. This outcome is typical and is predicted because insert earphones are more susceptible to calibration problems

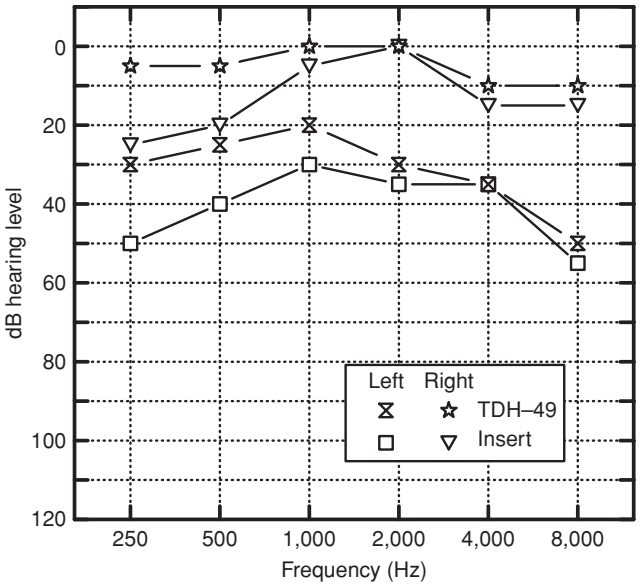


FIGURE 3.12 Audiograms obtained with two types of earphones from the same child who had bilateral perforations.

in the presence of perforations than are supra-aural earphones when calibration is based on coupler rather than real ear measurements (Voss et al., 2000). The thresholds obtained using the supra-aural earphones are more accurate in this instance and in any situations in which the effective volume of the ear canal is significantly larger than is typical.

Relation between Puretone Thresholds and Speech Measures

PT thresholds are often compared with speech audiometric test results. The two most common comparisons are with speech reception thresholds (SRT) and suprathreshold word-recognition scores (WRSs). (See Chapter 5 for a comprehensive review of speech audiometry.)

SRTs obtained using spondaic words (or spondees) agree well with PT thresholds for low frequencies. Spondees are easily recognized; listeners only need to recognize the vowels to identify these words correctly. Because of the importance of the vowels at low intensities, spondee thresholds are found to agree closely with the average of PT thresholds for 500 and 1,000 Hz (Carhart and Porter, 1971). In the event of a rising audiogram, better agreement between the spondee and PT thresholds is the average for 1,000 and 2,000 Hz. Spondee thresholds and a two-frequency PTA, as noted earlier, nearly always agree within ±10 dB in cooperative examinees. This agreement makes the threshold for spondaic words an excellent check on the validity and reliability of the audiogram. This comparison is important for most children. It is also a valuable tool for assessing the reliability of PT thresholds in adults who demonstrate inconsistent puretone responses or who may present with pseudohypacusis (Schlauch et al., 1996).

Suprathreshold word-recognition performance is assessed in most clinical settings by scoring a client's ability to repeat back a list of monosyllabic words. WRSs provide a valid estimate of speech understanding ability (Wilson and Margolis, 1983) and quantification of the distortion, if any, caused by sensory/neural hearing loss. WRSs are correlated with puretone audiometric thresholds in persons with cochlear losses (Pavlovic et al., 1986), but individuals' scores vary considerably depending on the type of damage to the auditory system. If the words are presented at a level high enough to make the speech sounds audible (overcoming the attenuation caused by the loss), persons with mild cochlear hearing loss are expected to have high WRSs, and those with severe to profound losses are likely to have fairly low scores. Dubno et al. (1995) and Yellin et al. (1989) have published tables relating WRSs and the average of PT thresholds for 500, 1,000, and 2,000 Hz for groups of persons with typical cochlear losses. WRSs that are abnormally low for a given PTA are associated with a variety of conditions including an acoustic tumor, multiple sclerosis, Ménière's disease, auditory neuropathy, and cochlear dead regions (Moore, 2004), to name a few. When there are dead regions (areas of missing inner hair cells in the cochlea), PT thresholds may appear artificially better than expected because of the spread of energy along the cochlea. Healthier cochlear cells adjacent to the missing cells will elicit a response to puretones presented at the dead region frequency.

Automated Audiometry

Clinical researchers automated the measurement of routine hearing thresholds to increase clinical efficiency (Rudmose, 1963). Devices were developed for this purpose, and several machines were manufactured and sold commercially. Some of these automated audiometers had the ability to vary intensity and frequency during a hearing test.

The Bekesy audiometer is an automated audiometer that was a common piece of equipment in major clinical and research settings in the 1960s. In its routine application, AC thresholds were assessed for interrupted tones and sustained tones for frequencies ranging from 100 to 10 kHz. Frequencies were swept through the range over time, typically at a rate of one octave per minute. The examinee controlled the level of the sound by depressing a handheld switch for as long as he or she heard a tone and released it when none was heard. The resulting brackets around threshold were recorded on an audiogram. Patterns of responses for sustained tones and interrupted tones were found to distinguish between different etiologies of hearing loss (see Chapter 33 on pseudohypacusis). In recent years, the use of Bekesy audiometry has decreased in medical settings, but it still has important applications in research, the military, and in hearing conservation programs.

Within the past few years, a new generation of automated audiometers has been developed (Margolis et al., 2010). The new automated audiometers are capable of measuring masked AC and BC thresholds, as well as WRSs, with only a single placement of the earphones and BC oscillator. Bekesy audiometry is still used along with some other automated methods (Laroche and Hetu, 1997), including ones that implement the threshold-finding procedure used in manual puretone audiometry (Margolis et al., 2010). Computer-based rules control the presentation of stimuli, examinee responses, and the plotting of thresholds. The goal is to automate threshold collection for routine cases, which will free audiologists to perform more complex measures or to work with difficult-to-test populations.

Calibration

Clinical data require accurate stimulus specification, or the results are meaningless. When most persons think of calibration of audiometers, the obvious examples include the accuracy of puretone frequency and level. However, puretone calibration involves much more, including an assessment of attenuator linearity, harmonic distortion, rise and fall times, and more. Consult ANSI (2010) and Chapter 2 on calibration in this book to learn more about this topic.

Puretone Thresholds and the Audiologic Test Battery

PT thresholds are measured on nearly everyone entering a diagnostic audiology clinic, but the test sequence and the extent of the measurements often differ across clinics. Most of these differences in protocol are implemented to save testing time, which contributes to the cost of running a clinic. ASHA's guide to manual PT threshold audiometry (2005) makes no recommendation concerning the puretone test sequence. In 2000, the Joint Audiology Committee on Practice Algorithms and Standards recommended an algorithm that listed puretone AC testing (with appropriate masking applied) followed by puretone BC testing with appropriate masking. They acknowledged that the assessment process may vary "based on patient need and the assessment setting." Furthermore, they stated that "decision-making . . . occurs(s) throughout this process."

Based on informal surveys of clinicians in a variety of settings, it seems that there is considerable variability in test protocols among clinics. In many clinics, BC thresholds are not usually obtained from persons with normal AC thresholds (near 0 dB HL) unless the case history or risk of middle-ear problems suggests otherwise. BC threshold testing is also omitted in some clinics for returning patients with pure sensory/neural losses if their AC thresholds match those of the prior visit. A common alternative test sequence is to begin with puretone AC thresholds followed by suprathreshold word-recognition testing. After word-recognition testing, BC thresholds are measured. Although it would be useful to have puretone BC thresholds prior to AC thresholds to know how much masking noise can be presented safely, this advantage is outweighed by the inconvenience of having to enter the booth multiple times to reposition the BC vibrator and earphones. Valid, masked AC thresholds can be obtained successfully from most clients before obtaining BC thresholds.

A few clinics begin with immittance testing, which usually includes a tympanogram and acoustic reflex thresholds. If the case history does not indicate a middle-ear problem and these tests of middle-ear function are normal, then BC thresholds may not be performed, and the loss, if present, is assumed to be a sensory/neural loss. A possible risk of this strategy is that, in rare instances, persons with middle-ear problems have normal immittance measures. In this situation, a conductive loss would be missed. This approach also adds the expense of immittance testing for each client. Studies should be done using an evidence-based practice model to determine whether the assessment of middle-ear status of each client using immittance or wideband reflectance (see Chapter 9) is justified. Another time-saving strategy might be to measure BC thresholds at two frequencies, a low and a high frequency, and if an air–bone gap is not observed, BC thresholds are not measured for other frequencies. A low frequency, such as 500 Hz, would assess stiffness-related middle-ear pathologies. A high frequency, such as 4,000 Hz, would identify mass-related middle-ear pathologies and collapsed canals. Since this method requires placement of the BC vibrator, the amount of time actually saved would be limited.

Despite the observed variability, it seems that it is possible for audiologists to obtain important diagnostic information about the degree, type, and configuration of hearing losses using a variety of valid, evidence-based puretone audiometric methods. Although at first glance, the puretone test procedure may appear elementary, it is clear that well-informed test procedures using appropriate and calibrated test equipment provide a necessary part of the complete audiologic test battery and form the basis for clinical decision making.

FOOD FOR THOUGHT

1. Given the known test–retest variability of PT thresholds, what is the threat to the quality of a hearing conservation program if the tester chooses not to make multiple estimates of baseline audiograms?
2. Think about examples of auditory pathology where the PT threshold might be misleading, when it might not reflect the full nature of the underlying cochlear injury.
3. Consider several cases where the ear canal volume (the volume under the earphone) might significantly affect the resulting PT threshold. Consider the client's total volume of the outer ear, perforations of the eardrum, and possible occlusions in the ear canal. What effect will these have on the resulting thresholds?

KEY REFERENCES

thePoint A full list of references for this chapter can be found at http://thePoint.lww.com. Below are the key references for this chapter.

American National Standards Institute. (1999) *Maximum Permissible Ambient Noise for Audiometric Test Rooms. ANSI S3.1–1999.* New York, NY: American National Standards Institute, Inc.

American National Standards Institute. (2010) *Specifications for Audiometers. ANSI S3.6–2010.* New York, NY: American National Standards Institute, Inc.

American Speech-Language Hearing Association. (1990) Guidelines for audiometric symbols. *ASHA.* 32, 25–30.

American Speech-Language-Hearing Association. (1994) Guidelines for the audiologic management of individuals receiving cochleotoxic drug therapy. *ASHA.* 36, 11–19.

American Speech-Language-Hearing Association. (2005) Guidelines for manual pure-tone threshold audiometry. Available online at: www.asha.org/policy/html/GL2005-00014.html.

Boothroyd A, Cawkwell S. (1970) Vibrotactile thresholds in pure tone audiometry. *Acta Otolaryngol.* 69, 381–387.

Browning GG. (1987) Is there still a role for tuning-fork tests? *Br J Audiol.* 21, 161–163.

Carhart R. (1945) An improved method of classifying audiograms. *Laryngoscope.* 5, 1–15.

Carhart R. (1950) Clinical application of bone conduction. *Arch Otolaryngol.* 51, 789–807.

Carhart R, Jerger J. (1959) Preferred method for clinical determination of pure-tone thresholds. *J Speech Hear Disord.* 24, 330–345.

Carhart R, Porter LS. (1971) Audiometric configuration and prediction of threshold for spondees. *J Speech Hear Res.* 14, 486–495.

Champlin CA, Muller SP, Mitchell SA. (1990) Acoustic measurements of objective tinnitus. *J Speech Hear Res.* 33, 816–821.

Chandler JR. (1964) Partial occlusion of the external auditory meatus: Its effect upon air and bone conduction hearing acuity. *Laryngoscope.* 74, 22–54.

Committee on Hearing and Equilibrium. (1995) Committee on Hearing and Equilibrium guidelines for the diagnosis and evaluation of Ménière's disease. *Otolaryngol Head Neck Surg.* 113, 181–185.

Dean MS, Martin FN. (2000) Insert earphone depth and the occlusion effect. *Am J Audiol.* 9, 131–134.

Dirks D. (1994) Bone-conduction thresholds testing. In: Katz J, ed. *Handbook of Clinical Audiology.* 4th ed. Baltimore, MD: William & Wilkins; pp 132–146.

Dubno JR, Eckert MA, Lee FS, Matthews LJ, Schmiedt RA. (2013) Classifying human audiometric phenotypes of age-related hearing loss from animal models. *J Assoc Res Otolaryngol.* 14, 687–701.

Dubno JR, Lee F, Klein A, Matthews L, Lam CF. (1995) Confidence limits for maximum word-recognition scores. *J Speech Hear Res.* 38, 490–502.

Fausti SA, Henry JA, Schaffer HI, Olson DJ, Frey RH, McDonald WJ. (1992) High frequency audiometric monitoring for early detection of ototoxicity. *J Infect Dis.* 165, 1026–1032.

Fria TJ, Cantekin EI, Eichler JA. (1985) Hearing acuity of children with otitis media with effusion. *Arch Otolaryngol.* 111, 10–16.

Gerhardt KJ, Rodriguez GP, Hepler EL, Moul ML. (1987) Ear canal volume and variability in patterns of temporary threshold shifts. *Ear Hear.* 8, 316–321.

Goodman A. (1965) Reference zero levels for pure tone audiometer. *Am Speech Hear Assoc.* 7, 262–263.

Henderson D, Hamernik RP. (1995) *Occupational Medicine: State of the Art Reviews, Biologic Bases of Noise-Induced Hearing Loss.* Philadelphia, PA: Hanley & Belfus, Inc.

Hughson W, Westlake H. (1944) Manual for program outline for rehabilitation of aural casualties both military and civilian. *Trans Am Acad Ophthalmol Otolaryngol.* (suppl 48), 1–15.

Jerger J, Jerger S. (1980) Measurement of hearing in adults. In: Paperella MM, Shumrick DA, eds. *Otolaryngology.* 2nd ed. Philadelphia, PA: W.B. Saunders.

Jin SH, Nelson PB, Schlauch RS, Carney E. (2013) Hearing conservation program for marching band members: Risk for noise-induced hearing loss? *Am J Audiol.* 22, 26–39.

Joint Audiology Committee on Practice Algorithms and Standards. (2000) Clinical practice guidelines and statements. *Audiol Today*. Special Issue.

Killion MC, Villchur E. (1989) Comments on "Earphones in audiometry." *J Acoust Soc Am.* 85, 1775–1778.

Kujawa SG, Liberman MC. (2009) Adding insult to injury: cochlear nerve degeneration after "temporary" noise-induced hearing loss. *J Neurosci.* 29 (45), 14077–14085.

Kumar M, Maheshwar S, Mahendran A, Oluwasamni A, Clayton MI. (2003) Could the presence of a Carhart notch predict the presence of glue at myringotomy? *Clin Otolaryngol.* 28, 183–186.

Laroche C, Hetu R. (1997) A study of the reliability of automatic audiometry by the frequency scanning method (AUDIOSCAN). *Audiology.* 36, 1–18.

Lloyd LL, Kaplan H. (1978) *Audiometric Interpretation: A Manual for Basic Audiometry.* Baltimore, MD: University Park Press.

Lobarinas E, Salvi R, Ding D. (2013) Insensitivity of the audiogram to carboplatin induced inner hair cell loss in chinchillas. *Hear Res.* 302, 113–120. http://dx.doi.org/10.1016/j.jeares.2013.03.012

Magdziarz DD, Wiet RJ, Dinces EA, Adamiec LC. (2000) Normal audiologic presentations in patients with acoustic neuroma: an evaluation using strict audiologic parameters. *Otolaryngol Head Neck Surg.* 122, 157–162.

Margolis RH, Eikelbloom RH, Johnson C, Ginter SM, Swanepoel DW, Moore, BCJ. (2013) False air-bone gaps at 4 kHz in listeners with normal hearing and sensorineural hearing loss. *Int J Audiol.* 52, 526–532. Early Online: 1–7.

Margolis RH, Glasberg BR, Creeke S, Moore BCJ. (2010) AMTAS®: Automated method for testing auditory sensitivity: Validation studies. *Int J Audiol.* 49 (3), 185–194.

Marshall L, Hanna TE. (1989) Evaluation of stopping rules for audiological ascending test procedures using computer simulations. *J Speech Hear Res.* 32, 265–273.

Marshall L, Jesteadt W. (1986) Comparison of pure-tone audibility thresholds obtained with audiological and two-interval forced-choice procedures. *J Speech Hear Res.* 29, 82–91.

Martin FN. (1994) *Introduction to Audiology.* 5th ed. Boston, MA: Allyn and Bacon.

Mineau SM, Schlauch RS. (1997) Threshold measurement for patients with tinnitus: Pulsed or continuous tones. *Am J Audiol.* 6, 52–56.

Moore BC. (2004) Dead regions in the cochlea: Conceptual foundations, diagnosis, and clinical applications. *Ear Hear.* 25 (2), 98–116.

Morgan DE, Dirks DD, Bower DR. (1979) Suggested threshold sound pressure levels for frequency-modulated (warble) tones in the sound field. *J Speech Hear Disord.* 44, 37–54.

National Institute for Occupational Safety and Health. (1998) *Criteria for a Recommended Standard: Occupational Noise Exposure: Revised Criteria.* Cincinnati, OH: National Institute for Occupational Safety and Health, US Department of Health and Human Services Report; pp 98–126.

National Institute on Deafness and Other Communication Disorders. (2005) Presbycusis. Available online at: http://www.nidcd.nih.gov/health/hearing/presbycusis.asp

Northern JL, Downs MP. (2002) *Hearing in Children.* 5th ed. New York, NY: Lippincott Williams & Wilkins.

Occupational Safety and Health Administration. (1983) Occupational noise exposure: hearing conservation amendment. Occupational Safety and Health Administration, 29 CFR 1910. 95; 48 Federal Register, 9738–9785.

Pavlovic CV, Studebaker GA, Sherbecoe RL. (1986) An articulation index based procedure for predicting the speech recognition performance of hearing-impaired subjects. *J Acoust Soc Am.* 80, 50–57.

Pfaltz CR, Matefi L. (1981) Ménière's disease – or syndrome? A critical review of diagnose criteria. In: Vosteen KH, Schuknecht H, Pfaltz CR, et al., eds. *Ménière's Disease.* New York, NY: Thieme.

Rance G, Beer DE, Cone-Wesson B, Shepherd RK, Dowell RC, King AM, et al. (1999) Clinical findings for a group of infants and young children with auditory neuropathy. *Ear Hear.* 20 (3), 238–252.

Ries DT, Rickert M, Schlauch RS. (1998) The peaked audiometric configuration in Ménière's disease: Disease related? *J Speech Lang Hear Res.* 42, 829–843.

Rosowski JJ, Relkin EM. (2001) Introduction to analysis of middle-ear function. In: Jahn AF, Santos-Sacchi J, eds. *Physiology of the Ear.* 2nd ed. San Diego, CA: Singular.

Scheperle A, Goodman SS, Neely S. (2011) Further assessment of forward pressure level for in situ calibration. *J Acoust Soc Am.* 130, 3882–3892.

Schlauch RS, Arnce KD, Olson LM, Sanchez S, Doyle TN. (1996) Identification of pseudohypacusis using speech recognition thresholds. *Ear Hear.* 17, 229–236.

Schlauch RS, Carney E. (2007) A multinomial model for identifying significant pure-tone threshold shifts. *J Speech Hear Res.* 150, 1391–1403.

Schlauch RS, Carney E. (2011) Are false-positive rates leading to an overestimation of noise-induced hearing loss? *J Speech Lang Hear Res.* 54 (2), 679–692.

Schlauch RS, Carney E. (2012) The challenge of detecting minimal hearing loss in audiometric surveys. *Am J Audiol* 21 (1), 106–119.

Schlauch RS, Levine S, Li Y, Haines S. (1995) Evaluating hearing threshold differences between ears as a screen for acoustic neuroma. *J Speech Hear Res.* 38, 1168–1175.

Schuknecht HF. (1974) *Pathology of the Ear.* Cambridge, MA: Harvard University Press.

Shaw EAG. (1974) The external ear. In: Kleidel WD, Neff WD, eds. *Handbook of Sensory Physiology.* Berlin: Springer; pp 455–490.

Snyder JM. (1989) Audiometric correlations in otology. In: Cummings CW, Fredrickson JM, Harker LS, et al., eds. *Otolaryngology Head and Neck Surgery: Update.* St Louis, MO: Mosby.

Stahle J, Klockhoff I. (1986) Diagnostic procedures, differential diagnosis, and general conclusions. In: Pfaltz CR, ed. *Controversial Aspects of Ménière's Disease.* New York, NY: Georg Thieme.

Studebaker G. (1967) Intertest variability and the air-bone gap. *J Speech Hear Disord.* 32, 82–86.

Ventry IM, Chaiklin JB. (1965) Multidisciplinary study of functional hearing loss. *J Audiol Res.* 5, 179–272.

Ventry IM, Chaiklin JB, Boyle WF. (1961) Collapse of the ear canal during audiometry. *Arch Otolaryngol.* 73, 727–731.

Voss SE, Herman BS. (2005) How does the sound pressure generated by circumaural, supraaural and insert earphones differ for adult and infant ears. *Ear Hear.* 26, 636–650.

Voss SE, Rosowski JJ, Merchant SN, Thornton AR, Shera CA, Peake WT. (2000) Middle ear pathology can affect the ear-canal sound pressure generated by audiologic earphones. *Ear Hear.* 21, 265–274.

Ward WD, Royster JD, Royster LH. (2000) Auditory and nonauditory effects of noise. In: Berger EH, Royster LH, Royster JD, Driscoll DP, Layne M, eds. *The Noise Manual.* 5th ed. Fairfax, VA: AIHA Press; pp. 123–147.

Watson CS, Gengel RW. (1969) Signal duration and signal frequency in relation to auditory sensitivity. *J Acoust Soc Am.* 46, 989–997.

Wilson RH, Margolis RH. (1983) Measurements of auditory thresholds for speech stimuli. In: Konkle DF, Rintelmann WF, eds. *Principles of Speech Audiometry.* Baltimore, MD: University Park Press.

CHAPTER 4

Bone Conduction Evaluation

James R. Steiger

INTRODUCTION

Puretone threshold measurements are routinely carried out in audiologic evaluations. By comparing air-conducted and bone-conducted thresholds, site-of-lesion information can be obtained. Disorders of the outer or middle ears disrupt the flow of energy from the earphone to the inner ear. However, much of the energy that is conducted by a bone vibrator on the skull bypasses the outer and middle ears and stimulates the inner ear essentially unimpeded. Therefore, this discrepancy favoring the bone-conducted threshold (called the air–bone gap) indicates a mechanical or conductive hearing loss (CHL). On the other hand when the inner ear is impaired, both pathways from the earphone and the bone vibrator are impacted. This bioelectrical disturbance is referred to as a sensory/neural hearing loss (SNHL). A hearing loss that has both sensory/neural and conductive elements is called a mixed hearing loss (MHL).

However, bone-conducted energy does not entirely bypass the outer ear and middle ear. Site-of-lesion diagnoses will be more accurate with an understanding that occlusion or disorder of the outer ear, middle ear, and/or inner ear components may affect bone conduction thresholds too. An understanding of bone conduction hearing is essential for audiologists to accurately apportion hearing loss among the possible sites of lesion and to identify the etiologies of hearing losses.

CHAPTER OVERVIEW

In this chapter, a historical context and a few basic principles in bone conduction as tested with tuning fork techniques are first introduced. Then highlights of the apparatus and evaluation procedures used in routine bone conduction evaluation today are considered. An overview is provided of the outer ear, middle ear, and inner ear components of bone conduction hearing, which lead to the main purpose of this chapter: a review of site-of-lesion diagnoses based on an understanding of air and bone conduction hearing. Of course, not all possible diagnoses can be covered in one chapter; rather, examples were selected of outer ear, middle ear, and inner ear disorders to highlight their role in the bone conduction components. The diagnoses reviewed include, in order of presentation:

- normal-hearing sensitivity,
- CHL from outer ear disorder (cerumen and osteoma examples),
- CHL from middle ear disorder (ossicular fixation and otitis media examples),
- MHL,
- SNHL from hair cell and/or neuron damage (presbycusis example),
- SNHL and pseudoSNHL from third-window disorders (superior semicircular canal dehiscence [SSCD] and large vestibular aqueduct examples), and
- pseudoSNHL from intracranial hypertension (syringohydromyelia example).

This chapter concludes with a review of a few important technical issues including vibrotactile responses, interaural attenuation, mastoid and forehead bone vibrator placement, and air and bone conduction threshold variability. Throughout, readers should be aware that the chapter's main focus is on persons with fully matured anatomy. Infants' bone conduction hearing may differ from adults because of immature temporal bones, outer ears, middle ears, and/or neurons (Hulecki and Small, 2011).

EARLY WRITINGS ON BONE CONDUCTION HEARING

In the 1500s Italian physicians Giovanni Filippo Ingrassia, Girolamo Cardano, and Hieronymus Capivacci were among the earliest known writers to describe bone conduction hearing (Feldmann, 1970). For instance, Capivacci recognized the diagnostic significance of bone conduction hearing to, as he described it, differentially diagnose disorders of the tympanic membrane (what we know today as CHL) from disorders of the cochlear nerve (what we know today as SNHL). For a test signal, Capivacci used the vibrations from a stringed musical instrument called a zither. He attached a metal rod to the zither strings, and his hearing-impaired listeners held the other end of the metal rod with their teeth. If the tone was heard by bone conduction, he concluded that the cochlear nerve was intact

and the hearing loss was caused by a tympanic membrane disorder blocking the pathway of air-conducted sound. In contrast, if the tone was not heard by bone conduction, he concluded that the listener's hearing loss was caused by a cochlear nerve disorder.

The Rinne Tuning Fork Test

In 1855, Heinrich Adolf Rinne (1819 to 1868) described the tuning fork test that bears his name (Feldmann, 1970). Rinne noted that the intensity of air-conducted tones was greater than that of bone-conducted tones, owing to the relatively lesser density of air in contrast to the greater density of bone. Most people, including normally hearing listeners and listeners with SNHL, therefore hear air-conducted tones louder than bone-conducted tones. In contrast, listeners with CHL hear bone-conducted tones louder than air-conducted tones for two reasons. First, listeners with CHL have outer ear occlusions or middle ear disorders that attenuate air-conducted tones. And second, outer or middle ear disorder can effectively trap bone-conducted tones that would otherwise radiate out of the ear canal; thus occlusions effectively intensify bone-conducted tones. This is the so-called occlusion effect.

Rinne's procedure was straightforward; listeners held the tuning fork with their teeth (dense bone conduction transmission) allowing vibrations to attenuate until no longer audible. Then the still vibrating tuning fork was moved in front of the ear canal (less dense air conduction transmission). If the tuning fork was audible by air conduction, the listener had normal hearing or an SNHL. If not audible by air conduction, the listener had a CHL. Today the bone conduction tuning fork placement is more likely to be on the mastoid rather than the teeth.

Audiologists to this day diagnose site of lesion by comparing the air and bone conduction thresholds of their patients. Audiometers, however, are calibrated so that 0 dB HL is a normal threshold referent for both air and bone conduction; air and bone conduction thresholds are therefore similar for normally hearing listeners as well as listeners with SNHL. For listeners with CHL, bone conduction thresholds are better than air conduction thresholds for the reasons stated above.

Weber Tuning Fork Test

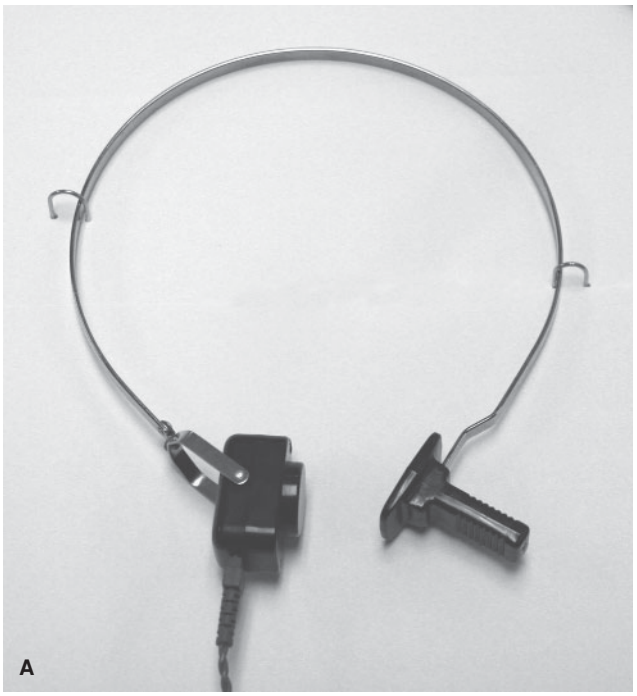
In 1827, both the German physician C.T. Tourtual and the English physicist Charles Wheatstone described lateralization of bone-conducted stimuli to an occluded ear due to the occlusion effect (Feldmann, 1970). Wheatstone experienced the occlusion effect when manually occluding an ear while listening to a vibrating tuning fork in contact with his skull. Tourtual used as his stimulus a pocket watch held in his mouth. In the 1800s Heinrich Weber observed the phenomenon in hearing-impaired listeners, and his work was carried on by both Bonafant and Schmalz in the 1840s. It was Schmalz who first wrote extensively on the diagnostic implications of what would become known as the Weber tuning fork test. Listeners with unilateral CHL hear bone-conducted tones *louder* in the impaired ear because of the occlusion effect, whereas listeners with unilateral SNHL hear bone-conducted tones *softer* in the impaired ear because of sensory/neural disorder. The Weber test is used to this day, with bone-conducted tones typically applied to the forehead by either tuning forks or bone vibrators.

APPARATUS

Bone vibrators are transducers composed of diaphragms encased in plastic. During bone conduction evaluation the circular vibrating plastic surface of the bone vibrator is held in contact with the patient's skull by the tension of a metal band. The American National Standards Institute (ANSI, 2004) specifies bone vibrator surface diameter, metal band tension, and the output characteristics of audiometers and transducers including bone vibrators. As stated above, the clinical apparatus is calibrated so that patients' air and bone conduction thresholds may be compared to the same 0 dB HL normal-hearing threshold referent. Figure 4.1 shows a common B-71 bone vibrator and also that same bone vibrator properly placed on the mastoid of a KEMAR manikin. Audiometric evaluations are conducted in test rooms compliant with ANSI S3.1-1999 (ANSI, 2003). The standard specifies maximum permissible ambient noise levels allowable for audiometric threshold testing as low as 0 dB HL, including when the ears are uncovered as during bone conduction threshold evaluations. A full discussion of calibration can be found in Chapter 2 of this book.

EVALUATION PROCEDURES

Common practice is to begin with the bone vibrator on the better hearing ear mastoid or the right mastoid if a better hearing ear is not known or suspected. Evaluation guidelines were published by the American Speech-Language-Hearing Association (ASHA) (2005), including a protocol for tone presentation, patient response modes, and the definition of threshold. Bone conduction thresholds should be tested at several frequencies and traditionally in this order: 1,000, 2,000, 3,000, and 4,000 Hz, a retest of 1,000, 500, and 250 Hz (ASHA, 1990). Because bone-conducted signals may reach either mastoid at similar intensities, contralateral ear masking may be necessary to obtain ear-specific bone conduction thresholds. A full discussion of threshold evaluation, masking procedures, and the symbols used for recording thresholds on an audiogram can be found in Chapters 3 and 6 of this book.



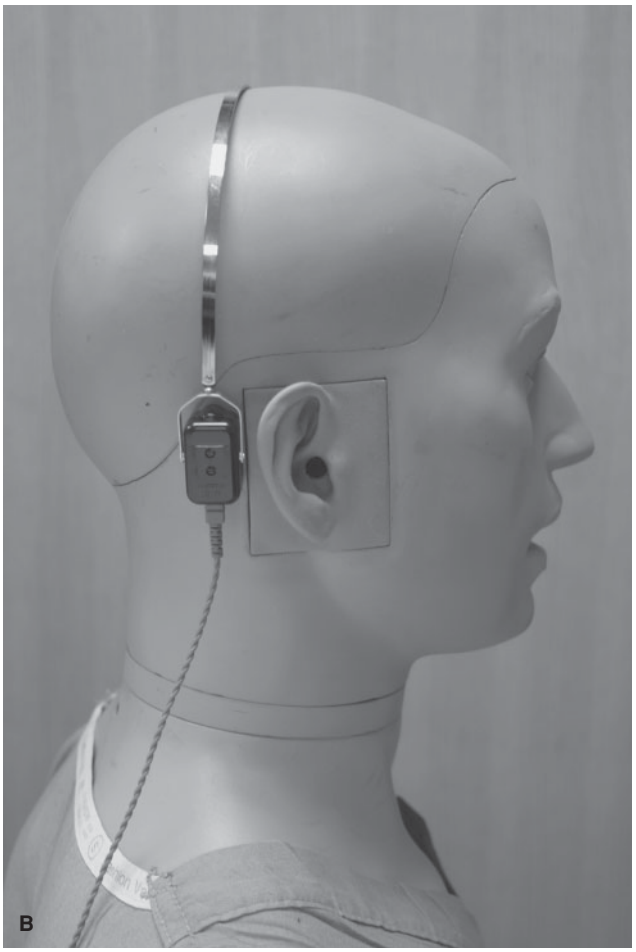


FIGURE 4.1 B-71 bone vibrator and also that same bone vibrator properly placed on the mastoid of a KEMAR manikin. [From Vento B, Durrant JD. [2009] Assessing bone conduction thresholds in clinical practice. In: Katz J, Medwetsky L, Burkard R, Hood L, eds. *Handbook of Clinical Audiology*. Philadelphia, PA: Lippincott Williams and Wilkins, http://lww.com by permission.]

Outer, Middle, and Inner Ear Components of Bone Conduction

Bone-conducted stimuli cause complex skull vibrations in several directions and with several resonances and antiresonances (Stenfelt and Goode, 2005), the details of which are beyond the scope of this chapter. Bone-conducted energy ultimately arrives at the cochlea by various transmission routes, which in turn cause basilar membrane-traveling waves to propagate from the stiffer basilar membrane base toward the more compliant basilar membrane apex as occurs for air conduction (Bekesy, 1960). Bone conduction transmission routes can be discussed in terms of outer ear, middle ear, and inner ear components.

Outer Ear Component of Bone Conduction

The outer ear bone conduction component arises from vibration of the bony and especially the cartilaginous walls of the outer ear canal that, in turn, causes sound waves to radiate into the outer ear canal (Stenfelt and Goode, 2005; Tonndorf, 1968). Vibration of the mandible may also add to the sound wave radiation into the ear canal (Stenfelt and Goode, 2005). These sound waves propagate through the middle ear and finally to the inner ear; thus, the complex phenomenon of bone conduction hearing includes exploitation of the air conduction pathway.

The outer ear component may play little role in the normal unoccluded ear canal, but its role is magnified by the occlusion effect (Stenfelt and Goode, 2005). Normally the outer ear canal acts as a high-pass filter (Tonndorf, 1968), that is, high-frequency energy is passed into the middle ear whereas low-frequency energy escapes through the ear canal opening. Outer ear canal occlusion traps this low-frequency energy and thereby enhances bone conduction hearing up to 20 dB in the lower audiometric test frequencies (Stenfelt and Goode, 2005).

Middle Ear Component of Bone Conduction

The middle ear component of bone conduction is the inertial lag of the ossicles (Barany, 1938). Middle ear ossicles are not directly attached to the skull, but are instead suspended

by ligaments and tendons and attached at either end to the elastic tympanic and oval window membranes. The ossicles are free to move out of phase with skull vibrations and will do so because of inertia, much as coffee would lag and spill from a cup moved precipitously. Middle ear ossicles vibrate relative to the skull in a like manner as during air conduction hearing, and thus energy is propagated into the inner ear. The middle ear component occurs mainly at and above 1,500 Hz and is especially significant near 2,000 Hz, the approximate resonant frequency of the middle ear (Stenfelt et al., 2003). Finally, some have proposed that bone conduction energy radiates from the walls of the middle ear into the middle ear space and sets the tympanic membrane into vibration, but that hypothesis has been challenged (Stenfelt and Goode, 2005).

Inner Ear Component of Bone Conduction

The inner ear component of bone conduction involves several contributing factors, including cochlear compression or distortion, cochlear fluid inertia, osseous spiral lamina inertia, and sound pressure transmission through skull contents (brain, membranes, and fluid). Inner ear bone conduction has been described as resulting from alternate compressions and expansions (Herzog and Krainz, 1926) or distortions (Tonndorf, 1968) of the bony cochlear capsule. In turn, cochlear fluids are displaced and basilar membrane-traveling waves are initiated. One factor making cochlear fluid displacement possible is the out-of-phase and disproportionate yielding of the round and oval cochlear windows, which creates alternating spaces for fluid displacement. Cochlear fluid movement, in turn, displaces the basilar membrane and initiates traveling waves. Also, the cochlear and vestibular aqueducts may serve as outlets for fluid displacement allowing for bone conduction hearing when the oval window is fixed as in otosclerosis (Stenfelt and Goode, 2005; Tonndorf, 1968). Fluid displacement is further enabled by a fluid volume differential between the scala tympani on one side of the scala media and the greater fluid volume of the scala vestibuli, vestibule, and semicircular canals on the other side of the scala media (Stenfelt and Goode, 2005; Tonndorf, 1968).

Inertia of the inner ear fluids contributes to inner ear bone conduction hearing, especially below 1,000 Hz (Stenfelt and Goode, 2005). Cochlear fluids and windows are free to vibrate out of phase with skull vibrations and will do so because of inertia, compared earlier to coffee that will lag and spill from a moving cup. Cochlear fluid movement, in turn, displaces the basilar membrane and initiates traveling waves. The spiral lamina may also be flexible and lag skull vibrations, thereby contributing to bone conduction hearing, especially at higher frequencies (Stenfelt et al., 2003).

Finally, bone conduction energy can travel in nonosseous skull contents, such as the brain, membranes, and fluids, and reach the cochlea through the cochlear and/or vestibular aqueducts (de Jong et al., 2011; Stenfelt and Goode, 2005). The nonosseous skull contents route may contribute little to normal bone conduction hearing, but it plays a role in some inner ear disorders as discussed below.

TRANSMISSION ROUTE MODEL

Figure 4.2 illustrates the air and bone conduction transmission routes. Note the orderly air conduction route through the outer, middle, and inner ear in contrast with more complex bone conduction hearing involving concomitant outer, middle, and inner ear components bilaterally.

EXAMPLES OF DIAGNOSES

Normal-hearing Sensitivity

Normally hearing patients are without disorder that would hinder energy from reaching the inner ear by the air conduction route. Owing to calibration, air conduction thresholds will be near 0 dB HL. Similarly, normally hearing patients

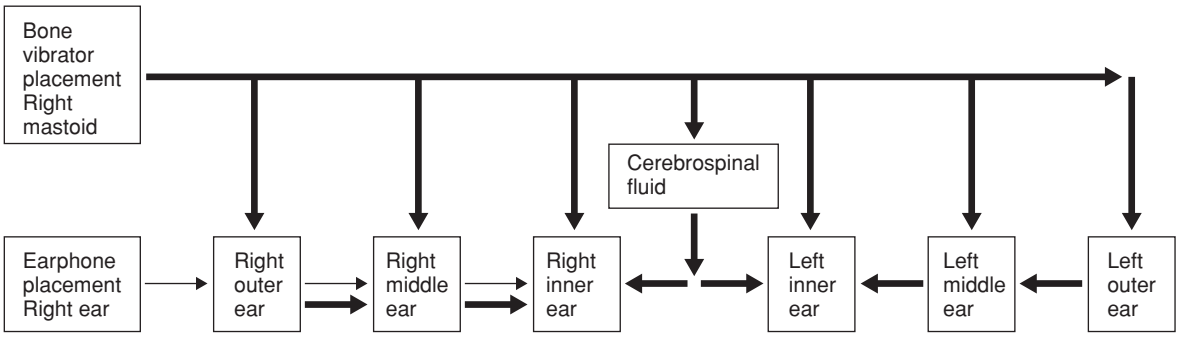


FIGURE 4.2 Transmission routes for air conduction, right ear example [*narrow arrows*] and bone conduction, right mastoid example [*bold arrows*]. Note: Higher intensity air-conducted signals can activate the bone conduction transmission route.

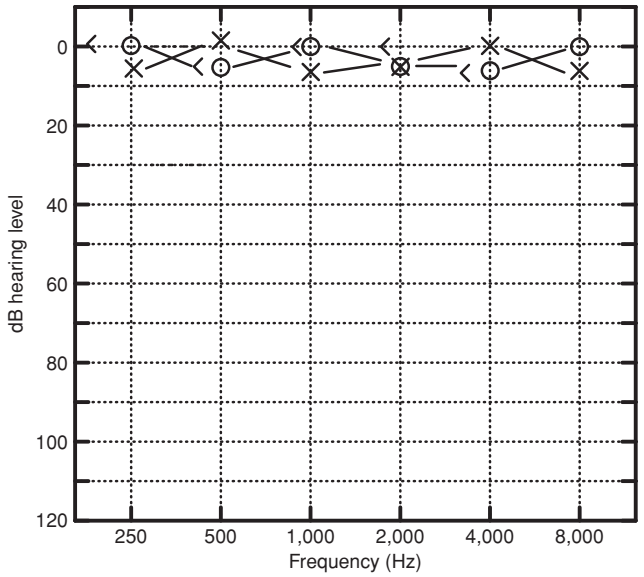


FIGURE 4.3 Audiogram depicting normal-hearing sensitivity.

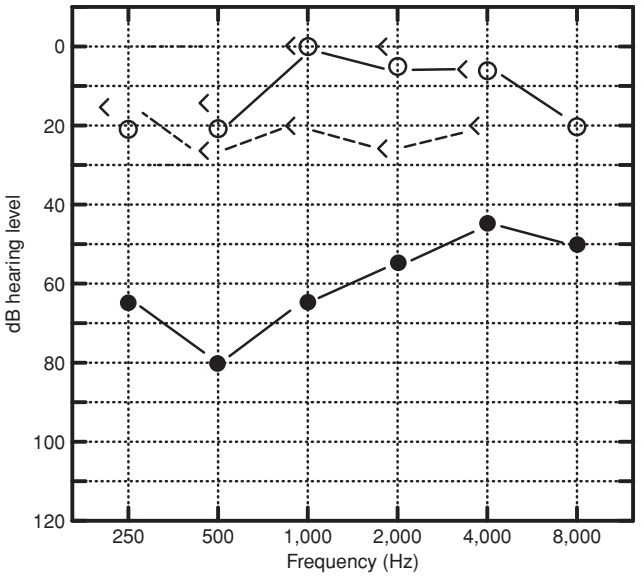


FIGURE 4.4 Audiogram depicting CHL from osteomas. Air conduction: presurgery thresholds represented with *filled circles* and postsurgery thresholds represented with *open circles.* Bone conduction: presurgery thresholds connected with a *dotted line,* postsurgery thresholds not connected. [Modified from Pinsker OT. [1972] Otological correlates of audiology. In: Katz J, ed. *Handbook of Clinical Audiology.* Baltimore, MD: Williams and Wilkins, http://lww.com by permission.]

are without disorder that would hinder any of the bone conduction components. Again owing to calibration, bone conduction thresholds will be near 0 dB HL and be similar to air conduction thresholds (±10 dB). Figure 4.3 shows an example audiogram of a patient with normal-hearing sensitivity.

CHL with Air–Bone Gaps of Outer Ear Origin

Outer ear occlusive disorder may hinder air conduction energy from reaching the inner ear. In such cases, air conduction thresholds would be poorer than 0 dB HL, at least at some frequencies, to a degree dictated by the occlusive disorder. Bone conduction thresholds, in contrast, may be unaffected if the outer ear bone conduction component is not hindered. Bone conduction thresholds would be near 0 dB HL and accurately reflect sensory/neural hearing (sensory/neural reserve or true sensory/neural capability). The result is a hallmark of CHL: air conduction thresholds >10 dB HL lower (poorer) than normal bone conduction thresholds. The maximum air–bone gap is approximately 60 dB; higher intensity air-conducted sound waves set the skull into vibration and induce bone conduction hearing thus limiting the maximum difference between air and bone conduction thresholds (Bekesy, 1960).

However, bone conduction thresholds are often improved by occlusive disorders due to the occlusion effect. In such cases, bone conduction thresholds overestimate sensory/neural reserve. In rarer cases, outer ear occlusive disorder may interfere with the outer or middle ear bone conduction components and thus lower bone conduction thresholds, a so-called pseudoSNHL because neither hair cell nor neuron damage is the cause (Hall and Croutch, 2008). In such cases, bone conduction thresholds underestimate sensory/neural reserve. Figure 4.4 shows an audiogram example involving occlusive osteomas; the presurgery audiogram shows CHL, but with bone conduction thresholds that underestimated the true sensory/neural reserve revealed by postsurgery bone conduction thresholds. Cerumen impaction that loads (adds mass to) the tympanic membrane can cause pseudoSNHL (Hall and Croutch, 2008; Tonndorf, 1968). Anderson and Barr (1971) reported pseudoSNHL with partial cerumen occlusion of the outer ear canal, though they attributed it to earphone artifact.

CHL with Air–Bone Gaps of Middle Ear Origin

Middle ear disorder may hinder air conduction energy from reaching the inner ear. In such cases, air conduction thresholds would be poorer than 0 dB HL, at least at some frequencies, to a degree dictated by the middle ear disorder. Bone conduction thresholds, in contrast, may be unaffected if the middle ear bone conduction component is not significantly hindered. Bone conduction thresholds would therefore be near 0 dB HL and accurately reflect sensory/neural reserve or be improved due to the occlusion effect.

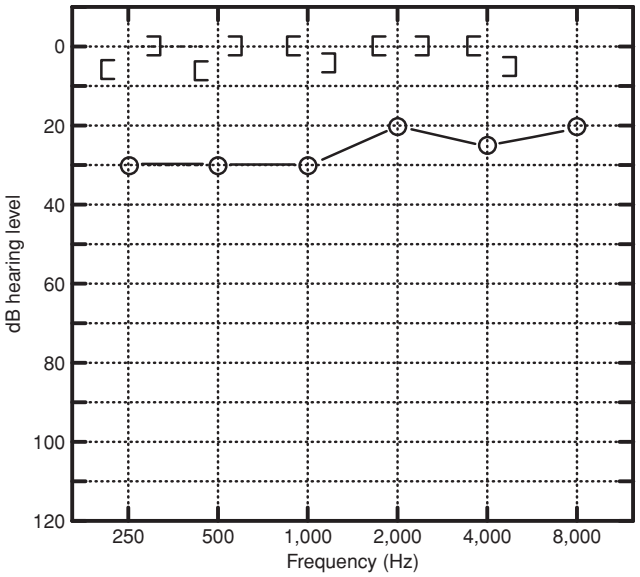


FIGURE 4.5 Audiogram representing the mean CHL from a group of children. [Modified from Schlauch RS, Nelson P. [2009] Puretone evaluation. In: Katz J, Medwetsky L, Burkard R, Hood L, eds. *Handbook of Clinical Audiology.* Philadelphia, PA: Lippincott Williams and Wilkins, http://lww.com by permission.]

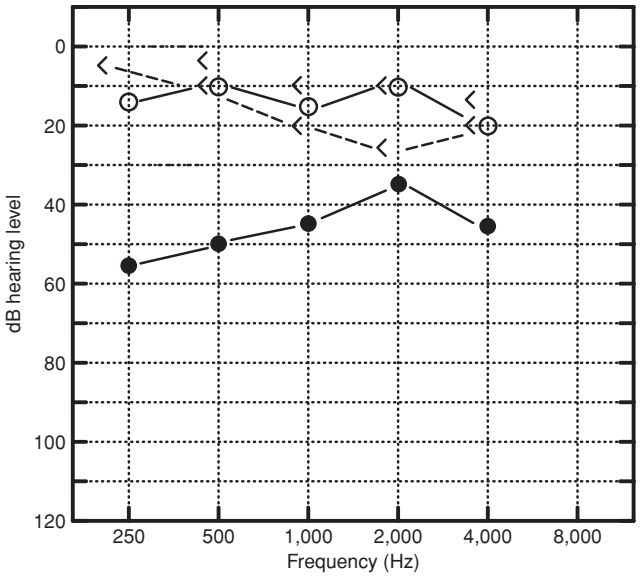


FIGURE 4.6 Audiogram depicting CHL from otosclerosis. Air conduction: presurgery thresholds represented with *filled circles* and postsurgery thresholds represented with *open circles.* Bone conduction: presurgery thresholds connected with a *dotted line,* postsurgery thresholds not connected. [Modified from Dirks D. [1985]. Bone-conduction testing. In: Katz J, ed. *Handbook of Clinical Audiology.* Baltimore, MD: Williams and Wilkins, http://lww.com by permission.]

The resulting air–bone gaps can be as great as approximately 60 dB. Air–bone gaps can be seen with many middle ear disorders; an example of a CHL audiogram is shown in Figure 4.5.

However, middle ear disorder often hinders the middle ear contribution to bone conduction and thus lowers (makes poorer) bone conduction thresholds. The result is a pseudoSNHL in addition to the CHL, with bone conduction thresholds underestimating sensory/neural reserve. For example, ossicular fixation caused by otosclerosis predictably manifests as lower (poorer) than normal bone conduction thresholds at the approximate middle ear resonant frequency of 2,000 Hz (Carhart, 1950). The air–bone gaps one expects with CHL may therefore be reduced or obliterated at and near 2,000 Hz. For stapedial fixation caused by otosclerosis, this pseudoSNHL is known as the Carhart notch. An audiogram example is shown in Figure 4.6; note the Carhart notch and CHL presurgery and the restoration of normal air and bone conduction thresholds postsurgery consistent with restoration to more normal middle ear resonance.

The middle ear bone conduction component can be affected by other disorders as well. For example, Dirks and Malmquist (1969) reported pseudoSNHL in addition to CHL in a case of mallear fixation. Similarly, pseudoSNHL in addition to CHL has been reported for subjects with otitis media (Carhart, 1950; Hall and Croutch, 2008). Yasan (2007) reported 1,000 Hz and in some cases 2,000 Hz bone conduction notches in patients with otitis media, and Kumar et al. (2003) reported 2,000 Hz bone conduction notches in patients with glue ear. Apparently, effusion may produce stiffening or loading effects, thus hindering the middle ear bone conduction component (Tonndorf, 1968). An example of CHL caused by otitis media is shown in Figure 4.7; note the pretreatment pseudoSNHL and CHL, and the resolution of the same posttreatment.

It should be noted here that true SNHL with otitis media has also been proposed. Pathogens in middle ear effusion may pass through the round window and cause damage to cochlear hair cells, and because of the round window proximity to the basal turn of the cochlea, high-frequency SNHL might result (Paparella et al., 1984).

SNHL

Neither sensory nor neural disorders hinder energy from reaching the inner ear by the air conduction route or by any of the bone conduction routes. Changes in air and bone conduction thresholds are affected only by the damage to sensory/neural structures which will lower (make poorer) air and bone conduction thresholds similarly. Air and bone conduction thresholds will therefore be similar (±10 dB). Figure 4.8 shows an example audiogram of a patient with SNHL from presbycusis.

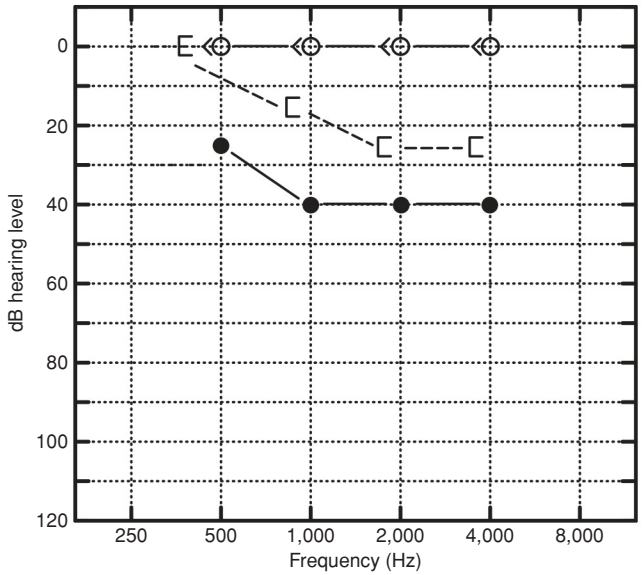


FIGURE 4.7 Audiograms depicting CHL from otitis media. Air conduction: pretreatment thresholds represented with *filled circles* and post-treatment thresholds represented with *open circles.* Bone conduction: pretreatment thresholds connected with a *dotted line,* post-treatment thresholds not connected. [Modified from Hall CM, Croutch C. [2008] Pseudosensory-neural hearing loss. *Hear Rev.* 16[1], 18–22, by permission.]

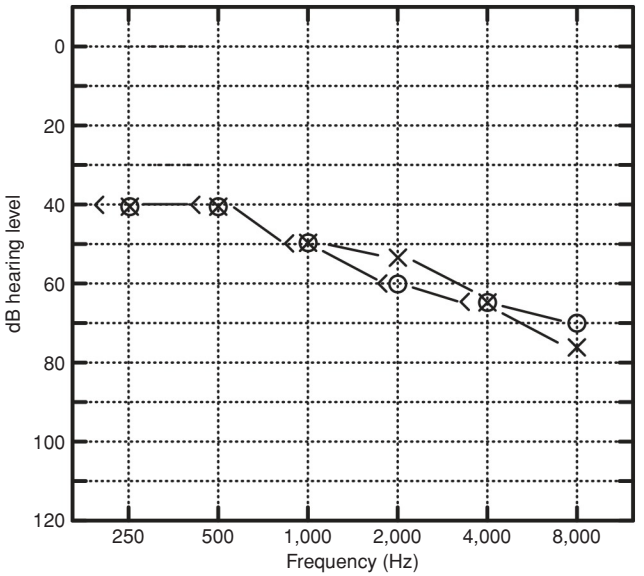


FIGURE 4.8 Audiogram depicting a sensory/neural hearing loss from presbycusis. [Modified from Harrell RW, Dirks D. [1994] In: Katz J, ed. *Handbook of Clinical Audiology.* Philadelphia, PA: Lippincott, Williams and Wilkins, http://lww.com by permission.]

MHL with Air–Bone Gaps of Outer or Middle Ear Origin

Patients may have an MHL. An MHL audiogram will therefore show evidence of both the SNHL (affected air and bone conduction thresholds) and the CHL (air–bone gaps >10 dB). Bone conduction thresholds may accurately reflect sensory/neural reserve or a pseudoSNHL may underestimate sensory/neural reserve. An MHL audiogram example is shown in Figure 4.9.

Superior Semicircular Canal Dehiscence with PseudoSNHL and Air–Bone Gaps of Inner Ear Origin

SSCD is a thinning or absence of the temporal bone over the membranous labyrinth of the superior semicircular canal. This condition opens a third elastic membranous inner ear window at the dehiscence, the other two windows of course being the oval and round windows of the cochlea (Merchant et al., 2007). The audiogram manifestation of SSCD may mimic CHL or MHL, with air–bone gaps that could approach 60 dB (Chien et al., 2012). Air conduction thresholds may be adversely affected because energy reaching the inner ear by the air conduction route is shunted away from the cochlea through the dehiscence, typically manifesting in the lower test frequencies consistent with a mathematical model analysis based on the anatomical dimensions of the inner ear (Merchant et al., 2007). Bone conduction thresholds, in contrast, may be improved by skull content sound pressure transmissions

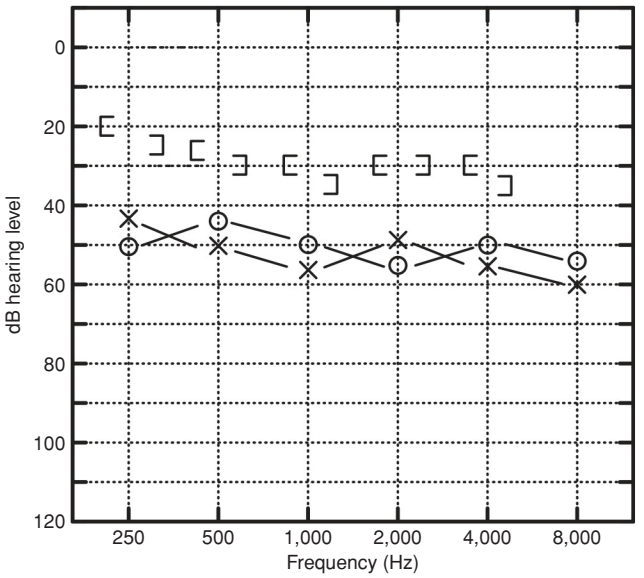


FIGURE 4.9 Audiogram depicting an MHL. [Modified from Schlauch RS, Nelson P. [2009] Puretone evaluation. In Katz J, Medwetsky L, Burkard R, Hood L, eds. *Handbook of Clinical Audiology.* Philadelphia, PA: Lippincott Williams and Wilkins, http://lww.com by permission.]

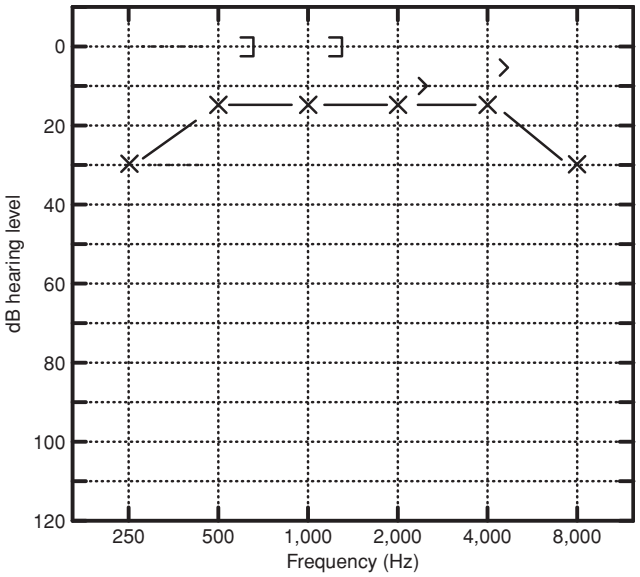


FIGURE 4.10 Audiogram depicting hearing loss from superior semicircular canal dehiscence.

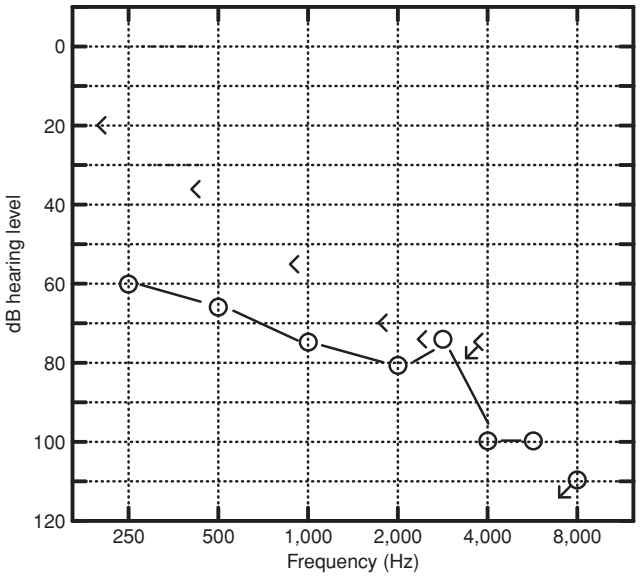


FIGURE 4.11 Audiogram depicting hearing loss from a large vestibular aqueduct.

through the dehiscence, that is, augmentation of the inner ear bone conduction component. The term air–bone gap of inner ear origin has been used to describe these findings (Attias et al., 2012). This is a useful term as it emphasizes that the air–bone gaps do not reflect either a CHL or MHL. Figure 4.10 shows an example audiogram from a patient with SSCD.

Large Vestibular Aqueducts with SNHL and Air–Bone Gaps of Inner Ear Origin

Large vestibular aqueducts (LVA) cause SNHL with air–bone gaps of inner ear origin (Attias et al., 2012; Jackler and De La Cruz, 1989). The cause of SNHL with LVA is unclear; it may result from traumatic endolymph pressure from the endolymphatic duct and sac that damages hair cells, or by endolymph electrolyte content that is harmful to the hair cells or stria vascularis (Campbell et al., 2011; Jackler and De La Cruz, 1989; Levinson et al., 1989). The audiometric findings for LVA may also be influenced by the third-window effect similar to SSCD. Air–bone gaps of inner ear origin are possible: Air conduction thresholds may be lowered (made poorer) because some energy is shunted away from the cochlea through the LVA, whereas bone conduction thresholds may be unaffected or improved by sound pressure transmissions through the skull contents to the LVA, (Attias et al., 2012; Merchant et al., 2007). Therefore, as with SSCD, the air–bone gaps seen with LVA do not reflect outer ear occlusion or middle ear disorder as in CHL or MHL. Figure 4.11 shows an example audiogram from a patient with LVA.

Intracranial Hypertension with PseudoSNHL

Intracranial hypertension with abnormal cerebrospinal fluid flow has been associated with a number of neurologic conditions including syringohydromyelia, Chiari malformations, trauma, tumors, arachnoiditis, subarachnoid hemorrhages, meningitis, and multiple sclerosis (Steiger et al., 2007). Resulting audiologic symptoms may include whooshing pulsatile tinnitus and low-frequency pseudoSNHL. The pulsatile tinnitus may arise from circle of Willis blood flow or pulsations of the walls of the dural sinuses (Rudnick and Sismanis, 2005), which travel through the cochlear or vestibular aqueducts to the cochlea (Marchbanks et al., 2005). The pseudoSNHL might be attributable to masking from the pulsatile tinnitus (Rudnick and Sismanis, 2005; Steiger et al., 2007) or from elevated cochlear fluid pressure stiffening the basilar, oval, and round window membranes (Sismanis, 1987). Stiffened cochlear membranes, in turn, may interfere with cochlear fluid motion and thus hinder the inner ear bone conduction component. Figure 4.12 shows an audiogram of a patient with intracranial hypertension.

TECHNICAL CLINICAL CAVEATS

Vibrotactile Responses

It is possible for a patient to feel bone conductor diaphragm vibrations during bone conduction evaluation, especially at high intensities and at lower test frequencies (Nober, 1964). When a patient responds to stimuli felt but not heard, the responses are called vibrotactile. Vibrotactile responses must not be recorded as auditory thresholds as two possible errors

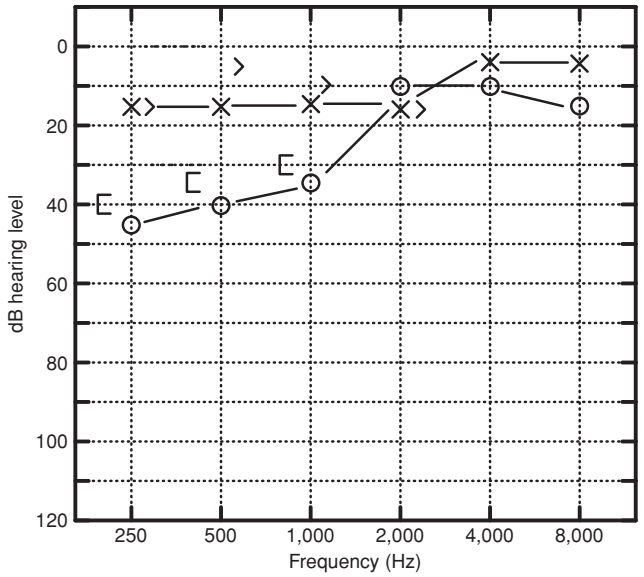


FIGURE 4.12 Audiogram depicting pseudoSNHL from intracranial hypertension. [Adapted from Steiger JR, Saccone PA, Watson KN. (2007) Assessment of objective pulsatile tinnitus in a patient with syringohydromyelia. *J Am Acad Audiol.* 18(3), 197–206.] Used with permission of the American Academy of Audiology.

might result. First, bone conduction vibrotactile responses could be better than the air conduction thresholds and therefore might result in erroneous air–bone gaps and misdiagnoses. Second, recording vibrotactile responses as bone conduction thresholds might erroneously suggest hearing in patients who are deaf (Nober, 1964). Individual sensitivity to vibrotactile sounds is variable (Boothroyd and Cawkwell, 1970). Perhaps the only way to know if responses are vibrotactile is to ask patients; this is recommended, especially when bone conduction thresholds appear inconsistent with other audiometric findings or history.

Interaural Attenuation, Masking, and the Occlusion Effect

Audiologists typically target a test ear for evaluation while being vigilant for the possibility of the patient hearing in the nontest ear. This vigilance is warranted during bone-conduction threshold evaluation; transducer placement on the test ear side mastoid bone results in activation of bone conduction bilaterally. The so-called cross-over from the test ear side to the nontest ear can occur with minimal interaural attenuation, ranging from 0 dB at 250 Hz to 15 dB at 4,000 Hz (Studebaker, 1967). Caution compels most audiologists to assume the worst-case scenario of 0 dB interaural attenuation, that is, equal tone intensity at the test and nontest ears. Bone conduction thresholds are therefore not typically considered to be ear-specific unless sufficient masking noise is delivered to the nontest ear. There are many variables to consider while masking, such as when to mask, masking noise type, masker intensity, and accounting for the occlusion effect caused by the earphone on the nontest ear (see Chapter 6 for details).

Mastoid versus Forehead Placement

During bone conduction hearing evaluation, audiologists may place the bone vibrator on either the mastoids or the foreheads of their patients. Mastoid placement is preferred by most audiologists (Martin et al., 1998). Perhaps the main advantage of mastoid placement is that the resulting bone conduction thresholds are up to 14.7 dB less than bone conduction thresholds measured with forehead transducer placement (Table 4.1). This allows for a greater testing range from threshold to equipment intensity limits or vibrotactile sensation. Moreover, vibrations from mastoid bone vibrator placement are in the same plane as middle ear ossicular motion, therefore engaging the middle ear bone conduction mechanism. This allows the audiologist

TABLE 4.1

Mean Differences between Bone Conduction Thresholds Measured with Forehead and Mastoid Bone Vibrator Placement[a]

	Frequency in Hz					
	250	500	1,000	2,000	3,000	4,000
	Forehead–Mastoid Corrections in dB					
ANSI S3.43-1992	12	14	8.5	11.5	12	8
Frank (1982)	14.3	14.7	8.7	12	12.4	13.5

ANSI, American National Standards Institute.

[a]The correction should be subtracted from the forehead thresholds to approximate mastoid thresholds.

Source: From Vento B, Durrant JD. (2009) In: Katz J, Medwetsky L, Burkard R, Hood L, eds. *Handbook of Clinical Audiology*. Philadelphia, PA: Lippincott Williams and Wilkins, http://lww.com by permission.

to record evidence of changes in the middle ear bone conduction component, for example, the middle ear resonance changes that are likely to occur with otosclerosis. Not surprisingly, audiometers in most clinics are calibrated for mastoid placement.

Forehead placement can be used if correction factors from Table 4.1 are applied or if the audiometer is calibrated for forehead transducer placement. Audiologists who prefer forehead transducer placement should consider purchasing a specifically designed bone vibrator and headband. Forehead placement has advantages, including low intrasubject and intersubject variability because of the less variable forehead placement surface and more uniform nonpneumatized forehead bone (Dirks, 1964). Also, vibrations from forehead placement are perpendicular to middle ear ossicular motion and may not engage the middle ear bone conduction mechanism as with mastoid placement. The resulting forehead bone conduction thresholds should be relatively unaffected by the changes in middle ear resonance and, in cases of ossicular fixation, reflect a truer measure of cochlear reserve than bone conduction thresholds obtained during mastoid transducer placement. Figure 4.13 shows an example audiogram with forehead and mastoid bone vibrator placement for a patient with ossicular fixation.

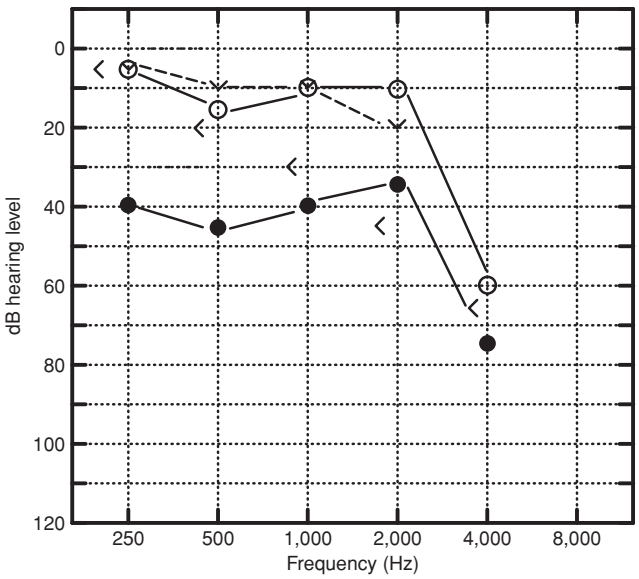


FIGURE 4.13 Audiogram depicting hearing loss from ossicular (mallear) fixation. Air conduction: presurgery thresholds represented with *filled circles* and postsurgery thresholds represented with *open circles.* Bone conduction: presurgery forehead placement thresholds connected with a *dotted line,* presurgery mastoid placement thresholds not connected. [Modified from Dirks D. (1985) Bone-conduction testing. In Katz J, ed. *Handbook of Clinical Audiology.* Baltimore, MD: Williams and Wilkins, http://lww.com by permission.]

Threshold Accuracy and the Air–Bone Gap

Throughout this chapter significant air–bone gaps were defined as >10 dB. However, patient response variability can result in underestimated or exaggerated air–bone gaps and even bone–air threshold gaps. Studebaker (1967), for example, calculated the standard deviation of air–bone gaps at 5 dB and noted that air–bone threshold gaps of ≥15 dB can sometimes be seen in the absence of CHL. Similarly, Margolis (2008) calculated a hypothetical air–bone threshold gap distribution based on the independent variability of air and bone conduction thresholds. Significant air–bone and even bone–air threshold gaps were predicted, of course, with greater threshold gaps occurring less frequently. Moreover, Margolis reported apparent tester bias; when testing patients with SNHL an expert audiologist measured more air–bone threshold gaps ≤5 dB than the distribution predicted. Audiologists should not rigidly adhere to untenable expectations regarding air–bone threshold gaps.

CONCLUSION

Bone conduction threshold evaluation is an integral component of the basic audiologic examination. When bone conduction thresholds are interpreted with an understanding of air and bone conduction hearing, more accurate site-of-lesion and etiology diagnoses can be made. It is hoped that with this chapter the author has informed and motivated readers to that end.

FOOD FOR THOUGHT

1. How might the air–bone gaps of patients with outer ear occlusion differ from the air–bone gaps of patients with middle ear disorder?
2. Why are air–bone gaps usually but not always indicative of CHL?
3. Why is worsening bone conduction hearing not always indicative of hair cell and/or neural disorder?

KEY REFERENCES

thePoint A full list of references for this chapter can be found at http://thePoint.lww.com. Below are the key references for this chapter.

American National Standards Institute. (2003) *Maximum Permissible Ambient Noise Levels for Audiometric Test Rooms (ANSI S3.1-1999; Rev. ed.).* New York, NY: Author.

American National Standards Institute. (2004) *Specifications for Audiometers (ANSI S3.6-2004).* New York, NY: Author.

American Speech-Language-Hearing Association. (1990) *Guidelines for Audiometric Symbols.* Rockville, MD: Author.

American Speech-Language-Hearing Association. (2005) *Guidelines for Manual Pure-Tone Threshold Audiometry.* Rockville, MD: Author.

Anderson H, Barr B. (1971) Conductive high-tone hearing loss. *Arch Otolaryngol.* 93(6), 599–605.

Attias J, Ulanovski D, Shemesh R, Kornreich L, Nageris B, Preis M, Peled M, Efrati M, Raveh E. (2012) Air-bone gap component of inner-ear origin in audiograms of cochlear implant candidates. *Otol Neurotol.* 33, 512–517.

Barany E. (1938) A contribution to the physiology of bone conduction. *Acta Otolaryngol.* (suppl 26), 1–4.

Bekesy G. (1960) *Experiments in Hearing.* New York, NY: McGraw Hill Book Co.

Boothroyd A, Cawkwell S. (1970) Vibrotactile thresholds in pure tone audiometry. *Acta Otolaryngol.* 69(1–6), 381–387.

Campbell AP, Adunka OF, Zhou B, Qaqish BF, Buchman CA. (2011) Large vestibular aqueduct syndrome. *Laryngoscope.* 121, 352–357.

Carhart R. (1950) Clinical application of bone conduction audiometry. *Arch Otolaryngol.* 51, 798–808.

Chien WW, Janky K, Minor LB, Carey JP. (2012) Superior semicircular canal dehiscence size: multivariate assessment of clinical impact. *Otol Neurotol.* 33, 810–815.

de Jong M, Perez R, Adelman C, Chordekar S, Rubin M, Kirksunov L, Sohmer H. (2011) Experimental confirmation that vibrations at soft tissue conduction sites induce hearing by way of a new mode of auditory stimulation. *J Basic Clin Physiol Pharmacol.* 22(3), 55–58.

Dirks D. (1964) Factors related to bone conduction reliability. *Arch Otolaryngol.* 79, 551–558.

Dirks D, Malmquist C. (1969) Comparison of frontal and mastoid bone conduction thresholds in various conduction lesions. *J Speech Hear Res.* 12, 725–746.

Feldmann H. (1970) A history of audiology: a comprehensive report and bibliography from the earliest beginnings to the present. In: Tonndorf J, ed. *Translations of the Beltone Institute for Hearing Research.* Chicago, IL: The Beltone Institute for Hearing Research; pp 11–111.

Frank T. (1982) Forehead versus mastoid threshold differences with a circular tipped vibrator. *Ear Hear*. 3, 91–92.

Hall CM, Croutch C. (2008) Pseudosensory-neural hearing loss. *Hear Rev.* 16(1), 18–22.

Herzog H, Krainz W. (1926) Das knochenleitungsproblem. *Z Hals Usw Heilk.* 15, 300–306.

Hulecki LR, Small SA. (2011) Behavioral bone conduction thresholds for infants with normal hearing. *J Am Acad Audiol.* 22, 81–92.

Jackler RK, De La Cruz A. (1989) The large vestibular aqueduct syndrome. *Laryngoscope.* 99(12), 1238–1243.

Kumar M, Maheshwar A, Mahendran S, Oluwasamni, Clayton MI. (2003) Could the presence of a Carhart notch predict the presence of glue at myringotomy? *Clin Otolaryngol.* 28(3), 183–186.

Levinson MJ, Parisier SC, Jacobs M, Edelstein DR. (1989) The large vestibular aqueduct syndrome in children: a review of 12 cases and the description of a new clinical entity. *Arch Otolaryngol.* 115, 54–58.

Marchbanks RJ, Burge DM, Martin AM, Bateman DE, Pickard J, Brightwell AP. (2005) The relationship between intracranial pressure and tympanic membrane displacement. *Br J Audiol.* 24(2), 123–129.

Margolis RH. (2008) The vanishing air-bone gap: audiology's dirty little secret. *Audiology Online.* Available online at: http://www.audiologyonline.com/articles/vanishing-air-bone-gap-audiology-901&referer=www.clickfind.com.au

Martin FN, Champlin CA, Chambers JA. (1998) Seventh survey of audiometric practices in the United States. *J Am Acad Audiol.* 9(2), 95–104.

Merchant SN, Rosowski JJ, McKenna MJ. (2007) Superior semicircular canal dehiscence mimicking otosclerotic hearing loss. *Adv Otorhinolaryngol.* 65, 137–145.

Nober EH. (1964) Pseudoauditory bone conduction thresholds. *J Speech Hear Disord.* 29, 469–476.

Paparella MM, Morizono T, Le CT, Mancini F, Sipilä P, Choo YB, Lidén G, Ki CS. (1984) Sensory-neural hearing loss in otitis media. *Ann Otol Rhinol Laryngol.* 93, 623–629.

Rudnick E, Sismanis A. (2005) Pulsatile tinnitus and spontaneous cerebrospinal fluid rhinorrhea: indicators of benign intracranial hypertension syndrome. *Otol Neurotol.* 26(2), 166–168.

Sismanis A. (1987) Otologic manifestations of benign intracranial hypertension syndrome. *Laryngoscope.* 97(8, Pt 2, suppl 42), 1–17.

Steiger JR, Saccone P, Watson KN. (2007) Assessment of objective pulsatile tinnitus in a patient with syringohydromyelia. *J Am Acad Audiol.* 18(3), 197–206.

Stenfelt S, Goode RL. (2005) Bone conducted sound: physiological and clinical aspects. *Otol Neurotol.* 26, 1245–1261.

Stenfelt S, Puria S, Hate N, Goode RL. (2003) Basilar membrane and osseous spiral lamina motion in human cadavers with air and bone conduction stimuli. *Hear Res.* 181, 131–143.

Studebaker GA. (1967) Clinical masking of the nontest ear. *J Speech Hear Disord.* 32, 360–371.

Tonndorf J. (1968) A new concept of bone conduction. *Arch Otolaryngol.* 87, 49–54.

Yasan H. (2007) Predictive role of Carhart's notch in pre-operative assessment for middle-ear surgery. *J Laryngol Oto.* 121, 219–221.

CHAPTER 5

Speech Audiometry

Rachel McArdle and Theresa Hnath-Chisolm

INTRODUCTION

Auditory assessment using speech stimuli has a long history in the evaluation of hearing. As early as 1804, there were scientific attempts to study hearing sensitivity for speech by assessing which classes of speech sounds an individual could hear: (1) vowels; (2) voiced consonants; or (3) voiceless consonants. In 1821, Itard, who is well known for his contributions to deaf education, differentiated individuals who were hard of hearing from those who were deaf by whether a person could understand some or none of a spoken message (Feldmann, 1970). This early focus on hearing for speech continued through the 19th century, and by the mid-1920s, the first speech audiometer, the Western Electric 4 A, which incorporated a phonograph with recorded digit speech stimuli, was employed in large-scale hearing screenings (Feldmann, 1970).

Hearing and understanding speech have unique importance in our lives. For children, the ability to hear and understand speech is fundamental to the development of oral language. For adults, difficulty in detecting and understanding speech limits the ability to participate in the communication interactions that are the foundation of numerous activities of daily living. Measures of sensitivity and understanding form the basis of speech audiometry. This chapter focuses on providing information that can lead to the implementation of evidence-based best practices in speech audiometry.

WHAT IS SPEECH AUDIOMETRY?

Speech audiometry refers to procedures that use speech stimuli to assess auditory function (Konkle and Rintelmann, 1983). Since the classic work of Carhart (1951), speech audiometry has involved the assessment of sensitivity for speech as well as assessment of clarity when speech is heard. These concepts were described by Plomp (1978), in his framework of hearing loss, as an audibility component (i.e., loss of sensitivity) and a distortion component (i.e., loss of clarity). The audibility component is quantified through assessment of speech recognition abilities in quiet. The distortion component is a reduction in the ability to understand speech, especially in a background of noise, regardless of the presentation level. Quantifying the distortion component typically involves percent correct recognition at suprathreshold levels for the speech recognition score (SRS). More recently, the signal-to-noise ratio (S/N) at which 50% correct recognition is achieved has been recommended instead of the traditional SRS (Killion et al., 2004; Wilson, 2003). Before discussing measurement of speech thresholds and speech recognition in quiet and noise, general considerations in speech audiometry related to terminology, stimulus calibration, presentation methods, response modes, and presentation levels are discussed.

SPEECH AUDIOMETRY TERMINOLOGY

There are two types of threshold measures using speech stimuli: speech detection threshold (SDT) and speech recognition threshold (SRT). SDT, as defined by the American Speech-Language-Hearing Association (ASHA, 1988), is an estimate of the level at which an individual perceives speech to be present 50% of the time and should be reported in decibels hearing level (dB HL). SDTs are commonly used to establish the level for awareness of speech stimuli by infants, young children, or adults who cannot respond verbally or whose speech recognition ability is so poor that they are unable to recognize spondaic (i.e., compound) words to obtain an SRT. SDT is sometimes called a speech awareness threshold (SAT), although SDT is the term preferred by ASHA (1988).

The SRT is an estimate of the level at which an individual can repeat back spondaic words (e.g., hotdog, baseball) 50% of the time; it is most commonly reported in dB HL or decibels sound pressure level (dB SPL). The most common suprathreshold measure in quiet is the SRS or word recognition score and is generally measured in percent correct at a level (dB HL) relative to either the SRT or an average of puretone thresholds. Word recognition has been referred to as speech discrimination; however, discrimination infers that an individual is judging between two or more specific stimuli, which is not the task in most suprathreshold speech recognition measures.

GENERAL CONSIDERATIONS FOR SPEECH AUDIOMETRY

Audiometers have to meet calibration standards set forth by the American National Standards Institute (ANSI, 2004). In

addition, recorded materials used as stimuli for speech audiometry must meet the ANSI standards (ANSI, 2004, Annex B). To reduce error of measurement and increase consistency from clinic to clinic, speech measures should employ accepted calibration procedures, methods and modes of presentation, test instructions, and response modes.

Method of Presentation

Historically, VU meters were used for the tester to "monitor" the energy of his or her voice while presenting speech stimuli via the speech audiometer. The development of analog audiotape followed by compact disc technology was instrumental in facilitating standardization of word lists used in speech audiometry (Wilson et al., 1990). ASHA guidelines (1988) for speech thresholds indicate that the use of recorded stimuli is preferred. The majority of audiologists, however, who responded to a survey of audiometric practices (Martin et al., 1998), still report using monitored live speech to determine thresholds for speech. Of the 218 audiologists who completed the survey, 94% reported using monitored live voice test methods.

We feel that it is even more important to use recorded speech for SRSs. Digitized speech recordings improve both the intrasubject and intersubject precision of threshold and suprathreshold measures by providing a consistent level for all test items and consistent speech patterns between patients. The reliability of a given set of speech stimuli can vary across speakers and across test time for a single speaker. Hood and Poole (1980) found that a speaker had a significant impact on the difficulty of particular monosyllabic word lists. Similarly, Roeser and Clark (2008) found significant differences in performance when the same subjects were tested via recorded materials and monitored live voice with the latter showing better performance. Other studies have found variability in recognition performance as a function of speaker–list interactions (Asher, 1958; Hirsh et al., 1954) such that the acoustic waveforms of two speakers can cause differences in recognition performance even when the word lists are the same. The reported contribution of the speaker to the recognition performance of each listener reinforces previous reports by Kruel et al. (1969), who stated that word lists should be thought of as a group of utterances and not as a written list of words because speaker differences may affect a person's performance on a particular list.

Presentation Level

Psychometric Function

Understanding the influence of presentation level on performance is best described by psychometric functions. In simple terms, a function is when you measure a change in a dependent variable (*y*-axis; e.g., number or percent correct, which is a psychological variable) based on changes of an independent variable (*x*-axis; e.g., presentation level in HL or SNR, which is a physical variable). Figure 5.1 is a graphic display of two psychometric functions. The function to the left is an SRT function whereas the function to the right is an SRS function. The characteristic audiogram thought to accompany this type of performance can be seen in the lower right quadrant. Presentation level is on the *x*-axis (dB HL), whereas percent correct performance is on the *y*-axis. As can be seen for both functions, the percent correct is low when the level is low, and as the level is increased, the percent correct increases. The dashed line in Figure 5.1 highlights the 50% point on the functions and indicates that an SRT was obtained about 40 dB HL. Also illustrated in Figure 5.1 is that the maximum point of performance (100%) was reached at approximately 75 dB HL for the SRS function. As the level is increased above 75 dB HL, no change in performance is observed. The highest percent correct score obtained by an individual is often referred to as PB_{max}, because historically SRSs were obtained using phonetically balanced (PB) word lists. Further discussion of PB word lists can be found later in this chapter under the section titled "Speech Recognition in Quiet."

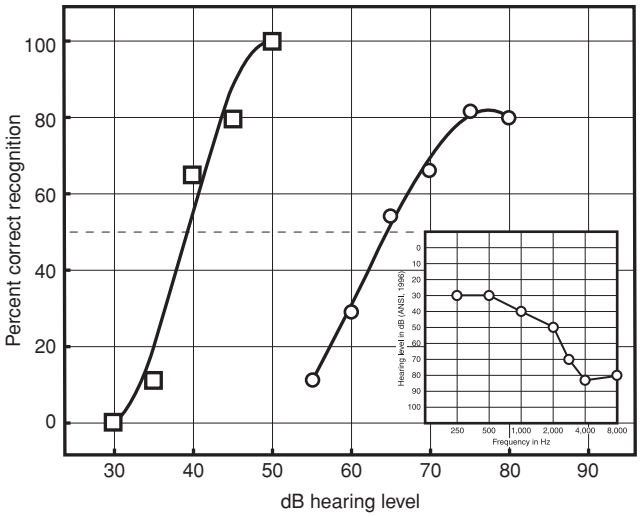


FIGURE 5.1 Psychometric functions of word recognition performance measured in percent correct (ordinate) for a listener with hearing loss as a function of presentation level (abscissa). The *dashed line* indicates the 50% point. The function to the left is the SRT function whereas the function to the right is the SRS function.

Because listeners with normal hearing, on average, achieve maximal performance on a speech recognition task at 30 to 40 dB sensation level (SL) re: SRT, clinicians will often test their patients at one of these levels, assuming this will result in maximal performance for the listener. Assessing only a single level may provide limited diagnostic or rehabilitative information. Conversely, assessing performance at multiple presentation levels for individuals with sensory/neural hearing loss provides greater diagnostic information

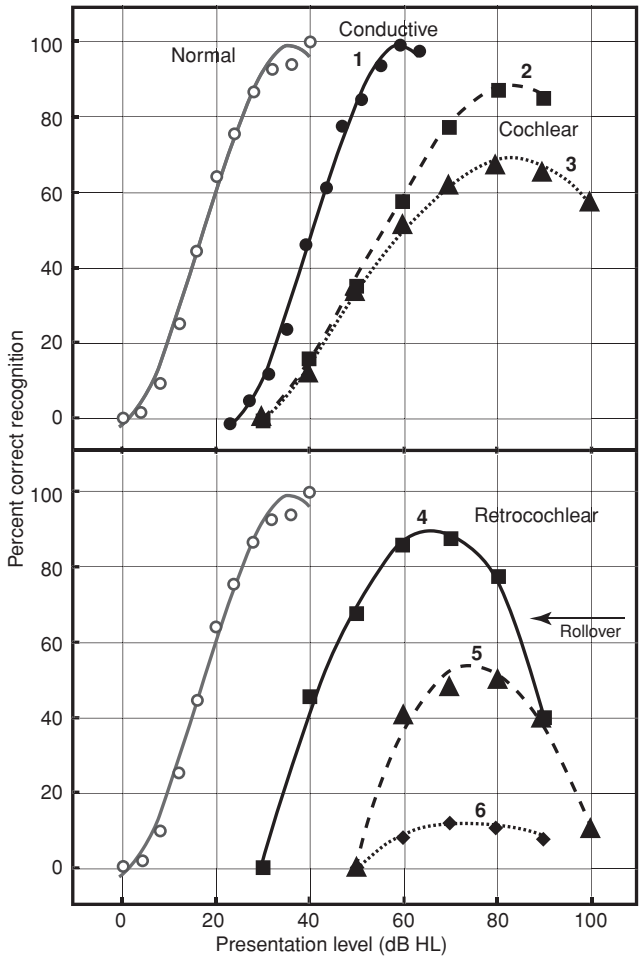


FIGURE 5.2 Psychometric functions of word recognition performance illustrating various types of hearing loss can be seen in both panels as a function of percent correct (ordinate) and presentation level (abscissa). The top panel illustrates a sample psychometric function for a listener with normal hearing (*open circles*), conductive hearing loss (curve *#1*), and cochlear hearing loss (curves *#2* and *#3*). The bottom panel shows possible psychometric functions for retrocochlear hearing loss (curves #4, #5, and #6). (Adapted from Department of Veterans Affairs (1997).)

as demonstrated by the example functions drawn in Figure 5.2. In the top panel of Figure 5.2, curve #2 shows a function that reaches maximum performance (88%) at 80 dB HL and plateaus through 100 dB HL. In the bottom panel of Figure 5.2, curve #4 shows a function that reaches maximum performance (85%) at approximately 60 dB HL, and then performance decreases as level is increased, which is depicted by a rollover in the shape of the function.

Also of importance when describing performance in terms of the psychometric function is the slope of the function. The slope of the function is typically calculated from the dynamic portion of the function that ranges between 20% and 80%. Scores below 20% are often affected by floor effects because the task difficulty is too great to show subtle changes in performance, whereas scores above 80% are often affected by ceiling effects because the task difficulty is too easy to be sensitive to performance changes. For an individual with a steep slope, the measurements should be made in small (dB) steps to obtain valid results, whereas a shallow function allows for larger step sizes to obtain valid results. When selecting test material, it is best to choose stimuli that produce a steep function, which suggests the materials are homogeneous with respect to the task (Wilson and Margolis, 1983).

Response Mode

The response mode for speech audiometry is generally verbal. However, for SDT the response mode can be similar to that of puretone thresholds, where patients can push a button or raise their hand when they hear the speech stimuli. A written response is generally avoided because of the increased test time and reliance on the patient's ability to write and spell. For testing children or nonverbal individuals, see Chapters 24 and 31.

SPEECH RECOGNITION THRESHOLD

Stimuli

Spondaic words are generally used for obtaining SDTs and SRTs and are recommended by ASHA (1988). Spondaic (adjective) words or spondees (noun) are two-syllable words with equal stress on both syllables. Lists of spondaic words for assessing hearing loss for speech were first developed at the Harvard Psychoacoustic Laboratories (PAL) by Hudgins et al. (1947). Criteria for selection of words included a high level of word familiarity, phonetic dissimilarity, and homogeneity with respect to audibility. Of the original 42 spondees identified by Hudgins et al. (1947), 36 of the most familiar were used in the development of the Central Institute for the Deaf (CID) W-1 and W-2 tests (Hirsh et al., 1952). Currently, ASHA (1988) recommends the use of 15 of the original 36 spondees used in the CID W-1 and W-2 tests for obtaining SRTs. These 15 words, shown in Table 5.1, are the most homogeneous with respect to audibility (Young et al., 1982), as is the list of 20 easily pictured spondees for use with children (Frank, 1980).

Recommended SRT Testing Protocol

The SRT measurement involves four steps: (1) instructions; (2) familiarization; (3) initial and test phase for the descending technique; and (4) calculation of threshold. Wilson et al. (1973) described these steps, which were subsequently set forth by ASHA (1988) as a guideline for determining an SRT.

TABLE 5.1

Spondaic Words Recommended by ASHA (1988)

15 Most Homogeneous Re: Audibility (Young et al., 1982)	20 Most Easy to Picture (Frank, 1980)
Toothbrush	Toothbrush
Inkwell	Hotdog
Playground	Baseball
Sidewalk	Airplane
Railroad	Cupcake
Woodwork	Popcorn
Baseball	Bathtub
Workshop	Fire truck
Doormat	Football
Grandson	Mailman
Eardrum	Snowman
Northwest	Ice cream
Mousetrap	Sailboat
Drawbridge	Flashlight
Padlock	Bluebird
	Toothpaste
	Reindeer
	Shoelace
	Seesaw

Step 1: Instructions

Patients need to be instructed regarding what stimuli will be used (i.e., spondaic words from the list) and how to respond during the testing procedure (i.e., written or verbal response). Also, it is important to make patients aware that the level of the stimulus will become quite soft and to encourage them to guess throughout the testing procedure.

Step 2: Familiarization

Each patient should be familiarized with the word list to be used during the testing procedure by listening to the list of test words at a level that is easily audible and repeating back each word as a demonstration of word recognition. If a patient is unable to repeat back a particular spondaic word from the test list, then that word should be removed from the test list. Another method of familiarization is to give the patient a written list of the test words to read.

Previous research has shown differences in SRT values obtained with and without familiarization (Conn et al., 1975; Tillman and Jerger, 1959). Specifically, Tillman and Jerger (1959) found poorer SRTs of almost 5 dB HL when individuals were not familiarized with the test list. The ASHA guideline strongly suggests that familiarization should not be eliminated from the test protocol.

Step 3: Determination of Threshold

a. Initial starting level—Present one spondaic word at a level 30 to 40 dB HL above the anticipated SRT. If a correct response is received, drop the level in 10-dB steps until an incorrect response occurs. Once an incorrect response is received, present a second spondaic word at the same level. If the second word is repeated correctly, drop down by 10-dB steps until two words are missed at the same level. Once you reach the level where two spondees are missed, increase the level by 10 dB. This is the starting level.
b. Threshold estimation—Thresholds have been estimated using 2- or 5-dB steps since most audiometers are equipped with those step sizes. Previous studies have shown that threshold differences as a function of step size are too small to be clinically significant (Wilson et al., 1973).

   2-dB step size—Present two spondaic words at the starting level. Drop the level by 2 dB and present two spondaic words. An individual should get the first five out of six words correct or else the starting level needs to be increased by 4 to 10 dB. If at least five of the first six words are correct, continue dropping the level by 2 dB until the individual misses five of six presentations.

   5-dB step size—Present five spondaic words at the starting level. An individual should get the first five spondaic words correct at the starting level. Drop the level by 5 dB and present five spondaic words. Continue dropping the level by 5 dB until the individual misses all five spondaic words at the same level.

Step 4: Calculation of Threshold

Calculation of an SRT is based on the Spearman–Kärber equation (Finney, 1952). An SRT is calculated by subtracting the number of words repeated correctly from the starting level and adding a correction factor of 1 dB when using the 2-dB step size and a correction factor of 2 dB when using the 5-dB step size. For a 5-dB step example, with a starting level of 40 dB, the patient got all five words; at 35 dB, three of the words were correct; and at 30 dB, none were correct. Eight of the 15 words were correct. Therefore, the SRT calculation would be $40 - 8 = 32, + 2$ for the correction, equals 34 dB HL.

Clinical Functions of SRT

The most recent surveys of audiometric practices in the United States reported that 99.5% (Martin et al., 1998) and 83% (ASHA, 2000) use SRT as part of their basic audiologic assessment. The reasons stated for using SRT were (1) cross validation for puretone thresholds; (2) measurement of communication disability; and (3) reference for suprathreshold measures. Unfortunately, most of the historical purposes lack scientific evidence to support routine clinical

use of an SRT (Wilson and Margolis, 1983). In addition, only 58% of audiologists complete the familiarization step of the test protocol, and 60% do not follow the recommended ASHA (1988) protocol but, instead, determine an SRT using a modified Hughson–Westlake procedure with two out of three criteria (Martin et al., 1998). These observations are of concern because the SRT is a valid and reliable procedure when standardized recorded materials are used with a specified testing procedure. The SRT is also particularly useful when assessing response reliability in an individual who appears to be malingering (see Chapter 33).

SPEECH RECOGNITION IN QUIET

The purpose of speech recognition testing in quiet is to assess how well a person can understand speech in a quiet environment when the level of the speech is loud enough to obtain a maximum SRS (PB_{max}). The level necessary for a person with hearing loss to perform maximally is highly variable from person to person and is dependent on the materials used to obtain the SRS (Jerger and Hayes, 1977). We feel that it is unfortunate that, in most audiology clinics, speech recognition testing is assessed only at one presentation level (Wiley et al., 1995). The majority of audiologists select a single presentation level 30 to 40 dB SL re: SRT, meaning that the materials are presented 30 to 40 dB above the SRT (Martin et al., 1998; Wiley et al., 1995). Kamm et al. (1983) found that speech recognition testing at 40 dB SL re: SRT did not approximate maximal performance for 40% of their 25 subjects with hearing loss. Evidence suggests that evaluating speech recognition abilities at more than one level captures a portion of the psychometric function and allows a better estimation of performance at PB_{max}. A procedure suggested by Wilson (2005, Personal communication) suggests the use of at least two levels with 25 words presented at each level. For persons with normal hearing or mild hearing loss as evidenced by a puretone average (PTA) of ≤35 dB HL for 500, 1,000, and 2,000 Hz, the first level should be 50 dB HL followed by the second level of 70 dB HL. For persons with greater hearing loss, the first level should be 10 dB greater than their PTA of 500, 1,000, and 2,000 Hz, and the second level should be 20 dB greater than the first level. If you are unable to raise the second level 20 dB greater than the first level because of loudness discomfort issues, raise the second level as high as possible over the first level.

Several types of materials are used to assess speech recognition ability in quiet such as sentences, nonsense syllables, and the most commonly used stimuli, monosyllabic words. Previous research has shown that nonsense syllables are the most difficult of the three materials mentioned above for individuals to recognize, whereas sentences are the easiest. Recognition performance of monosyllabic words falls on the performance continuum somewhere between nonsense syllables and sentences. Although monosyllables are the most commonly used stimuli in clinical settings for measuring speech recognition performance in quiet, it is important to note that empirical data (Bilger, 1984) support that speech recognition performance is a single construct and performance at one level of linguistic complexity (e.g., sentences) can be predicted by performance at another level (e.g., monosyllabic words).

The systematic relationship between recognition performances at various levels of linguistic complexity by adults with acquired hearing losses was demonstrated by Olsen et al. (1997). Performance for phonemes, words in isolation, and words in sentences was measured for 875 listeners with sensory/neural hearing loss. They found that the scores for words in isolation and in sentences were predictable from the phoneme recognition scores, with mean prediction errors of only 6% and 12%, respectively. Thus, for example, a person scoring 60% correct on a phoneme recognition task would be predicted to score 22% (±6%) for the recognition of words in isolation and 42% (±12%) for the recognition of words in sentences.

Monosyllabic Words

Historically, word lists such as the Northwestern University Auditory Test Number 6 (NU No. 6; Tillman and Carhart, 1966), the CID Auditory Test W-22 (CID W-22; Hirsh et al., 1952), and the Phonetically Balanced 50 (PB-50; Egan, 1948) have been used to assess word recognition performance in a quiet background during audiologic evaluations.

The initial work of Egan (1944) outlined six principal criteria that the Psychoacoustics Lab at Harvard used to develop the PB-50 word lists. The six criteria were (1) monosyllabic structure, (2) equal average difficulty of lists, (3) equal range of difficulty of lists, (4) equal phonetic composition of lists, (5) representative sample of American English, and (6) familiar words. According to Hood and Poole (1980), it was assumed by Egan that meeting criteria 1, 4, 5, and 6 would ensure criteria 2 and 3. Further work to revise the PB-50 word lists by Hirsh et al. (1952) and Tillman et al. (1963) utilized the six criteria to create the W-22 word lists and the NU No. 6 word lists, respectively.

PB-50

The initial use of monosyllabic words for speech recognition testing is attributed to Egan (1948) who worked in the PAL at Harvard University. His original pool of 1,000 words was divided into 20 lists of 50 words, which collectively are known as the PAL PB-50 word lists. Each list was considered to be phonetically balanced such that the 50 words that composed a list were a proportionally correct representation of the phonetic elements in English discourse.

CID W-22

Hirsh et al. (1952) had five judges rate the familiarity of the 1,000 monosyllabic words selected by Egan for the PB-50

word lists, and 120 of the PB-50s were selected along with 80 other words to compose the new word lists. These 200 very common words were selected and phonetically balanced into four 50-word lists known as the CID W-22 word lists. The CID W-22 word lists were recorded onto magnetic tape as spoken by Ira Hirsh who monitored his voice on a VU meter stating the carrier phrase "You will say" and letting each target word fall naturally at the end of the phrase. The CID W-22 word lists are some of the most popular word lists used by audiologists for measuring suprathreshold word recognition ability in quiet.

NU NO. 6

Lehiste and Peterson (1959) devised lists of CNCs (consonant–syllable nucleus [vowel]–consonant) that were phonemically balanced versus phonetically balanced. That is, lists that were developed to be phonetically balanced did not take into account the position of the sound in a word and how the acoustic realization of the sound would be affected by coarticulatory factors. Lehiste and Peterson argued that phonemic balancing could be accomplished by allowing for the frequency of occurrence of each initial consonant, vowel nucleus, and final consonant to be similar across CNC word lists. The Lehiste and Peterson lists were condensed into four lists of 50 words known today as the NU No. 6.

Historically, 50 words were included in each test list to facilitate phonetic balancing and to allow for a simple conversion from number correct to percent correct following testing. Studies have examined the benefits of abbreviating the number of words used per list from 50 to 25 with mixed results in terms of test–retest reliability (Beattie et al., 1978; Elpern, 1961). The most important work regarding this issue of half versus full lists was the examination of speech recognition data as a binomial variable by Thornton and Raffin (1978). As discussed in the earlier section on psychometric functions, performance ability between 20% and 80% is the most variable, whereas performance ability is least variable at either extreme of the function (Egan, 1948). The results of Thornton and Raffin (1978) support these early views on performance using the binomial distribution to mathematically model word recognition performance. It indicates that the accuracy between scores for the same listener depends on the number of words used per list and the listener's level of performance. In addition, Thornton and Raffin created a table of the lower and upper limits of the 95% critical differences for percentage scores as a function of test items. Table 5.2 shows the critical differences a retest score would need to exceed to be considered statistically different for the original test score. As seen in Table 5.2, as the number of items increases, the range decreases, suggesting that as the set size increases, the variability in the scores decreases, allowing for the detection of more subtle differences in performance. One way to increase set size without increasing test time is to move from whole-word scoring to phoneme scoring (Boothroyd, 1968). In a 25-word list of monosyllables, you have 25 items to score using whole-word scoring, whereas you would have 50 to 75 possible items to score using phoneme scoring.

TABLE 5.2

Critical Difference Ranges (95%) for Select Percent Correct Scores as a Function of Number of Test Items

% Correct	10 Words	25 Words	50 Words
0	0–20	0–8	0–4
10	0–50		2–24
20	0–60	4–44	8–36
30	10–70		14–48
40	10–80	16–64	22–58
50	10–90		32–68
60	20–90	36–84	42–78
70	30–90		52–86
80	40–100	56–96	64–92
90	50–100		76–98
100	80–100	92–100	96–100

From Thornton and Raffin (1978).

Sentence Tests

Sentence-level tests were developed at Bell Laboratories (Fletcher and Steinberg, 1929) and were used during World War II to evaluate military communication equipment (Hudgins et al., 1947). Until the development of the CID Everyday Sentences (Silverman and Hirsh, 1955), no sentence test had received clinical acceptance. The CID sentences consist of 10 lists of 10 sentences each with 50 key words in each list. Interrogative, imperative, and declarative sentences are included. Responses can be spoken or written and are scored as the percentage of key words correctly recognized.

The basis for the use of sentences in the clinical assessment of speech recognition abilities is that sentences provide a more "realistic" listening condition for everyday communication than does the use of isolated words or nonsense syllables (Bess, 1983; Silverman and Hirsh, 1955). Although sentences may have greater face validity than other stimuli, they also provide semantic, syntactic, and lexical clues (i.e., extrinsic redundancies). Thus it is difficult to distinguish individuals who do well on a speech recognition task because they have good speech recognition skills or because they make good use of top-down (linguistic, cognitive) processing skills. Another complication of the use of sentence materials is that, as length exceeds seven to nine syllables, memory constraints, particularly in the elderly, may impact performance (Miller, 1956). Despite these potential limitations, several clinically useful sentence tests have been developed. Because the ability to use context is preserved even in older adults with hearing loss, for most patient populations,

sentence tests are typically too easy (ceiling effect) and, therefore, fail to distinguish among levels of difficulty. However, they are well suited as adaptive noise procedures (see "Speech Recognition in Noise" section) instead of suprathreshold quiet procedures. An exception to this trend is the use of sentence tests in quiet for individuals with severe-to-profound hearing losses.

For the profoundly impaired patient population, the City University of New York (CUNY) Sentences (Boothroyd et al., 1988), which consist of 72 sets of topic-related sentences, were designed to assess the use of cochlear implants and tactile aids as supplements to speech reading. Each sentence in a set is about one of 12 topics: food, family, work, clothes, animals, homes, sports/hobbies, weather, health, seasons/holidays, money, or music. Each set contains four statements, four questions, and four commands and one sentence of each length from 3 to 12 words, for a total of 102 words per set. Performance is scored as the number of words correct. Original recordings were on laser-video disc and were presented via the Computer Assisted Speech Perception Software (CASPER; Boothroyd, 1987) program. The CUNY Sentences are being converted to DVD format with upgrades to the CASPER software as part of current work at the Rehabilitation Engineering Research Center (RERC) on Hearing Enhancement at Gallaudet University (http://www.hearingresearch.org/).

Nonsense Syllable Tests/Phoneme Recognition Tests

The effects of lexical context and word familiarity on test performance can be minimized by the use of nonsense syllable and/or closed-set phoneme recognition tests. Nonsense syllables were one of the first materials used to assess speech recognition ability during the development of telephone circuits at Bell Telephone Laboratories (Fletcher and Steinberg, 1929). However, clinical use of nonsense syllables for those with hearing loss did not occur until the 1970s when two carefully developed tests became available—the CUNY Nonsense Syllable Test (CUNY-NST; Levitt and Resnick, 1978) and the Nonsense Syllable Test (NST; Edgerton and Danhauer, 1979). The CUNY-NST is a closed-set test consisting of seven subtests, each of which has seven to nine consonant–vowel (CV) or vowel–consonant (VC) syllables. The CUNY-NST assesses perception of the consonants most likely to be confused by individuals with hearing loss using three vowel contexts. The Edgerton–Danhauer NST is an open-set test consisting of 25 nonsense bisyllabic CVCV items, allowing for assessment of the perception of 50 consonant and 50 vowel stimuli. More recently, Boothroyd et al. (1988) described the Three Interval Forced Choice Test of speech pattern contrast perception (THRIFT), an NST that can be used with children 7 years of age or older (Hnath-Chisolm et al., 1998). The THRIFT measures the perception of nine speech pattern contrasts presented in varying phonetic context. Contrasts include intonation; vowel height and place; and initial and final consonant voicing, continuance, and place. In addition to minimizing the effects of lexical context and word familiarity on performance, the use of nonsense syllables allows for detailed examination of phonetic errors. Despite these advantages, nonsense syllables lack face validity with regard to being representative of everyday speech communication.

Minimization of lexical context and word familiarity effects, while allowing for the analysis of errors and confusions, can also be accomplished through the use of closed-set tests using real word stimuli. Classic tests of phoneme recognition include the Modified Rhyme Test (MRT; House et al., 1955; Kruel et al., 1968) and its variations (e.g., Rhyming Minimal Contrasts Test (Griffiths, 1967) and the California Consonant Test (CCT; Owens and Schubert, 1977). The MRT consists of 50 test items each with six response alternatives. Twenty-five of the items differ by the initial consonant (i.e., bent, went, sent, tent, dent, and rent), and the other 25 items differ by the final consonant (i.e., peas, peak, peal, peace, peach, and peat). The CCT also consists of 100 items but uses a four-choice, rather than a six-choice, response format in assessing the perception of 36 initial consonant items and 64 final consonant items. The perception of medial vowels as well as initial and final consonants was added in the University of Oklahoma Closed Response Speech Test by Pederson and Studebaker (1972).

A closed-set format is also used in the Speech Pattern Contrast (SPAC) test (Boothroyd, 1984), which was designed to assess the ability to perceive both suprasegmental (i.e., stress and intonation) and segmental phonologically (i.e., vowel height and place, initial and final consonant voicing, continuance, and place) relevant distinctions. Test length of SPAC is minimized by combining two segmental contrasts in one subset (e.g., final consonant voicing and continuance) with four items (e.g., seat-seed-cease-sees). Although the SPAC as well as other speech feature tests and NSTs are not routinely used in clinical audiology, the information provided about the details of an individual's speech perception ability can be quite useful when assessing the need for and the benefits of hearing aids and cochlear implants for both children and adults.

SPEECH RECOGNITION IN NOISE

The most common complaint expressed by adults with hearing loss is the inability to understand a speaker when listening in an environment of background noise. In 1970, Carhart and Tillman suggested that an audiologic evaluation should include some measure of the ability of an individual to understand speech when in a background of speech noise. Prior to the revival of the directional microphone in the late 1990s, however, the information gained from a speech-in-noise task for most rehabilitative audiologists was not pertinent to the selection of amplification because of the fact that

most hearing aids were mainly selected based on gain, slope, and output curves. Thus in the technology-driven field of audiology, speech-in-noise testing failed to gain a place in the traditional audiologic evaluation. The revolution of digital hearing aids and their multitude of features, such as directional microphones, noise reduction strategies, and digital signal processing strategies, have created an important reason for utilizing speech-in-noise tasks on a routine basis when evaluating an individual with hearing loss.

For the past 40 years, researchers have observed that listeners with hearing loss show a greater disadvantage when listening in a competing speech background compared with listeners with normal hearing, such that the S/N needed for the listener with hearing loss is 10 to 15 dB greater than that needed by listeners with normal hearing (e.g., Carhart and Tillman, 1970). Plomp (1978) reported that for every 1-dB increase in signal over the competing noise, a listener with hearing loss would receive, on average, an improvement of 3% in terms of ability to recognize the signal. Thus, a 10-dB improvement in S/N should add 30% in terms of intelligibility as measured by open-set, speech recognition tests for listeners with hearing loss.

The addition of background noise to a speech recognition task has been shown to improve the sensitivity and validity of the measurement (Beattie, 1989; Sperry et al., 1997). In terms of improving sensitivity, the addition of multiple S/Ns increases the difficulty of the task and allows for separation between individuals with normal hearing and those with hearing loss (Beattie, 1989; McArdle et al., 2005b). Typically, individuals with sensory/neural hearing loss require the signal to be 10 to 12 dB higher than the noise to obtain 50% performance on the psychometric function, whereas individuals with normal hearing on average obtain 50% performance at an S/N of 2 to 6 dB. McArdle et al. (2005a, 2005b) found mean performance on the Words-in-Noise (WIN) test (Wilson, 2003) to be 12.5 and 6 dB S/N for 383 listeners with hearing loss and 24 listeners with normal hearing, respectively. Similarly, under similar experimental conditions, Dirks et al. (1982) and Beattie (1989) who used CID W-22 word lists in noise found 50% points of 12 and 11.3 dB S/N, respectively, for listeners with hearing loss.

Several studies have examined the possibility of predicting the ability of an individual to understand speech-in-noise using puretone audiograms and SRSs in quiet without success (Beattie, 1989; Carhart and Tillman, 1970; Cherry, 1953; Dirks et al., 1982; Killion and Niquette, 2000; Plomp, 1978; Wilson, 2003). The data in Figure 5.3 were compiled from two studies (McArdle et al., 2005a, 2005b). In the figure, performance on a word recognition in quiet task at 80 dB HL is graphed on the ordinate as a function of 50% points on the WIN test along the abscissa. The same words spoken by the same speaker were used for both the recognition task in quiet and in noise. The shaded area of the figure represents

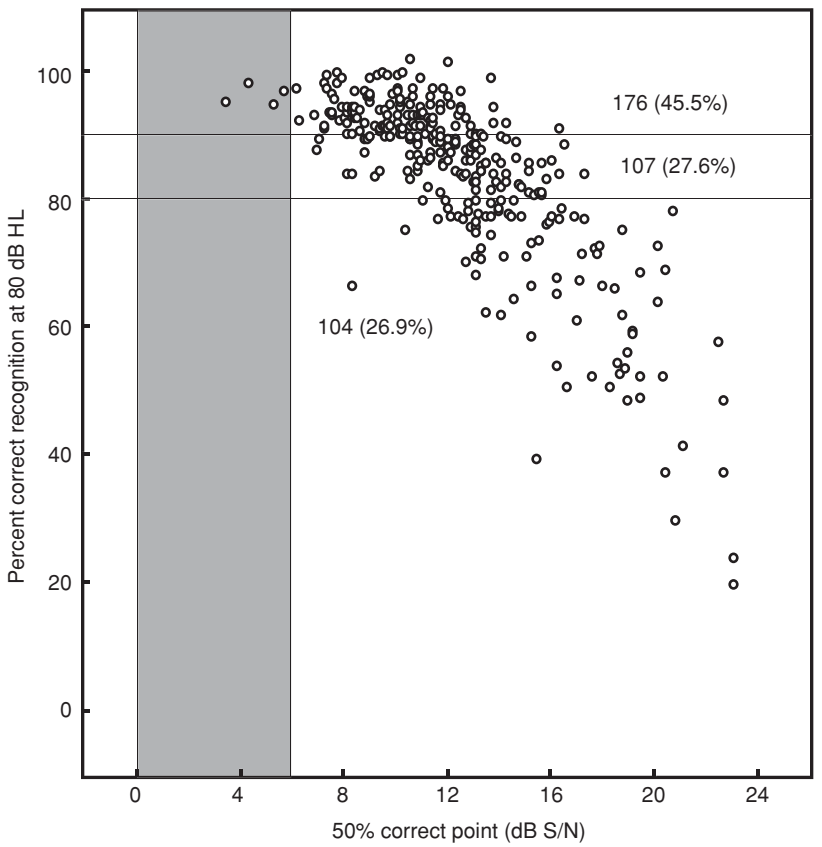


FIGURE 5.3 A plot of word recognition performance in quiet in percent correct (*y*-axis) versus the 50% point of recognition performance in multitalker babble on the Words-in-Noise (WIN) test (*x*-axis). The *shaded area* of the figure defines the range of performances (10th to 90th percentiles) obtained by listeners with normal hearing on the WIN test. The numbers represent the number of listeners who had word recognition scores in quiet ≥90%, ≥80%, and ≥70% correct on the words in quiet. The data are combined from McArdle et al. (2005a, 2005b). (Reprinted with permission from the *Journal of Rehabilitative Research and Development.*)

the range of performance by 24 listeners with normal hearing on the WIN.

Two main observations can be seen in the data in Figure 5.3: (1) only 5 out of 387 listeners with hearing loss performed in the normal range on both the recognition task in quiet and in noise; and (2) 45.5% of the 387 listeners with hearing loss had word recognition scores in quiet at 80 dB HL that were ≥90% correct. Thus, it is of interest to note that although 73% of the listeners with hearing loss had word recognition scores in quiet ≥80%, the overwhelming majority of these listeners displayed abnormal performance on a word recognition task in noise. This finding suggests that speech-in-noise testing may be considered a stress test of auditory function (Wilson, 2013, Personal communication). In addition, it is clear that word recognition ability in noise is not easily predicted by word recognition in quiet for listeners with hearing loss other than to say that listeners with poor recognition ability in quiet also perform poorly on word recognition tasks in noise. Because we are unable to predict the ability of an individual to understand speech in a noisy background, audiologists should use the tests available for quantifying the S/N needed by the listener to understand speech in noise. Several materials, described in the following section, have been developed to measure speech-in-noise performance.

Materials

Initially, efforts in speech-in-noise testing were focused on sentence-level materials to make the task more of a real-world experience; however, normal everyday sentences were too easy, and further manipulation of the sentences was needed to obtain the 50% correct point of performance for a listener on a speech-in-noise task. Speaks and Jerger (1965) developed the Synthetic Sentence Identification (SSI) test to minimize the effect of contextual cues that often made it easy to understand sentence-level materials even in a background of noise. The stimuli are called synthetic sentences because they are not actual sentences, but rather they contain normal English phonemes and syntax but no semantic context. An example of a sentence is "Small boat with a picture has become." The task of the listener is to select which one of 10 sentences displayed on a response form is perceived when presented against a competing story describing the life of Davy Crockett. The competing story can be presented either contralaterally or ipsilaterally.

Another interesting sentence-level test, the Speech Perception in Noise (SPIN) test (Kalikow et al., 1977), varies the amount of semantic context that leads to the last word of each sentence, which is a monosyllabic target word. The SPIN test has eight forms of 50 sentences each that are presented at a fixed S/N of 8 dB. The target word in 25 of the sentences has low predictability (LP) given the limited clues from the preceding context, and the other 25 have high predictability (HP) from the preceding sentence context. Recognition performance is scored as the percentage of LP and HP words correctly perceived. By providing both LP and HP scores, the SPIN test not only allows for the assessment of the acoustic-phonetic components of speech, but also examines the ability of an individual to utilize linguistic context.

In the 1980s, two additional tests designed to assess recognition of everyday speech based on correct word recognition performance in sentence length stimuli were developed. The Connected Speech Test (CST; Cox et al., 1987), which was developed as a criterion measure in studies of hearing aid benefit, consists of 48 passages of conversationally produced connected speech. Each passage is about a familiar topic and contains 10 sentences. Sentence length varies from 7 to 10 words, and there is a total of 25 key words in each passage. Sentences are presented at an individually determined S/N, and performance is scored as the number of key words correct.

The most recent application of sentence length stimuli is in tests that are scored in terms of the decibel-to-noise ratio required to achieve 50% correct performance. The two most common tests are the Hearing in Noise Test (HINT; Nilsson et al., 1994) and the Quick Speech-in-Noise (QuickSIN) test (Killion et al., 2004). The two tests vary in the type of sentences and type of noise used. The HINT uses the Bamford–Kowal–Bench (BKB) Standard Sentence Lists (Bench et al., 1979) that were compiled from the utterances of hearing-impaired children and contain straightforward vocabulary and syntax. Sentences are presented in sets of 10 sentences, and the listener must repeat the entire sentence correctly to receive credit. The noise used is speech-spectrum noise that is held constant while the signal is varied to find the 50% correct point. The QuickSIN uses the Harvard Institute of Electrical and Electronics Engineers (IEEE, 1969) sentences, which are a collection of low-context, meaningful sentences, whose phonetic balance is similar to that of English. In the QuickSIN, there are six sentences per list, and each sentence contains five key words. All sentences are presented in multitalker babble with the five key words in each sentence scored as correct or incorrect. Recently, the BKB-SIN test (Etymotic Research, 2005) was developed for use with children (ages ≥5), cochlear implant patients, and adults for whom the QuickSIN test is too difficult.

More recently, monosyllabic and digit materials in multitalker babble have been developed at the Auditory Research Lab of the James H. Quillen Veterans Affairs Medical Center (Wilson, 2003; Wilson and Strouse, 2002; Wilson and Weakley, 2004). The word and digit materials have been shown to be sensitive to the different recognition abilities of normal-hearing and hearing-impaired adults in multitalker babble (Wilson et al., 2003; Wilson and Weakley, 2004). McArdle et al. (2005b) examined the effect of material type (i.e., digits, words, and sentences) on S/N loss for young listeners with normal hearing and older listeners with hearing impairment. The three speech-in-noise tests that were examined

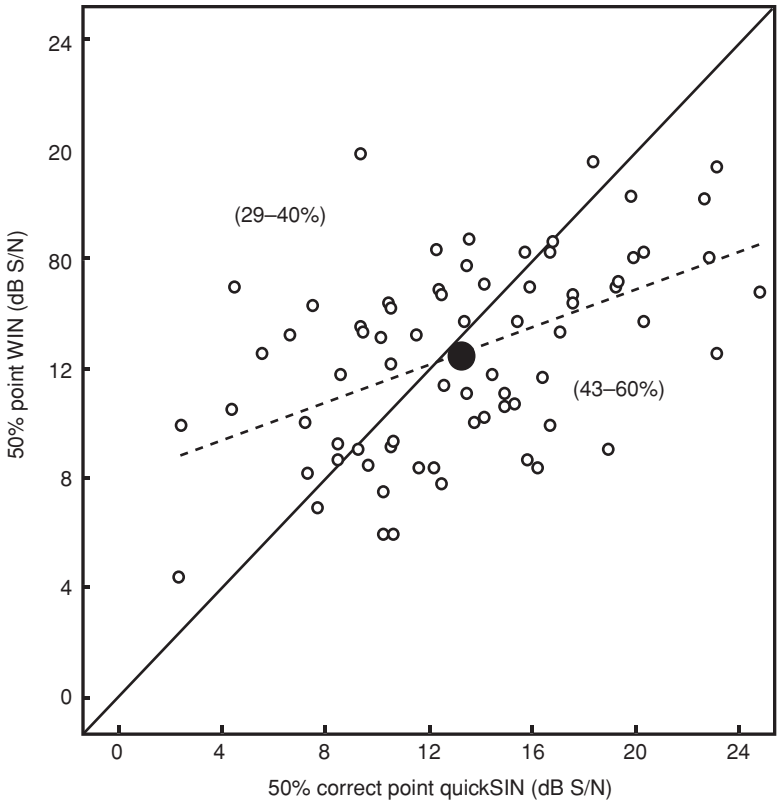


FIGURE 5.4 Bivariate plot of the 50% points (in dB S/N) on the Words-in-Noise (WIN) test (ordinate) and on the Quick Speech-in-Noise (QuickSIN) test (abscissa). The *diagonal line* represents equal performance, with the *larger filled symbol* indicating the mean datum point. The *dashed line* is the linear regression fit to the data. The *numbers in parentheses* are the number of performances above and below the line of equal performances. (Reprinted with permission from the *Journal of Rehabilitative Research and Development*.)

include: (1) QuickSIN (Etymotic Research, 2001); (2) WIN test (Wilson and Strouse, 2002; Wilson, 2003); and (3) digit triplets-in-multitalker babble (Wilson and Weakley, 2004). As expected, the younger listeners performed better than the older listeners on all three tasks. For the older listeners with hearing loss, the S/N required for 50% recognition of each material type presented was −4, 12.4, and 11.7 dB S/N for digits, words, and sentences, respectively. Figure 5.4 shows a bivariate plot of the 50% points for the older listeners with hearing loss on both the QuickSIN (abscissa) and the WIN (ordinate). The diagonal line in Figure 5.4 represents equal performance on both QuickSIN and the WIN. As can be seen, mean performance, as indicated by the bold filled circle, is close to the diagonal line, suggesting that either the use of monosyllabic words or IEEE sentences in this population provided a similar measure of performance in noise. More importantly, the performance difference at the 50% point between normal-hearing listeners and hearing-impaired listeners was 7.6 dB for both words and sentences, suggesting that words and sentences in a descending speech-in-noise task were equally sensitive to the effects of hearing loss. For a more in-depth discussion of the use of words or sentences in speech-in-noise testing, see Wilson and McArdle (2005).

A new body of literature has evolved in the area of speech-in-noise testing focused on informational masking, which is defined as nonenergetic masking that increases threshold as a result of uncertainty (Wilson et al., 2012). Although the term informational masking is more recent, the construct was described by Carhart et al. (1969) and termed perceptual masking. Energetic masking is described in the literature as peripheral masking, such that a stimulus interferes with the perception of a second stimulus making the first stimulus a "masker." Nonenergetic masking, or informational masking, occurs when the target stimulus is similar to the masking stimulus, creating uncertainty for the listener as to whether he or she is hearing the target or the masker. Informational masking can occur at different processing levels (e.g., phonetic, semantic) and is greater for a speech masker than noise, especially when the talker is the same gender or, even worse, the same talker for both the target and the masker (Brungart, 2001). Informational masking has a greater effect when the masker is a single speaker versus a background of multiple talkers since once you add more than a couple of speakers the background "information" in the masker becomes hard to distinguish. Most commercially available speech-in-noise tests involve multitalker babble, which decreases the effects of informational masking but future studies in this area are warranted.

CONSIDERATIONS FOR SPEECH AUDIOMETRY IN CHILDREN AND OTHER SPECIAL POPULATIONS

Speech stimuli are used for the behavioral assessment of the auditory function of a child from birth onward. With very young infants, speech stimuli might be used to elicit a startle

response, and as the infant develops, SDTs and SRTs can be obtained using a variety of behavioral techniques, such as visual response audiometry or play audiometry. The technique used will be dependent on the motor capabilities of the child. In addition to considering the motor capacity of the child for responding (e.g., head turn, picture pointing), the phonologic, receptive, and expressive language skills of the child need to be considered during speech recognition testing. For example, by the time a child can function at about a 5-year-old level, conventional SRTs can be obtained as long as the spondee words used are within the receptive vocabulary of the child (ASHA, 1988). Similarly, several suprathreshold pediatric speech recognition tests, such as the Word Intelligibility Picture Identification (WIPI) test (Ross and Lerman, 1970) and the Northwestern University Children's Perception of Speech (NU-CHIPS) test (Elliot and Katz, 1980), are comprised of words expected to be within the receptive vocabulary of a child.

A variety of speech recognition tests are available for use with children. For example, both the WIPI and NU-CHIPS use monosyllabic words presented in a closed-set format. Other test paradigms allow for the assessment of the perception of speech feature contrasts (e.g., Imitative Test of Speech Pattern Contrast Perception [IMSPAC]; Kosky and Boothroyd, 2003; Visually Reinforced Audiometry Speech Pattern Contrast Perception [VRASPAC]; Eisenberg et al., 2004), syllabic pattern and stress (e.g., Early Speech Perception [ESP] test; Moog and Geers, 1990), lexically easy versus lexically hard words (e.g., the Lexical Neighborhood Test [LNT]; Kirk et al., 1995), and words in sentences presented in quiet (e.g., BKB sentences; Bamford and Wilson, 1979) and in noise (e.g., BKB-SIN test; Etymotic Research, 2005), a task which requires word and sentence recognition in both quiet and noise (e.g., Pediatric Speech Intelligibility [PSI] test; Jerger and Jerger, 1984).

In addition to children, special consideration also needs to be given to the assessment of speech perception abilities in profoundly hearing-impaired adults, nonverbal patients, and multilingual patients (Wilson and Strouse, 1999). Profoundly hearing-impaired adults typically obtain scores of zero on standard speech recognition tests. As a result, batteries such as the Minimal Auditory Capabilities (MAC) battery have been developed (Owens et al., 1985). Tasks included in the MAC battery involve discrimination of syllabic number, noise versus voice, and statements versus questions; recognition of spondaic words and consonants and vowels in real words in closed-set tasks; and more standard open-set recognition of words in isolation and sentences.

Nonverbal patients are often encountered in medical settings where patients may have medical conditions such as laryngectomies or cerebral vascular accidents. For these patients, written responses or picture pointing tasks may be appropriate. Increases in the ethnic diversity of the US population can result in the audiologist assessing a patient who speaks little to no English. Limited knowledge of English could impact on speech perception performance in the same way that the developing linguistic abilities of a child are important to consider in assessment. Although recorded materials are available in languages such as Spanish (Wesilender and Hodgson, 1989), unless the audiologist speaks Spanish, errors could be made in determining correct from incorrect responses. Wilson and Strouse (1999) suggest the use of a multimedia approach similar to that used by McCullough et al. (1994) with nonverbal patients. Stimulus words are presented in the patient's native language, and the person responds by selecting the perceived word from a closed set of alternatives shown on a touchscreen monitor. Scoring could be done automatically through a software program.

CLINICAL FUNCTIONS OF SPEECH RECOGNITION MEASURES

One of the historical purposes for the use of speech recognition testing in the clinical test battery was as a diagnostic tool for determining the location of peripheral auditory pathology. Figure 5.2 illustrates typical psychometric functions obtained in quiet for the recognition of monosyllabic words by listeners with normal auditory function as well as those with conductive, sensory (cochlear), and neural (retrocochlear) hearing losses. For normal-hearing listeners, regardless of word recognition materials used, when the presentation level is about 30 dB higher than the dB level needed for 50% performance (i.e., SRT), a score of 90% or better can be expected. For individuals with hearing loss, when listening at a moderate level, scores may range anywhere from 100% correct to 0% correct. Because of this wide dispersion of speech recognition performance across individuals with various types of hearing loss, speech recognition testing provides only limited diagnostic information if testing is done at only one intensity level (see, for discussion, Bess, 1983; Penrod, 1994). When testing is completed at several intensity levels, however, certain patterns of performance can be expected with certain hearing losses (Wilson and Strouse, 1999).

Individuals with conductive hearing loss tend to exhibit little difficulty on speech recognition tests, with performance typically at 90% or better when testing is conducted at moderate SLs (curve #1 of Figure 5.2). A patient with a sensory/neural hearing loss will generally have poorer SRSs than would a person with the same degree of hearing loss due to conductive pathology. Although a very wide range of scores are found across patients with cochlear as well as retrocochlear hearing losses, SRSs tend to be poorest among those with retrocochlear pathology. Although some individuals with cochlear losses will demonstrate a slight decrease in recognition performance when intensity levels are increased beyond the initial level needed for obtaining maximum performance (curve #3 of Figure 5.2), marked decreases in performance with increasing intensity after maximum performance is achieved are typically characteristic of a neural

loss (curves #4 and #5 of Figure 5.2). The phenomenon of reduced SRSs with increasing intensity that occurs with retrocochlear pathology is referred to as the "rollover" effect (Bess et al., 1979; Dirks et al., 1977). In addition to rollover, retrocochlear pathology would be suspected in the presence of a significant discrepancy in SRSs between two ears or lower than expected performance at all presentation levels (curve #6 of Figure 5.2).

Assessment of the central auditory system also uses measures of speech recognition performance. Tasks can be presented either monaurally or binaurally. Monaural tasks use distorted, degraded, or low-redundancy speech stimuli to reduce extrinsic redundancies. Methods of degradation include filtering (Bocca et al., 1955), time compression (Wilson et al., 1994), and reverberation (Nabelek and Robinson, 1982). Binaural tests were designed to assess the ability of the central auditory nervous system to integrate or resynthesize the different parts of a signal that are presented to each of the two ears. For example, in the Binaural Fusion test (Matzker, 1959), a low-pass filtered version of a word is presented to one ear, whereas a high-pass filtered version of the same word is presented to the opposite ear. A normal-functioning central auditory nervous system is able to integrate the information from each ear and respond with the correct target word. On the other hand, binaural dichotic tasks involve the presentation of different speech signals simultaneously to both ears. The patient must repeat either or both of the signals depending on the test used. Common clinical dichotic tests include Dichotic Digits (Kimura, 1961), the Staggered Spondaic Word test (Katz, 1962), and the Dichotic Sentence Identification test (Fifer et al., 1983). The interpretation of performance on tests designed to assess auditory processing abilities is beyond the scope of the present chapter and is discussed in detail in Chapters 27 and 29.

In addition to diagnostic applications, speech recognition testing has an important role in estimating the adequacy and effectiveness of communication and in the planning and evaluation of (re)habilitative efforts, including the selection and fitting of hearing aids and cochlear implants. For example, many audiologists label speech recognition performance for monosyllabic words presented in quiet performance as "excellent," "good," "fair," or "poor" in an attempt to link performance to the adequacy of communication in everyday settings. However, research designed to demonstrate systematic relationships between recognition performance in quiet and actual everyday communication has been largely unsuccessful (Davis, 1948; High et al., 1964). A better estimate of the impact of a hearing loss on daily communication might be obtained with the use of speech-in-noise tests such as the WIN, QuickSIN, or HINT. As Wilson and McArdle (2005) discuss, speech-in-noise testing allows for the assessment of the most common complaint of patients—the inability to understand speech in background noise; and thus, test results provide important information for use in counseling. Furthermore, test results can provide insight into the use of appropriate amplification and/or cochlear implant speech processing strategies.

In addition to testing in noise, Brandy (2002) points out that audiologists can gain insight into the (re)habilitative needs of patients through recording incorrect word responses, with subsequent examination of speech feature error patterns (e.g., fricatives, stops, glides). Other rehabilitative applications of speech audiometry include the use of materials that allow for the assessment of use of linguistic context (Flynn and Dowell, 1999) and auditory–visual performance (Boothroyd, 1987) and for the determination of most comfortable and uncomfortable listening levels (Punch et al., 2004). Information obtained with a variety of materials presented in a variety of paradigms can be useful in determining optimal device settings, starting points for therapeutic intervention, and directions for patient counseling.

FOOD FOR THOUGHT

1. Given the use of SRTs as a verification for puretone thresholds in every patient has been questioned since 1983, what is the cost benefit of measuring SRTs in a busy practice? Might the time be better spent gathering other information about the patient's auditory functioning?
2. Determining the presentation level for word recognition testing in quiet historically has been 40 dB SL, re: SRT. Given the evidence for this level is based on listeners with normal hearing, what is most appropriate for determining the presentation level(s) for listeners with hearing loss? Additionally in an audiologic evaluation, what are the benefits of using 25 words at each of two or three presentation levels versus 50 words at one presentation level?
3. Speech recognition in quiet has been performed by audiologists since the 1950s. Given that the most common complaint of listeners with hearing loss is their difficulty communicating in noisy situations, should the standard comprehensive audiometric battery be modified to include speech-in-noise measures?

KEY REFERENCES

thePoint A full list of references for this chapter can be found at http://thePoint.lww.com. Below are the key references for this chapter.

American National Standards Institute (ANSI). (1996) *Specifications for Audiometers. S3.6–1996.* New York,NY: American National Standards Institute.

American National Standards Institute. (2004) *Specifications for Audiometers. S3.6–2004.* New York, NY: American National Standards Institute.

American Speech-Language-Hearing Association. (1988) Guidelines for determining threshold level for speech. *ASHA.* 30, 85–89.

American Speech-Language-Hearing Association. (2000) *Audiology Survey.* Rockville, MD: American Speech-Language-Hearing Association.

Asher WJ. (1958) Intelligibility tests: A review of their standardization, some experiments, and a new test. *Speech Monogr.* 25, 14–28.

Bamford J, Wilson I. (1979) Methodological considerations and practical aspects of the BKB sentence lists. In: Bench J, Bamford JM, eds. *Speech-Hearing Tests and the Spoken Language of Hearing Impaired Children.* London: Academic Press; pp 148–187.

Beattie RC. (1989) Word recognition functions for the CID W-22 Test in multitalker noise for normally hearing and hearing-impaired subjects. *J Speech Hear Disord.* 54, 20–32.

Beattie RC, Svihovec DA, Edgerton BJ. (1978) Comparison of speech detection and spondee thresholds and half- versus full-list intelligibility scores with MLV and taped presentation of NU-6. *J Am Audiol Soc.* 3, 267–272.

Bench J, Kowal A, Bamford J. (1979) The BKB (Bamford-Kowal-Bench) sentence lists for partially-hearing children. *Br J Audiol.* 13, 108–112.

Bess FH. (1983) Clinical assessment of speech recognition. In: Konkle DF, Rintelmann WF, eds. *Principles of Speech Audiometry.* Baltimore, MD: University Park Press; pp 127–201.

Bess FH, Josey AF, Humes, LE. (1979). Performance intensity functions in cochlear and eighth nerve disorders. *Am J Otolaryngol.* 1, 27–31.

Bilger RC. (1984) Speech recognition test development. In: Elkins E, ed. *Speech Recognition by the Hearing Impaired. ASHA Reports 14.* Rockville, MD: ASHA; pp 2–7.

Bocca E, Calaero C, Cassinari V, Migilavacca F. (1955) Testing "cortical" hearing in temporal lobe tumors. *Acta Otolaryngol.* 45, 289–304.

Boothroyd A. (1968) Developments in speech audiometry. *Sound.* 2, 3–10.

Boothroyd A. (1984) Auditory perception of speech contrasts by subjects with sensorineural hearing loss. *J Speech Hear Res.* 27, 134–144.

Boothroyd A. (1987) CASPER: Computer Assisted Speech Perception Evaluation and Training. *Proceedings of the 10th Annual Conference on Rehabilitation Technology.* Washington, DC: Association for the Advancement of Rehabilitation Technology.

Boothroyd A, Hnath-Chisolm T, Hanin L, Kishon-Rabin L. (1988) Voice fundamental frequency as an aid to the speechreading of sentences. *Ear Hear.* 9, 335–341.

Brandy WT. (2002) Speech audiometry. In: Katz J, ed. *Handbook of Clinical Audiology.* 4th ed. Baltimore, MD: Lippincott Williams & Wilkins; pp 96–110.

Brungart DS. (2001) Informational and energetic masking effects in the perception of two simultaneous talkers. *J Acoust Soc Am.* 109, 1101–1109.

Carhart R. (1951) Basic principles of speech audiometry. *Acta Otolaryngol.* 40, 62–71.

Carhart R, Tillman TW. (1970) Interaction of competing speech signals with hearing losses. *Arch Otolaryngol.* 91, 273–279.

Carhart R, Tillman TW, Greetis ES. (1969) Perceptual masking in multiple sound backgrounds. *J Acoust Soc Am.* 45, 694–703.

Cherry EC. (1953) Some experiments on the recognition of speech with one and with two ears. *J Acoust Soc Am.* 25, 975–979.

Conn MJ, Dancer J, Ventry IM. (1975) A spondee list for determining speech reception threshold without prior familiarization. *J Speech Hear Disord.* 40, 388–396.

Cox RM, Alexander GC, Gilmore C. (1987) Development of the Connected Speech Test (CST). *Ear Hear.* 8, 119S–126S.

Davis H. (1948) The articulation area and the social adequacy index for hearing. *Laryngoscope.* 58, 761–778.

Department of Veterans Affairs. (1997) *The Audiology Primer for Students and Health Care Professionals.* Mountain Home, TN: Veterans Affairs Medical Center.

Dirks D, Kamm D, Bower D, Betsworth A. (1977) Use of performance intensity function in diagnosis. *J Speech Hear Disord.* 27, 311–322.

Dirks DD, Morgan DE, Dubno JR. (1982) A procedure for quantifying the effects of noise on speech recognition. *J Speech Hear Disord.* 47, 114–123.

Edgerton BJ, Danhauer JL. (1979) *Clinical Implications of Speech Discrimination Testing Using Nonsense Stimuli.* Baltimore, MD: University Park Press.

Egan JP. (1944) *Articulation Testing Methods, II. OSRD Report No. 3802.* Cambridge, MA: Psychoacoustic Laboratory, Harvard University.

Egan JP. (1948) Articulation testing methods. *Laryngoscope.* 58, 955–991.

Eisenberg LS, Martinez AS, Boothroyd A. (2004) Perception of phonetic contrasts in infants. In: Miyamoto RT, ed. *Cochlear Implants: International Congress Series 1273.* Amsterdam: Elsevier; pp 364–367.

Elliot L, Katz D. (1980) *Northwestern University Children's Perception Speech (NU-CHIPS).* St. Louis, MO: Auditec.

Elpern BS. (1961) The relative stability of half-list and full-list discrimination tests. *Laryngoscope.* 71, 30–36.

Etymotic Research. (2001) *QuickSINTM (Compact Disc).* Elk Grove Village, IL: Etymotic Research.

Etymotic Research. (2005) *BKB-SINTM (Compact Disc).* Elk Grove Village, IL: Etymotic Research.

Feldmann H. (1970) A history of audiology: A comprehensive report and bibliography from the earliest beginnings to the present. *Transl Beltone Inst Hear Res.* 22, 1–111. [Translated by J. Tonndorf from Die geschichtliche entwicklung der horprufungsmethoden, kuze darstellung and bibliographie von der anfongen bis zur gegenwart. In: Leicher L, Mittermaiser R, Theissing G, eds. *Zwanglose Abhandungen aus dem Gebeit der Hals-Nasen-Ohren-Heilkunde.* Stuttgart, Germany: Georg Thieme Verlag; 1960.]

Fifer RC, Jerger JF, Berlin CI, Tobey EA, Campbell JC. (1983) Development of a dichotic sentence identification test for hearing-impaired adults. *Ear Hear.* 4, 300–305.

Finney DJ. (1952) *Statistical Method in Biological Assay.* London: C. Griffen.

Fletcher H, Steinberg J. (1929) Articulation testing methods. *Bell Syst Techn J.* 8, 806–854.

Flynn MC, Dowell RC. (1999) Speech perception in a communicative context: An investigation using question/answer pairs. *J Speech Lang Hear Res.* 42, 540–552.

Frank T. (1980) Clinically significance of the relative intelligibility of pictorially represented spondee words. *Ear Hear.* 1, 46–49.

Griffiths JD. (1967) Rhyming minimal contrasts: A simplified diagnostic articulation test. *J Acoust Soc Am.* 42, 236–241.

High WS, Fairbanks G, Glorig A. (1964) Scale for self-assessment of hearing handicap. *J Speech Hear Disord.* 29, 215–230.

Hirsh IJ, Davis H, Silverman SR, Reynolds EG, Eldert E, Benson RW. (1952) Development of materials for speech audiometry. *J Speech Hear Disord.* 17, 321–337.

Hirsh IJ, Palva T, Goodman A. (1954) Difference limen and recruitment. *AMA Arch Otolaryngol.* 60, 525–540.

Hnath-Chisolm T, Laipply E, Boothroyd A. (1998) Age-related changes on speech perception capacity. *J Speech Hear Res.* 41, 94–106.

Hood JD, Poole JP. (1980) Influence of the speaker and other factors affecting speech intelligibility. *Audiology.* 19, 434–455.

House AS, Williams CE, Hecker MHL, Kryter KD. (1955) Articulation-testing methods: Consonantal differentiation with a closed-response set. *J Acoust Soc Am.* 37, 158–166.

Hudgins CV, Hawkins JE Jr, Karlin JE, Stevens SS. (1947) The development of recorded auditory tests for measuring hearing loss for speech. *Laryngoscope.* 57, 57–89.

Institute of Electrical and Electronics Engineers. (1969) IEEE recommended practice for speech quality measurements. *IEEE Trans Audio Electroacoust.* 17, 227–246.

Jerger J, Hayes D. (1977) Diagnostic speech audiometry. *Arch Otolaryngol.* 103, 216–222.

Jerger S, Jerger J. (1984) *Pediatric Speech Intelligibility Test.* St. Louis, MO: Auditec.

Kalikow DN, Stevens KN, Elliot LL. (1977) Development of a test of speech intelligibility in noise using sentence materials with controlled word predictability. *J Acoust Soc Am.* 61, 1337–1351.

Kamm CA, Morgan DE, Dirks DD. (1983) Accuracy of adaptive procedure estimates of PB-max level. *J Speech Hear Disord.* 48, 202–209.

Katz J. (1962) The use of staggered spondaic words for assessing the integrity of the central auditory nervous system. *J Audit Res.* 2, 327–337.

Killion MC, Niquette PA. (2000) What can the pure-tone audiogram tell us about a patient's SNR loss? *Hear J.* 53, 46–53.

Killion MC, Niquette PA, Gudmundsen GI, Revit LJ, Banerjee S. (2004). Development of a quick speech-in-noise test for measuring signal-to-noise ratio loss in normal-hearing and hearing-impaired listeners. *J Acoust Soc Am.* 116, 2395–2405.

Kimura D. (1961) Some effects of temporal lobe damage on auditory perception. *Can J Psychol.* 15, 157–1165.

Kirk KI, Pisoni DB, Osberger MJ. (1995) Lexical effects of unspoken word recognition by pediatric cochlear implant users. *Ear Hear.* 16, 470–481.

Konkle DF, Rintelmann WF. (1983) Introduction to speech audiometry. In: Konkle DF, Rindtelman WF, eds. *Principles of Speech Audiometry.* Baltimore, MD: University Park Press; pp 1–10.

Kosky C, Boothroyd A. (2003) Validation of an on-line implementation of the Imitative Test of Speech Pattern Contrast Perception (IMSPAC). *J Am Acad Audiol.* 14, 72–83.

Kruel EJ, Bell DW, Nixon JC. (1969) Factors affecting speech discrimination test difficulty. *J Speech Hear Res.* 12, 281–287.

Kruel EJ, Nixon JC, Kryter KD, Bell DW, Lang JS, Schubert ED. (1968) A proposed clinical test of speech discrimination. *J Speech Hear Res.* 11, 536–552.

Lehiste I, Peterson G. (1959) Linguistic considerations in the study of speech intelligibility. *J Acoust Soc Am.* 31, 280–286.

Levitt H, Resnick S. (1978) Speech reception by the hearing impaired. *Scand Audiol.* 6 (suppl), 107–130.

Martin FN, Champlin CA, Chambers JA. (1998) Seventh survey of audiometric practices in the United States. *J Am Acad Audiol.* 9, 95–104.

Matzker J. (1959) Two new methods for the assessment of central auditory functions in cases of brain disease. *Ann Otol Rhinol Laryngol.* 68, 1185–1197.

McArdle R, Chisolm TH, Abrams HB, Wilson RH, Doyle PJ. (2005a) The WHO-DAS II: measuring outcomes of hearing aid intervention for adults. *Trends Amplif.* 9, 127–143.

McArdle R, Wilson RH, Burks CA. (2005b) Speech recognition in multitalker babble using digits, words, and sentences. *J Am Acad Audiol.* 16, 726–739.

McCullough JA, Wilson RH, Birck JD, Anderson LG. (1994) A multimedia approach for estimating speech recognition in multilingual clients. *Am J Audiol.* 3, 19–22.

Miller GA. (1956) The magical number seven, plus or minus two: Some limits on our capacity for processing information. *Psychol Rev.* 63, 81–97.

Moog JS, Geers AE. (1990) *Early Speech Perception Test for Profoundly Deaf Children.* St. Louis, MO: Central Institute for the Deaf.

Nabelek A, Robinson P. (1982) Monaural and binaural speech perception in reverberation for listeners of various ages. *J Acoust Soc Am.* 71, 1242–1248.

Nilsson M, Soli S, Sullivan J. (1994) Development of the Hearing in Noise Test for the measurement of speech reception thresholds in quiet and in noise. *J Acoust Soc Am.* 95, 1085–1099.

Olsen WO, Van Tassell DJ, Speaks CE. (1997) Phoneme and word recognition for words in isolation and in sentences. *Ear Hear.* 18, 175–188.

Owens E, Kessler DT, Raggio MW, Schubert ED. (1985) Analysis and revision of the Minimum Auditory Capabilities (MAC) battery. *Ear Hear.* 6, 280–290.

Owens E, Schubert ED. (1977) Development of the California Consonant Test. *J Speech Hear Res.* 20, 463–474.

Pederson OT, Studebaker GA. (1972) A new minimal-contrast closed-response-set speech test. *J Audit Res.* 12, 187–195.

Penrod JP. (1994) Speech threshold and word recognition/discrimination testing. In: Katz J, ed. *Handbook of Clinical Audiology.* 4th ed. Baltimore, MD: Williams & Wilkins; pp 147–164.

Plomp R. (1978) Auditory handicap of hearing impairment and the limited benefit of hearing aids. *J Acoust Soc Am.* 63, 533–549.

Punch J, Joseph A, Rakerd B. (2004) Most comfortable and uncomfortable loudness levels: six decades of research. *Am J Audiol.* 13, 144–157.

Roeser RJ, Clark JL. (2008). Live voice speech recognition audiometry – stop the madness! *Audiol Today.* 20,32–33.

Ross M, Lerman J. (1970) A picture identification task for hearing-impaired children. *J Speech Hear Res.* 13, 44–53.

Silverman SR, Hirsh IJ. (1955) Problems related to the use of speech in clinical audiometry. *Ann Otol Rhinol Laryngol.* 64, 1234–1244.

Speaks C, Jerger J. (1965) Performance-intensity characteristics of synthetic sentences. *J Speech Hear Res.* 9, 305–312.

Sperry JL, Wiley TL, Chial MR. (1997) Word recognition performance in various background competitors. *J Am Acad Audiol.* 8, 71–80.

Thornton AR, Raffin MJM. (1978) Speech-discrimination scores modeled as a binomial variable. *J Speech Hear Res.* 21, 507–518.

Tillman TW, Carhart R. (1966) *An Expanded Test for Speech Discrimination Utilizing CNC Monosyllabic Words. Northwestern University Auditory Test No. 6.* Brooks Air Force Base, TX: US Air Force School of Aerospace Medicine.

Tillman TW, Carhart R, Wilber L. (1963) *A Test for Speech Discrimination Composed of CNC Monosyllabic Words. Northwestern*

University Auditory Test No. 4. Technical Documentary Report No. SAM-TDR-62–135. Brooks Air Force Base, TX: US Air Force School of Aerospace Medicine.

Tillman TW, Jerger JF. (1959) Some factors affecting the spondee threshold in normal-hearing subjects. *J Speech Hear Res.* 2, 141–146.

Wesilender P, Hodgson WR. (1989) Evaluation of four Spanish word recognition ability lists. *Ear Hear.* 10, 387–392.

Wiley TL, Stoppenbach DT, Feldhake LI, Moss KA, Thordardottir ET. (1995) Audiologic practices: What is popular versus what is supported by evidence. *Am J Audiol.* 4, 26–34.

Wilson RH. (2003) Development of a speech in multitalker babble paradigm to assess word-recognition performance. *J Am Acad Audiol.* 14, 453–470.

Wilson RH, Abrams HB, Pillion AL. (2003) A word-recognition task in multitalker babble using a descending presentation mode from 24 dB to 0 dB in signal to babble. *J Rehabil Res Dev.* 40, 321–328.

Wilson RH, Margolis RH. (1983) Measurements of auditory thresholds for speech stimuli. In: Konkle DF, Rintelmann WF, eds. *Principles of Speech Audiometry.* Baltimore, MD: University Park Press; pp 79–126.

Wilson RH, McArdle R. (2005) Speech signals used to evaluate the functional status of the auditory system. *J Rehabil Res Dev.* 42 (suppl 2), 79–94.

Wilson RH, Morgan DE, Dirks DD. (1973) A proposed SRT procedure and its statistical precedent. *J Speech Hear Disord.* 38, 184–191.

Wilson RH, Preece JP, Salamon DL, Sperry JL, Bornstein SP. (1994) Effects of time compression and time compression plus reverberation on the intelligibility of Northwestern University Auditory Test No. 6. *J Am Acad Audiol.* 5, 269–277.

Wilson RH, Preece JP, Thornton AR. (1990) Clinical use of the compact disc in speech audiometry. *ASHA.* 32, 3247–3251.

Wilson RH, Strouse A. (1999) Auditory measures with speech signals. In: Musiek FE, Rintelmann WF, eds. *Contemporary Perspectives in Hearing Assessment.* Needham Heights, MA: Allyn & Bacon; pp 21–66.

Wilson RH, Strouse A. (2002) Northwestern University Audiology Test #6 in multitalker bubble: A preliminary report. *J Rehabil Res Dev.* 39, 105–113.

Wilson RH, Trivette CP, Williams DA, Watts KA. (2012). The effects of energetic and informational masking on the Words-in-Noise Test (WIN). *J Am Acad Audiol.* 23, 522–533.

Wilson RH, Weakley DG. (2004) The use of digit triplets to evaluate word-recognition abilities in multitalker babble. *Semin Hear.* 25, 93–111.

Young L, Dudley B, Gunter MB. (1982) Thresholds and psychometric functions of the individual spondaic words. *J Speech Hear Res.* 25, 586–593.

CHAPTER 6

Clinical Masking

William S. Yacullo

In the first edition of this text, Sanders (1972) wrote the following introduction to his chapter on clinical masking:

> Of all the clinical procedures used in auditory assessment, masking is probably the most often misused and the least understood. For many clinicians the approach to masking is a haphazard hit-or-miss bit of guesswork with no basis in any set of principles. (p 111)

Unfortunately, this statement may still hold true today.

The principles of clinical masking are difficult for many beginning clinicians to understand. Although the clinician can apply masking formulas and procedures appropriately in most clinical situations, a lack of understanding of the underlying theoretical constructs becomes evident during cases where modification of a standard procedure is required. A lack of understanding of the underlying concepts of masking often leads to misuse of clinical procedures.

Theoretical and empirical bases of masking provide a strong foundation for the understanding of applied clinical masking procedures. It will become evident throughout this chapter that there is not a single "correct" approach to clinical masking. Any approach to clinical masking that is based on sound theoretical constructs and verified through clinical experience is correct. One approach will not meet all clinical needs. A strong foundation in the underlying concepts of clinical masking serves three purposes. First, it allows the clinician to make correct decisions about the need for masking. Second, it allows the clinician to make a well-informed decision when selecting a specific approach to clinical masking. Finally, it allows the clinician to apply and modify a clinical masking procedure appropriately.

THE NEED FOR MASKING

A major objective of the basic audiologic evaluation is assessment of auditory function of each ear. There are situations during both air-conduction and bone-conduction testing when this may not occur. Although a puretone or speech stimulus is being presented through a transducer to the test ear, the nontest ear can contribute partially or totally to the observed response. Whenever it is suspected that the nontest ear is responsive during evaluation of the test ear, a masking stimulus must be applied to the nontest ear to eliminate its participation.

Air-Conduction Testing

Cross hearing occurs when a stimulus presented to the test ear "crosses over" and is perceived in the nontest ear. There are two parallel pathways by which sound presented through an earphone (i.e., an air-conduction transducer) can reach the nontest ear. Specifically, there are both bone-conduction and air-conduction pathways between an air-conduction signal presented at the test ear and the sound reaching the nontest ear cochlea (Studebaker, 1979). First, the earphone can vibrate with sufficient force to cause deformations of the bones of the skull. An earphone essentially can function as a bone vibrator at higher sound pressures. Because both cochleas are housed within the same skull, the outcome is stimulation of the nontest ear cochlea through bone conduction. Second, sound from the test earphone can travel around the head to the nontest ear, enter the opposite ear canal, and finally reach the nontest ear cochlea through an air-conduction pathway. Because the opposite ear typically is covered during air-conduction testing, sound attenuation provided by the earphone will greatly minimize or eliminate the contribution of the air-conduction pathway to the process of cross hearing. Consequently, cross hearing during air-conduction testing is considered primarily a bone-conduction mechanism.

Cross hearing is the result of limited interaural attenuation (IA). IA refers to the "reduction of energy between ears." Generally, it represents the amount of separation or the degree of isolation between ears during testing. Specifically, it is the decibel difference between the hearing level (HL) of the signal at the test ear and the HL reaching the nontest ear:

$$IA = dB\,HL_{Test\,Ear} - dB\,HL_{Nontest\,Ear}$$

Consider the following hypothetical examples presented in Figure 6.1. You are measuring puretone air-conduction threshold using traditional supra-aural earphones. A puretone signal of 90 dB HL is presented to the test ear. Because of limited IA, a portion of the test signal can

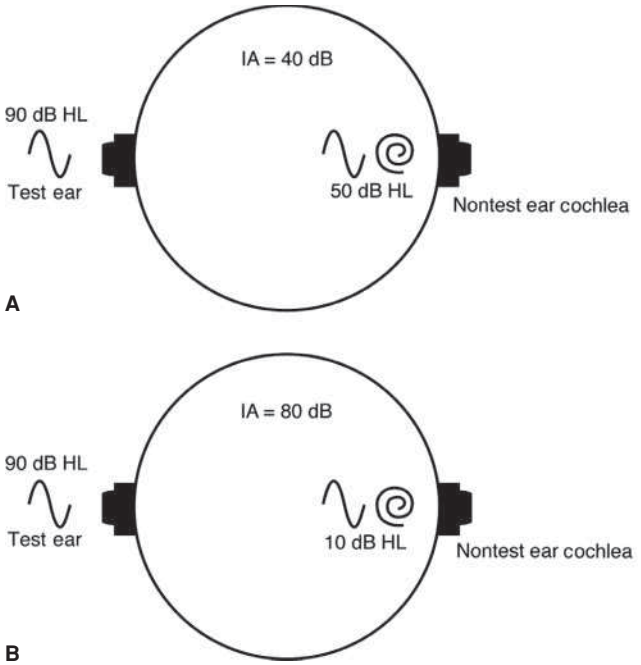


FIGURE 6.1 Interaural attenuation (IA) is calculated as the difference between the hearing level (HL) of the signal at the test ear and the HL reaching the nontest ear cochlea. A puretone signal of 90 dB HL is being presented to the test ear through traditional supra-aural earphones. **Example A:** If IA is 40 dB, then 50 dB HL is reaching the nontest ear cochlea. **Example B:** If IA is 80 dB, then 10 dB HL is reaching the nontest ear cochlea. (From Yacullo WS. (1996) *Clinical Masking Procedures*. 1st ed. Boston, MA: Allyn & Bacon, © 1996, p 3. Adapted by permission of Pearson Education, Inc., Upper Saddle River, NJ.)

reach the nontest ear cochlea. If IA is 40 dB, then 50 dB HL theoretically is reaching the nontest ear:

$$\begin{aligned} \text{IA} &= \text{dB HL}_{\text{Test Ear}} - \text{dB HL}_{\text{Nontest Ear}} \\ &= 90\ \text{dB HL} - 50\ \text{dB HL} \\ &= 40\ \text{dB} \end{aligned}$$

If IA is 80 dB, then only 10 dB HL is reaching the nontest ear. It should be apparent that a greater portion of the test signal can reach the nontest ear when IA is small. Depending on the hearing sensitivity in the nontest ear, cross hearing can occur.

IA during earphone testing is dependent on three factors: Transducer type, frequency spectrum of the test signal, and individual subject. There are three major types of earphones currently used during audiologic testing: Supra-aural, circumaural, and insert (American National Standards Institute/Acoustical Society of America [ANSI/ASA], 2010). Supra-aural earphones use a cushion that makes contact solely with the pinna. Circumaural earphones use a cushion that encircles or surrounds the pinna, making contact with the skin covering the cranial skull. Insert earphones are coupled to the ear by insertion into the ear canal.

Generally, IA increases as the contact area of the transducer with the skull decreases (Zwislocki, 1953). More specifically, IA is greater for supra-aural than circumaural earphones. Furthermore, IA is greatest for insert earphones (Killion et al., 1985; Sklare and Denenberg, 1987), partly because of their smaller contact area with the skull. (The reader is referred to Killion and Villchur, 1989; Zwislocki et al., 1988, for a review of advantages and disadvantages of earphones in audiometry.) Because supra-aural and insert earphones are most typically used during audiologic testing, they will be the focus of this discussion.

There are different approaches to measuring IA for air-conducted sound (e.g., "masking" method, "compensation" method, method of "best beats"; the reader is referred to Zwislocki, 1953, for discussion). The most direct approach, however, involves measurement of transcranial thresholds (Berrett, 1973). Specifically, IA is measured by obtaining unmasked air-conduction (AC) thresholds in subjects with unilateral, profound sensory/neural hearing loss and then calculating the threshold difference between the normal and impaired ears:

$$\text{IA} = \text{Unmasked AC}_{\text{Impaired Ear}} - \text{Unmasked AC}_{\text{Normal Ear}}$$

For example, if unmasked air-conduction thresholds are obtained at 60 dB HL in the impaired ear and 0 dB HL in the normal ear, then IA is calculated as 60 dB:

$$\begin{aligned} \text{IA} &= 60\ \text{dB HL} - 0\ \text{dB HL} \\ &= 60\ \text{dB} \end{aligned}$$

There is the assumption that air- and bone-conduction thresholds are equal (i.e., no air-bone gaps) in the ear with normal hearing.

Figure 6.2 illustrates the expected *unmasked* puretone air-conduction thresholds in an individual with normal hearing in the left ear and a profound sensory/neural hearing loss in the right ear. Unmasked bone-conduction thresholds, regardless of bone vibrator placement, are expected at HLs consistent with normal hearing in the left ear. If appropriate contralateral masking is not used during air-conduction testing, then a shadow curve will result in the right ear. Because cross hearing is primarily a bone-conduction mechanism, unmasked air-conduction thresholds in the right ear will "shadow" the bone-conduction thresholds in the left (i.e., better) ear by the amount of IA. For example, if IA for air-conducted sound is equal to 60 dB at all frequencies, then unmasked air-conduction thresholds in the right ear theoretically will be measured 60 dB above the bone-conduction thresholds in the better ear. The shadow curve does not represent true hearing thresholds in the right ear. Rather, it reflects cross-hearing responses from the better (i.e., left) ear.

When using supra-aural earphones, IA for puretone air-conducted signals varies considerably, particularly across

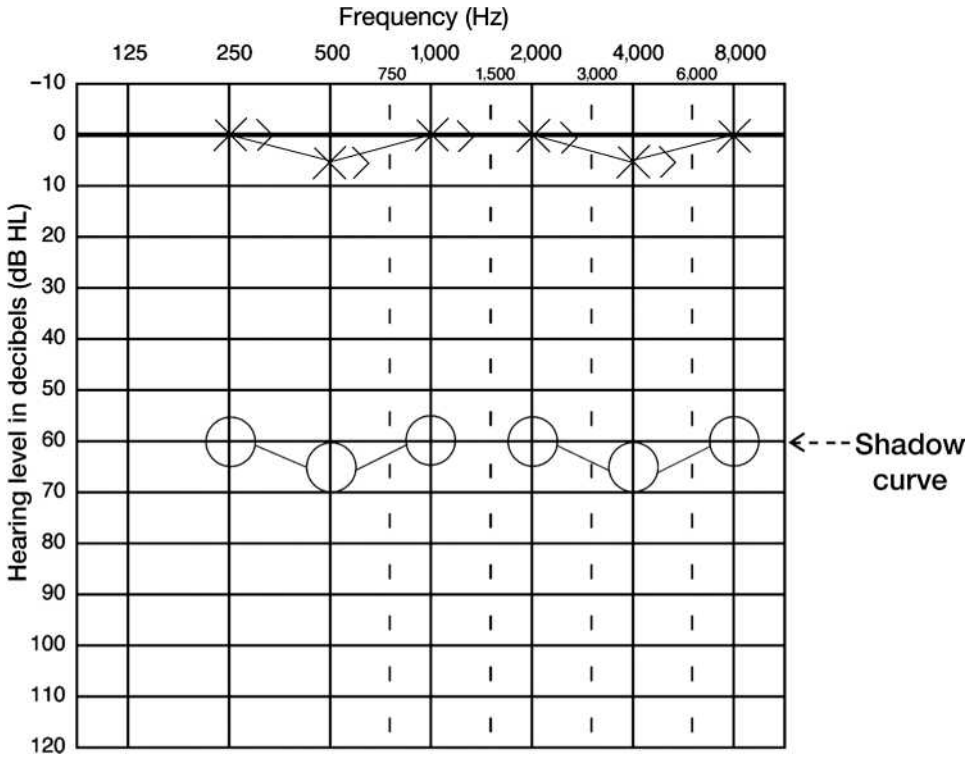


FIGURE 6.2 Expected unmasked puretone air- and bone-conduction thresholds in an individual with normal hearing in the left ear and a profound sensory/neural hearing loss in the right ear.Without the use of appropriate contralateral masking, *a shadow curve* will result in the right ear. Unmasked air-conduction thresholds in the right ear will shadow the bone-conduction thresholds in the better [i.e., left] ear by the amount of interaural attenuation.[From Yacullo WS. [1996] *Clinical Masking Procedures*. 1st ed. Boston, MA: Allyn & Bacon, © 1996, p 7. Adapted by permission of Pearson Education, Inc., Upper Saddle River, NJ.]

subjects, ranging from about 40 to 85 dB (Berrett, 1973; Chaiklin, 1967; Coles and Priede, 1970; Killion et al., 1985; Sklare and Denenberg, 1987; Smith and Markides, 1981; Snyder, 1973). Your assumption about IA will influence the decision about the need for contralateral masking. The use of a smaller IA value assumes that there is smaller separation between ears. Consequently, contralateral masking will be required more often. When making a decision about the need for contralateral masking during clinical practice, a single value defining the lower limit of IA is recommended (Studebaker, 1967a).

Based on currently available data, a conservative estimate of IA for supra-aural earphones is 40 dB at all frequencies.

Although this very conservative estimate will take into account the IA characteristics of all individuals, it will result in the unnecessary use of masking in some instances.

Commonly used insert earphones are the Etymotic Research ER-3A (Killion, 1984) and the E-A-RTONE 3A (E-A-R Auditory Systems, 1997). The ER-3A and the E-A-RTONE 3A insert earphones are considered functionally equivalent because they are built to identical specifications (Frank and Vavrek, 1992). Each earphone consists of a shoulder-mounted transducer, a plastic sound tube of specified length, a nipple adaptor, and a disposable foam eartip. A major advantage of the 3A insert earphone is increased IA for air-conducted sound, particularly in the lower frequencies (Hosford-Dunn et al., 1986; Killion et al., 1985; Sklare and Denenberg, 1987; Van Campen et al., 1990). This is clearly illustrated in the results of a study by Killion et al. (1985) (Figure 6.3).

Increased IA with 3A insert earphones is the result of two factors: (1) Reduced contact area of the transducer with the skull and (2) reduction of the occlusion effect (OE). Zwislocki (1953) evaluated IA for three types of earphones: circumaural, supra-aural, and insert. Results suggested that IA for air-conducted sound increased as the contact area of the earphone with the skull decreased. When an acoustic signal is delivered through an earphone, the resultant sound pressure acts over a surface area of the skull determined by the earphone cushion. The surface area associated with a small eartip will result in a smaller applied force to the skull, resulting in reduced bone-conduction transmission.

Chaiklin (1967) has also suggested that IA may be increased in the low frequencies with a deep insert because of a reduction of the OE. ANSI/ASA (2010) defines the OE as an increase in loudness for bone-conducted sound at

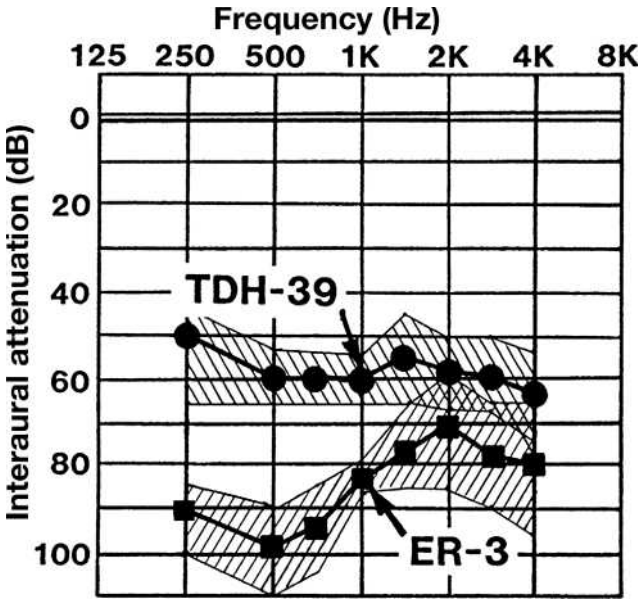


FIGURE 6.3 Average and range of interaural attenuation values obtained on six subjects using two earphones: TDH-39 encased in MX-41/AR supra-aural cushion (●) and ER-3A insert earphone with deeply inserted foam eartip (■). (From Killion MC, Wilber LA, Gudmundsen GI. (1985) Insert earphones for more interaural attenuation. *Hear Instrum.* 36, 34, 36. Reprinted with permission from *Hearing Instruments,* 1985, p 34. *Hearing Instruments* is a copyrighted publication of Advanstar Communications Inc. All rights reserved.)

frequencies below 2,000 Hz when the outer ear is covered or occluded. There is evidence that the OE influences the measured IA for air-conducted sound (e.g., Berrett, 1973; Chaiklin, 1967; Feldman, 1963; Killion et al., 1985; Littler et al., 1952; Van Campen et al., 1990; Zwislocki, 1953). In fact, there is an inverse relationship between magnitude of the OE and the measured IA in the lower frequencies. Specifically, an earphone that reduces the OE will exhibit increased IA for air-conducted sound. Recall that cross hearing occurs primarily through the mechanism of bone conduction. When the nontest ear is covered or occluded by an air-conduction transducer, the presence of an OE will enhance hearing sensitivity for bone-conducted sound in that ear. Consequently, the separation between ears (i.e., IA) is reduced. The increased IA for air-conducted sound observed in the lower frequencies when using 3A insert earphones (with deeply inserted foam eartips) is primarily related to the significant reduction or elimination of the OE. The OE is presented in greater detail later in this chapter in the section on clinical masking procedures during bone-conduction audiometry.

If increased IA is a primary goal when selecting an insert earphone, then the 3A is the transducer of choice. Evidence suggests that the 3A insert earphone provides significantly greater IA, particularly in the lower frequencies, than the "button" transducer (Blackwell et al., 1991; Hosford-Dunn et al., 1986). Blackwell et al. (1991) compared the IA obtained with a standard supra-aural earphone (TDH-50P) and a button transducer fitted with a standard immittance probe cuff. Although greater IA was observed with the button transducer, the difference between the insert and supra-aural earphone did not exceed 10 dB at any frequency.

There are only limited data available regarding IA of 3A insert earphones using deeply or intermediately inserted foam eartips. IA values vary across subjects and frequency, ranging from about 75 to 110 dB at frequencies of ≤1,000 Hz and about 50 to 95 dB at frequencies >1,000 Hz (Killion et al., 1985; Sklare and Denenberg, 1987; Van Campen et al., 1990). Based on Studebaker's (1967a) recommendation, we will again use the smallest IA values reported when making a decision about the need for contralateral masking. To take advantage of the significantly increased IA proved by the 3A insert in the lower frequencies, a single value of IA will not be employed across the frequency range.

Based on currently available data, conservative estimates of IA for 3A insert earphones with deeply inserted foam eartips are 75 dB at ≤1,000 Hz and 50 dB at frequencies >1,000 Hz.

The IA values recommended clinically for 3A earphones assume that deeply inserted foam eartips are used. Maximum IA is achieved in the low frequencies when a deep eartip insertion is used (Killion et al., 1985). The recommended deep insertion depth is achieved when the outer edge of the eartip is 2 to 3 mm inside the entrance of the ear canal. Conversely, a shallow insertion is obtained when the outer edge of the eartip protrudes from the entrance of the ear canal (E-A-R Auditory Systems, 1997). An intermediate insertion is achieved when the outer edge of the eartip is flush with the opening of the ear canal (Van Campen et al., 1990). There are limited data suggesting that IA is similar for either intermediate or deep insertion of the foam eartip. However, a shallow insertion appears to significantly reduce IA (Killion et al., 1985; Sklare and Denenberg, 1987; Van Campen et al., 1990). Remember that a major factor contributing to superior IA of the 3A insert earphone is a significantly reduced OE. There is evidence that the OE is negligible when using either deeply or intermediately inserted insert earphones. In fact, the advantage of a greatly reduced OE is lost when a shallow insertion is used (Berger and Kerivan, 1983). To achieve maximum IA with 3A insert earphones, deeply inserted eartips are strongly recommended.

More recently, E-A-R Auditory Systems (2000a, 2000b) introduced a next-generation insert earphone, the E-A-RTONE 5A. The lengthy plastic sound tube that conducted sound from the body-level transducer of the 3A has been eliminated in the 5A model; rather, the foam eartip is coupled directly to an ear-level transducer. Very limited data obtained with only two subjects (unpublished research by Killion, 2000, as cited in E-A-R Auditory Systems, 2000b) suggest that the average IA for puretone stimuli ranging from 250 to 4,000 Hz is equivalent (within approximately

5 dB) to the average values reported for the 3A insert earphone (Killion et al., 1985).

IA for speech is typically measured by obtaining speech recognition thresholds (SRTs) in individuals with unilateral, profound sensory/neural hearing loss. Specifically, the difference in threshold between the normal ear and impaired ear without contralateral masking is calculated:

$$\text{IA} = \text{Unmasked SRT}_{\text{Impaired Ear}} - \text{SRT}_{\text{Normal Ear}}$$

Recall that SRT represents the lowest HL at which speech is recognized 50% of the time (ANSI/ASA, 2010; American Speech-Language-Hearing Association [ASHA], 1988). IA for spondaic words presented through supra-aural earphones varies across subjects and ranges from 48 to 76 dB (Martin and Blythe, 1977; Sklare and Denenberg, 1987; Snyder, 1973). Again, a single value defining the lower limit of IA is recommended when making a decision about the need for contralateral masking (Studebaker, 1967a). A conservative estimate of IA for spondees, therefore, is 45 dB when using supra-aural earphones (Konkle and Berry, 1983). The majority of audiologists measure SRT using a 5-dB step size (Martin et al., 1998). Therefore, the IA value of 48 dB is typically rounded down to 45 dB.

There is considerable evidence that speech can be detected at a lower HL than that required to reach SRT. Speech detection threshold (SDT) is defined as the lowest HL at which speech can be detected or "discerned" 50% of the time (ASHA, 1988). The SRT typically requires an average of about 8 to 9 dB greater HL than that required for the detection threshold (Beattie et al., 1978; Chaiklin, 1959; Thurlow et al., 1948). Given this relationship between the two speech thresholds, Yacullo (1996) has suggested that a more conservative value of IA may be appropriate when considering the need for contralateral masking during measurement of SDT.

Consider the following hypothetical example. You are measuring speech thresholds in a patient with normal hearing in the right ear and a profound, sensory/neural hearing loss in the left ear. If the patient exhibits the minimum reported IA value for speech of 48 dB, then an SRT of 0 dB HL would be measured in the right ear and an *unmasked* SRT of 48 dB HL would be measured in the left ear. If an unmasked SDT is subsequently measured in the left ear, it is predicted that the threshold would occur at an HL of about 8 to 9 dB lower than the unmasked SRT. An unmasked SDT would be expected to occur at about 39 to 40 dB HL. Comparison of the unmasked SDT in the impaired ear with the SRT in the normal ear theoretically would result in measured IA of approximately 39 to 40 dB. When an unmasked SDT is measured and the response is compared to the SRT in the nontest ear, a more conservative estimate of IA for speech may be appropriate.

It should be noted that the actual IA for speech does not change during measurement of speech detection and recognition thresholds. Rather, a different response task when measuring *different* speech thresholds in each ear (i.e., SDT in one ear and SRT in the other) can affect the measured IA for speech. Comparison of SRTs between ears or SDTs between ears generally should result in the same measured IA. Smith and Markides (1981) measured IA for speech in 11 subjects with unilateral, profound hearing loss. IA was calculated as the difference between the SDT in the better ear and the unmasked SDT in the poorer ear. The range of IA values was 50 to 65 dB. It is interesting to note that the lowest IA value reported for speech using a detection task in each ear was 50 dB, a value comparable to the lowest minimum reported IA value (i.e., 48 dB) for spondaic words (e.g., Martin and Blythe, 1977; Snyder, 1973).

There is also some evidence that it may be appropriate to use a more conservative estimate of IA when making a decision about the need for contralateral masking during assessment of suprathreshold speech recognition. Although IA for the speech signal remains constant during measurement of threshold or suprathreshold measures of speech recognition (i.e., the decibel difference between the level of the speech signal at the test ear and the level at the nontest ear cochlea), differences in the performance criterion for each measure must be taken into account when selecting an appropriate IA value for clinical use. SRT is defined relative to a 50% response criterion. However, suprathreshold speech recognition performance can range from 0% to 100%.

Konkle and Berry (1983) provide an excellent rationale for the use of a more conservative estimate of IA when measuring suprathreshold speech recognition. They suggest that the fundamental difference in percent correct criterion requires the specification of nontest ear cochlear sensitivity in a different way than that used for threshold measurement. If suprathreshold speech recognition materials are presented at an HL equal to the SRT, then a small percentage of the test items can be recognized. It should be noted that the percentage of test words that can be recognized at an HL equal to SRT is dependent on the type of speech stimuli, as well as on the talker and/or recorded version of a speech recognition test. Regardless of the type of speech stimulus (e.g., meaningful monosyllabic words, nonsense syllables, or sentences) and the specific version (i.e., talker/recording) of a speech recognition test, 0% performance may not be established until an HL of about −10 dB relative to the SRT. Konkle and Berry (1983) recommend that the value of IA used for measurement of suprathreshold speech recognition should be estimated as 35 dB. That is, the IA value of 45 dB (rounded down from 48 dB) based on SRT measurement is adjusted by subtracting 10 dB. This adjustment in the estimate of IA reflects differences in percent correct criterion used for speech threshold and suprathreshold measurements.

The majority of audiologists use an IA value of 40 dB for all air-conduction measurements, both puretone and

speech, when making a decision about the need for contralateral masking (Martin et al., 1998). The use of a single IA value of 40 dB for both threshold and suprathreshold speech audiometric measurements can be supported. Given the smallest reported IA value of 48 dB for spondaic words, a value of 40 dB is somewhat too conservative during measurement of SRT. However, it should prove adequate during measurement of SDT and suprathreshold speech recognition when a more conservative estimate of IA (by approximately 10 dB) may be appropriate.

Unfortunately, there are only very limited data available about IA for speech when using insert earphones. Sklare and Denenberg (1987) reported IA for speech (i.e., SRT using spondaic words) in seven adults with unilateral, profound sensory/neural hearing loss using ER-3A insert earphones. IA ranged from 68 to 84 dB. It should be noted that the smallest reported value of IA for spondaic words (i.e., 68 dB) is 20 dB greater when using 3A insert earphones with deeply inserted foam eartips (Sklare and Denenberg, 1987) than when using supra-aural earphones (i.e., 48 dB) (Martin and Blythe, 1977; Snyder, 1973). Therefore, a value of 60 dB represents a very conservative estimate of IA for speech when using 3A insert earphones. This value is derived by adding a correction factor of 20 dB to the conservative IA value used with supra-aural earphones (i.e., 40 dB) for all threshold and suprathreshold measures of speech.

Based on currently available data, conservative estimates of IA for all threshold and suprathreshold measures of speech are 40 dB for supra-aural earphones and 60 dB for 3A insert earphones with deeply inserted foam eartips.

Bone-Conduction Testing

There are two possible locations for placement of a bone vibrator (typically, the Radioear B-71) during puretone threshold audiometry: The mastoid process of the temporal bone and the frontal bone (i.e., the forehead). Although there is some evidence that a forehead placement produces more reliable and valid thresholds than a mastoid placement (see Dirks, 1994, for further discussion), the majority (92%) of audiologists in the United States continue to place a bone-conduction transducer on the mastoid process (Martin et al., 1998).

IA is greatly reduced during bone-conduction audiometry. IA for bone-conducted sound when using a bone vibrator placed at the forehead is essentially 0 dB at all frequencies; IA when using a mastoid placement is approximately 0 dB at 250 Hz and increases to about 15 dB at 4,000 Hz (Studebaker, 1967a). Regardless of the placement of a bone vibrator (i.e., mastoid vs. forehead), it is generally agreed that IA for bone-conducted sound at all frequencies is negligible and should be considered 0 dB (e.g., Dirks, 1994; Hood, 1960; Sanders and Rintelmann, 1964; Studebaker, 1967a). When a bone vibrator, regardless of its location, sets the bones of the skull into vibration, both cochleas can be potentially stimulated. Consequently, an unmasked bone-conduction threshold can reflect a response from either cochlea or perhaps both. Although a bone vibrator may be placed at the side of the test ear, it cannot be assumed that the observed response is in fact from that ear.

Consider the following example. You have placed a bone vibrator at the right mastoid process. A puretone signal of 50 dB HL is presented. If IA is considered to be 0 dB, then it should be assumed that a signal of 50 dB HL is potentially reaching both cochleas. It should be apparent that there is essentially no separation between the two cochleas during unmasked bone-conduction audiometry.

Based on currently available data, a conservative estimate of IA for bone-conducted sound is 0 dB at all frequencies.

WHEN TO MASK

Contralateral masking is required whenever there is the possibility that the test signal can be perceived in the nontest ear. IA is one of the major factors that will be considered when evaluating the need for masking. The basic principles underlying the decision-making processes of when to mask during puretone and speech audiometry will now be addressed.

Puretone Audiometry: Air Conduction

When making a decision about the need for masking during puretone air-conduction testing, three factors need to be considered: (1) IA, (2) unmasked air-conduction threshold in the test ear (i.e., HL at the test ear), and (3) bone-conduction hearing sensitivity (i.e., threshold) in the nontest ear. Recall that when cross hearing occurs, the nontest ear is stimulated primarily through the bone-conduction mechanism. When a decision is made about the need for contralateral masking, the unmasked air-conduction threshold in the test ear ($AC_{Test\ Ear}$) is compared to the bone-conduction threshold in the nontest ear ($BC_{Nontest\ Ear}$). If the difference between ears equals or exceeds IA, then air-conduction threshold in the test ear must be remeasured using contralateral masking. The rule for when to mask during puretone air-conduction testing can be stated as follows:

Contralateral masking is required during puretone air-conduction audiometry when the unmasked air-conduction threshold in the test ear equals or exceeds the apparent bone-conduction threshold (i.e., the unmasked bone-conduction threshold) in the nontest ear by a conservative estimate of IA:

$$AC_{Test\ Ear} - BC_{Nontest\ Ear} \geq IA$$

This rule is consistent with the guidelines for manual puretone threshold audiometry recommended by ASHA (2005).

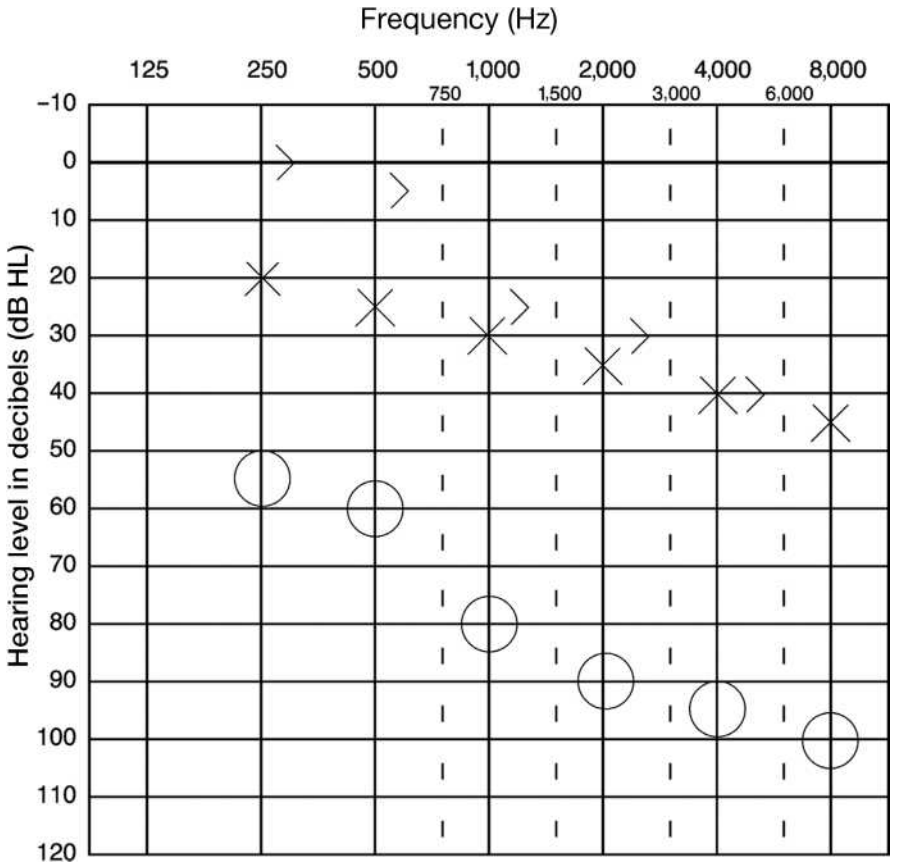


FIGURE 6.4 Audiogram illustrating the need for contralateral masking during puretone air-conduction audiometry. See text for discussion.

Note that the term "apparent" bone-conduction threshold is considered when making a decision about the need for masking. Remember that an unmasked bone-conduction threshold does not convey ear-specific information. It is assumed that the bone-conduction response can originate from either or both ears. Therefore, the unmasked bone-conduction response is considered the apparent or possible threshold for either ear.

Consider the unmasked puretone audiogram* presented in Figure 6.4. Because IA for bone-conducted sound is considered 0 dB, unmasked bone-conduction thresholds are traditionally obtained at only one mastoid process. During air-conduction threshold testing, the potential for cross hearing is greatest when there is a substantial difference in hearing sensitivity between the two ears and when a stimulus is presented at higher HLs to the poorer ear. Consequently, there is greater potential for cross hearing when measuring puretone thresholds in the right ear.

First consider the need for contralateral masking assuming that air-conduction thresholds were measured using supra-aural earphones. A conservative estimate of IA is 40 dB. We will use the following equation when making a decision about the need for contralateral masking:

$$AC_{Test\,Ear} - BC_{Nontest\,Ear} \geq IA$$

Because it is not possible to measure bone-conduction threshold at 8,000 Hz, it is necessary to predict an unmasked threshold given the findings at other test frequencies. In this particular example, unmasked bone-conduction threshold at 8,000 Hz will probably have a similar relationship with the air-conduction thresholds in the better (i.e., left) ear. Because there is no evidence of air-bone gaps at the adjacent high frequencies, we will assume that a similar relationship exists at 8,000 Hz. Therefore, our estimate of unmasked bone-conduction threshold is 45 dB HL.

It will be necessary to remeasure puretone thresholds at all test frequencies in the right ear using contralateral masking because the difference between ears equals or exceeds our estimate of IA.

*The puretone audiogram and audiometric symbols used throughout this chapter are those recommended in ASHA's (1990) most recent guidelines for audiometric symbols (see Chapter 3).

Right Ear (Test Ear)		Masking Needed?
250 Hz	55–0 ≥40?	Yes
500 Hz	60–5 ≥40?	Yes
1,000 Hz	80–25 ≥40?	Yes
2,000 Hz	90–30 ≥40?	Yes
4,000 Hz	95–40 ≥40?	Yes
8,000 Hz	100–45 ≥40?	Yes

However, contralateral masking is not required when testing the left ear. The difference between ears does not equal or exceed the estimate of IA.

Left Ear (Test Ear)		Masking Needed?
250 Hz	20–0 ≥40?	No
500 Hz	25–5 ≥40?	No
1,000 Hz	30–25 ≥40?	No
2,000 Hz	35–30 ≥40?	No
4,000 Hz	40–40 ≥40?	No
8,000 Hz	45–45 ≥40?	No

Many audiologists will obtain air-conduction thresholds prior to measurement of bone-conduction thresholds. A preliminary decision about the need for contralateral masking can be made by comparing the air-conduction thresholds of the two ears.

Contralateral masking is required during puretone air-conduction audiometry when the unmasked air-conduction threshold in the test ear ($AC_{Test\ Ear}$) equals or exceeds the air-conduction threshold in the nontest ear ($AC_{Nontest\ Ear}$) by a conservative estimate of IA:

$$AC_{Test\ Ear} - AC_{Nontest\ Ear} \geq IA$$

It is important to remember, however, that cross hearing for air-conducted sound occurs primarily through the mechanism of bone conduction. Consequently, it will be necessary to re-evaluate the need for contralateral masking during air-conduction testing following the measurement of unmasked bone-conduction thresholds.

Consider again the audiogram presented in Figure 6.4. Let us assume that we have not yet measured unmasked bone-conduction thresholds. We can make a preliminary decision about the need for contralateral masking by considering the difference between air-conduction thresholds in the two ears. Based on the air-conduction responses only, it appears that contralateral masking is needed only when testing the right ear at octave frequencies from 1,000 through 8,000 Hz. Yet, once unmasked bone-conduction thresholds are measured, it becomes apparent that contralateral masking will also be required when testing the right ear at 250 and 500 Hz.

It is conventional to obtain air-conduction thresholds prior to bone-conduction thresholds. However, an alternative (and recommended) approach involves obtaining unmasked bone-conduction thresholds before obtaining unmasked air-conduction thresholds. Decisions about the need for masking during air-conduction testing then can be made using the important bone-conduction responses.

3A insert earphones are often substituted for the supra-aural configuration during audiometric testing. We now will take a second look at the audiogram in Figure 6.4 and assume that air-conduction thresholds were obtained with 3A insert earphones. Recall that conservative estimates of IA for 3A insert earphones with deeply inserted foam eartips are 75 dB at ≤1,000 Hz and 50 dB at frequencies >1,000 Hz. Previously, we determined that contralateral masking was not required when testing the better (i.e., left) ear using supra-aural earphones. Given the greater IA offered by 3A insert earphones, it is easy to understand that contralateral masking again should not be required when testing the left ear. However, a different picture results when considering the need for contralateral masking when testing the right ear.

Right Ear (Test Ear)		Masking Needed?
250 Hz	55–0 ≥75?	No
500 Hz	60–5 ≥75?	No
1,000 Hz	80–25 ≥75?	No
2,000 Hz	90–30 ≥50?	Yes
4,000 Hz	95–40 ≥50?	Yes
8,000 Hz	100–45 ≥50?	Yes

Because of the greater IA provided by 3A insert earphones in the lower frequencies, the need for contralateral masking is eliminated at 250, 500, and 1,000 Hz. It should be apparent that the process of evaluating the need for contralateral masking when using either supra-aural or insert earphones is the same. The only difference is the substitution of different values of IA in our equations.

Puretone Audiometry: Bone Conduction

Remember that a conservative estimate of IA for bone-conducted sound is 0 dB. Theoretically, masked bone-conduction measurements are always required if ear-specific information is needed. However, given the goal of bone-conduction audiometry, contralateral masking is not always required. Generally, bone-conduction thresholds are primarily useful for determining gross site of lesion (i.e., conductive, sensory/neural, or mixed). The presence of air-bone gaps suggests a conductive component to a hearing loss.

The major factor to consider when making a decision about the need for contralateral masking during bone-conduction audiometry is whether the unmasked bone-conduction threshold (Unmasked BC) suggests the presence of a significant conductive component in the test ear.

The use of contralateral masking is indicated whenever the results of unmasked bone-conduction audiometry suggest

the presence of an air-bone gap in the test ear (Air-Bone Gap$_{Test\ Ear}$) of 15 dB or greater:

$$\text{Air-Bone Gap}_{\text{Test Ear}} \geq 15 \text{ dB}$$

where

$$\text{Air-Bone Gap} = \text{AC}_{\text{Test Ear}} - \text{Unmasked BC}$$

ASHA (2005) recommends that contralateral masking should be used whenever a potential air-bone gap of 10 dB or greater exists. When taking into account the variability inherent in bone-conduction measurements (Studebaker, 1967b), however, a criterion of 10 dB may be too stringent. There is a certain degree of variability between air- and bone-conduction threshold, even in individuals without conductive hearing loss. If we assume that there is a normal distribution of the relationship between air- and bone-conduction thresholds in individuals without significant air-bone gaps, then an air-bone difference of ±10 dB is not unexpected.

If unmasked bone-conduction thresholds suggest air-bone gaps of 10 dB or less, then contralateral masking is not required. Although unmasked bone-conduction thresholds do not provide ear-specific information, we have accomplished our goal for bone-conduction testing. If unmasked bone-conduction thresholds suggest no evidence of significant air-bone gaps, then we have ruled out the presence of a significant conductive component. Consequently, our assumption is that the hearing loss is sensory/neural in nature.

Figure 6.5 provides three examples of the need for contralateral masking during bone-conduction audiometry.

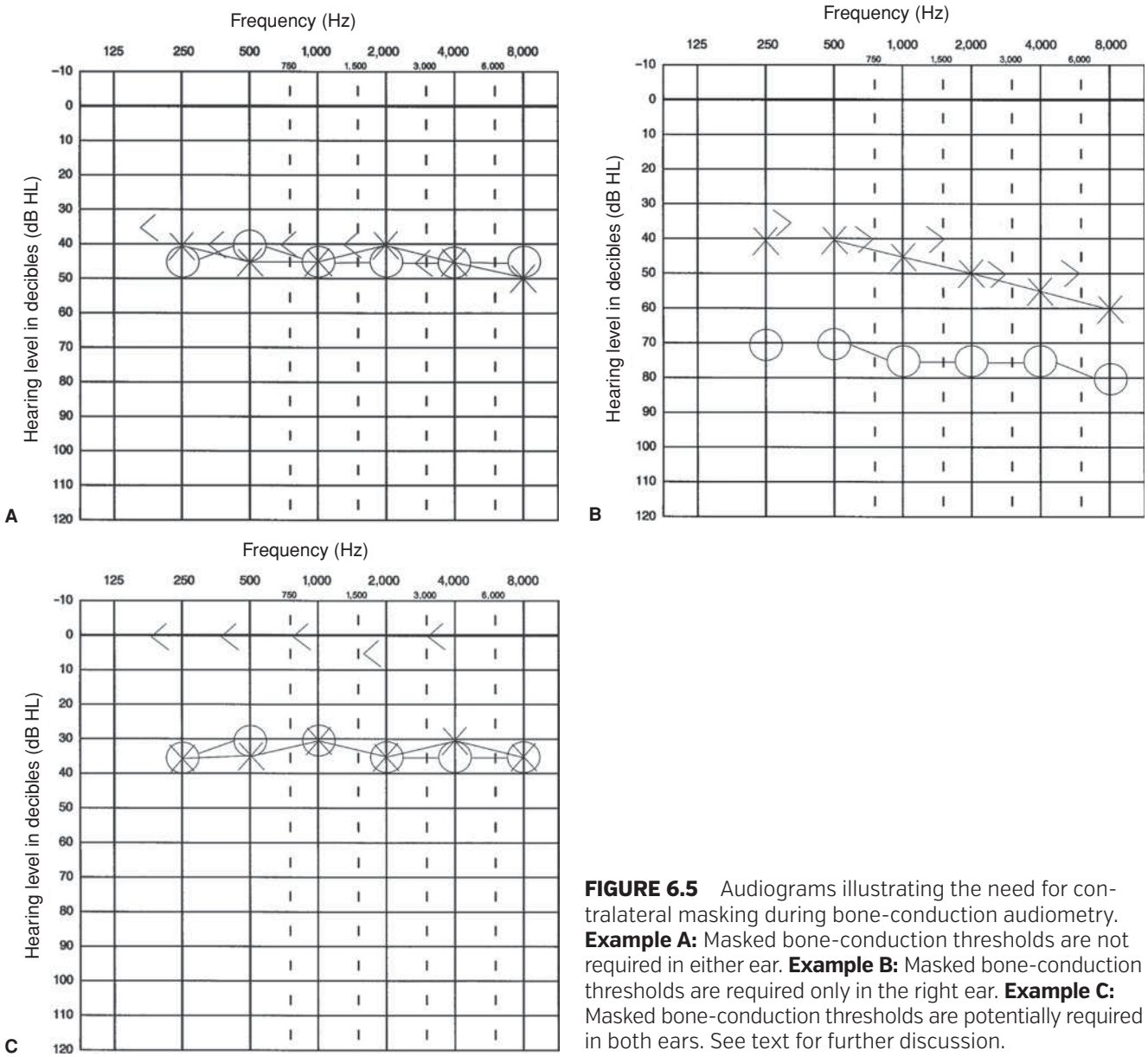


FIGURE 6.5 Audiograms illustrating the need for contralateral masking during bone-conduction audiometry. **Example A:** Masked bone-conduction thresholds are not required in either ear. **Example B:** Masked bone-conduction thresholds are required only in the right ear. **Example C:** Masked bone-conduction thresholds are potentially required in both ears. See text for further discussion.

Unmasked air- and bone-conduction thresholds are provided in each case.

Example A. Contralateral masking is not required during bone-conduction testing in either ear. When we compare the unmasked bone-conduction thresholds to the air-conduction thresholds in each ear, there are no potential air-bone gaps of 15 dB or greater. For example, consider the thresholds at 2,000 Hz. Comparison of the unmasked bone-conduction threshold to the air-conduction thresholds suggests a potential air-bone gap of 5 dB in the right ear and 0 dB in the left ear. Because the unmasked bone-conduction threshold does not suggest the presence of significant air-bone gaps in either ear, our conclusion is that the hearing loss is sensory/neural bilaterally. Obtaining masked bone-conduction thresholds, although they would provide ear-specific information, would not provide additional diagnostic information.

Example B. Comparison of unmasked bone-conduction thresholds to the air-conduction thresholds in the left ear does not suggest the presence of significant air-bone gaps. Consequently, masked bone-conduction thresholds are not required in the left ear. Our conclusion is that the hearing loss is sensory/neural.

However, masked bone-conduction thresholds will be required in the right ear. Comparison of unmasked bone-conduction thresholds to the air-conduction thresholds in the right ear suggests potential air-bone gaps ranging from 25 to 35 dB. The unmasked bone-conduction thresholds may reflect hearing in the better (i.e., left) ear. Bone-conduction thresholds in the right ear may be as good as the unmasked responses. They also may be as poor as the air-conduction thresholds in that ear. Because we do not have ear-specific information for bone-conduction thresholds, the loss in the right ear can be either mixed or sensory/neural. To make a definitive statement about the type of hearing loss, it will be necessary to obtain masked bone-conduction thresholds in the right ear.

Example C. There is evidence that contralateral masking will be required when measuring bone-conduction thresholds in both ears. Comparison of unmasked bone-conduction thresholds to the air-conduction thresholds suggests potential air-bone gaps ranging from 30 to 35 dB in each ear. As in the previous example, bone-conduction thresholds in each ear may be as good as the unmasked responses. They may also be as poor as the air-conduction thresholds in that ear. To make a definitive statement about the type of hearing loss, it will be necessary to obtain masked bone-conduction thresholds in both ears.

Speech Audiometry

Because speech audiometry is an air-conduction procedure, the rules for when to mask will be similar to those used during puretone air-conduction audiometry. There are three factors to consider when making a decision about the need for contralateral masking during speech audiometry: (1) IA, (2) presentation level of the speech signal (in dB HL) in the test ear, and (3) bone-conduction hearing sensitivity (i.e., threshold) in the nontest ear.

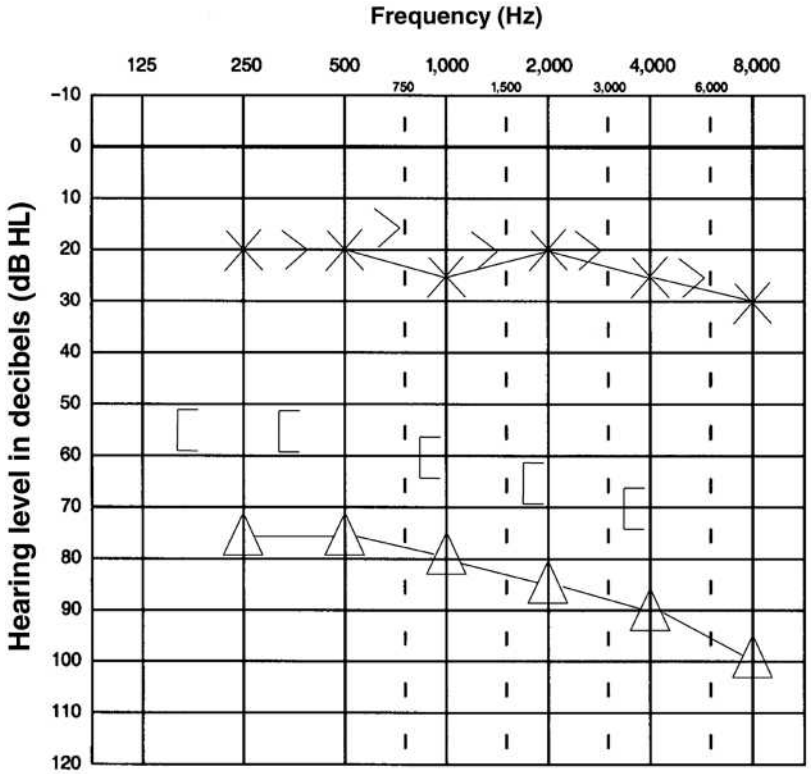
Contralateral masking is indicated during speech audiometry whenever the presentation level of the speech signal (in dB HL) in the test ear (Presentation Level$_{Test\ Ear}$) equals or exceeds the best puretone bone-conduction threshold in the nontest ear (Best BC$_{Nontest\ Ear}$) by a conservative estimate of IA:

$$\text{Presentation Level}_{\text{Test Ear}} - \text{Best BC}_{\text{Nontest Ear}} \geq \text{IA}$$

Because speech is a broadband signal, it is necessary to consider bone-conduction hearing sensitivity at more than a single puretone frequency. Konkle and Berry (1983) and Sanders (1991) recommend the use of the bone-conduction puretone average of 500, 1,000, and 2,000 Hz or some other average that is predictive of the SRT. ASHA (1988) recommends that the puretone bone-conduction thresholds at 500, 1,000, 2,000, and 4,000 Hz should be considered. Martin and Blythe (1977) suggest that 250 Hz can be eliminated from any formula for determining the need for contralateral masking when measuring the SRT. Yet, the nontest ear bone-conduction threshold at 250 Hz may be an important consideration when measuring the SDT. Olsen and Matkin (1991) state that the SDT may be most closely related to the best threshold in the 250 to 4,000 Hz range when audiometric configuration steeply rises or slopes. Therefore, following the recommendation of Coles and Priede (1975), the most conservative approach involves considering the best bone-conduction threshold in the 250- to 4,000-Hz frequency range.

The examples presented in Figures 6.6 and 6.7 illustrate the need for contralateral masking during threshold and suprathreshold speech audiometry, respectively. First consider the audiogram presented in Figure 6.6. Audiometry was performed using supra-aural earphones. Puretone testing (using appropriate contralateral masking during both air- and bone-conduction audiometry) reveals a severe-to-profound, sensory/neural hearing loss of gradually sloping configuration in the right ear. There is a very mild, sensory/neural hearing loss of relatively flat configuration in the left ear. Given the difference between ears observed during puretone audiometry, it is anticipated that contralateral masking may be needed during assessment of SRT in the poorer ear.

There are different approaches that can be used when determining the need for contralateral masking during measurement of SRT. The most efficient and recommended approach involves predicting the speech threshold using the puretone threshold data in the poorer ear and, on that basis, determining the need for contralateral masking. For example, SRT is measured at 20 dB HL in the left ear, a finding consistent with the puretone results. Given the relatively low HL at which the SRT was established in the better (i.e.,



Response

Modality	Ear: Left	Ear: Unspecified	Ear: Right
Air conduction Earphones			
Unmasked	×		○
Masked	□		△
Bone conduction Mastoid			
Unmasked	>	^	<
Masked	⌋		⌊

No response

Modality	Ear: Left	Ear: Unspecified	Ear: Right
Air conduction Earphones			
Unmasked	×		○
Masked	□		△
Bone conduction Mastoid			
Unmasked	>	^	<
Masked	⌋		⌊

Effective masking levels to nontest ear

Test condition	Nontest ear	125	250	500	750	1,000	1,500	2,000	3,000	4,000	6,000	8,000
AC	L		50	50		55		55		60		75
	R											
BC	L		90	90		95		80		90		
	R											

Abbreviations

AC	Air conduction
BC	Bone conduction
CNT	Could not test
DNT	Did not test
EM	Effective masking
FA	Fletcher average
LDL	Loudness discomfort level
MCL	Most comfortable loudness
NR	No response
PTA	Puretone average
SDT	Speech detection threshold
SL	Sensation level
SRT	Speech recognition threshold

Speech audiometry

	PTA	SRT	SDT	MCL / LDL	Speech recognition
					Test:
					SL / % ; SL / %
Right ear	80	80?			
Effective masking in left ear		dB EM	dB EM		dB EM ; dB EM
Left ear	22	20			
Effective masking in right ear		dB EM	dB EM		dB EM ; dB EM

Comments:

FIGURE 6.6 Audiogram illustrating the need for contralateral masking during measurement of speech recognition threshold (SRT). See text for further discussion.

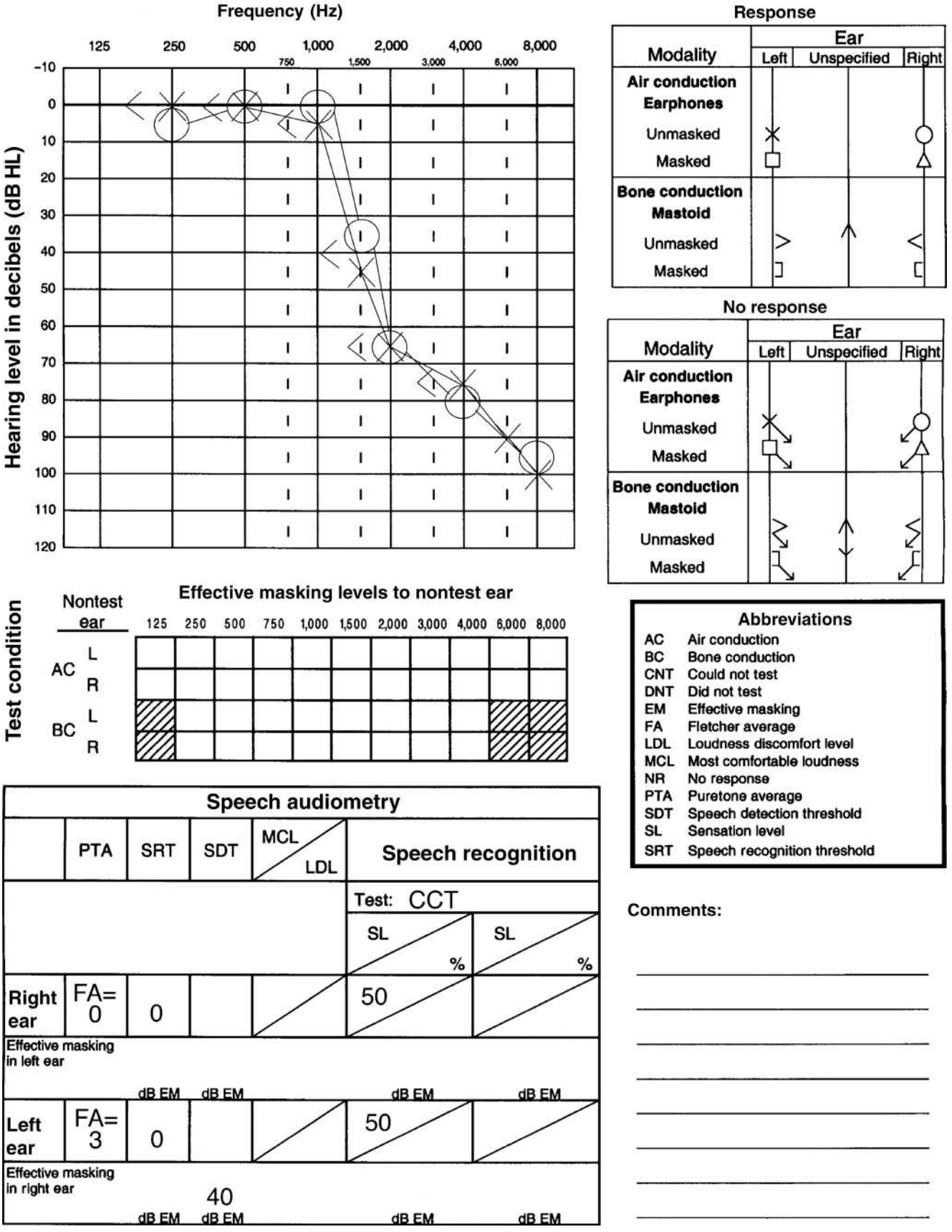


FIGURE 6.7 Audiogram illustrating the need for contralateral masking during measurement of suprathreshold speech recognition. See text for further discussion.

left) ear, it is expected that contralateral masking will not be required when measuring the SRT. Specifically, the SRT of 20 dB HL in the left ear does not equal or exceed the best bone-conduction threshold of 55 dB HL in the nontest ear by a conservative estimate of IA (40 dB):

$$\text{Presentation Level}_{\text{Test Ear}} - \text{Best BC}_{\text{Nontest Ear}} \geq \text{IA}$$

$$20\ \text{dB HL} - 55\ \text{dB HL} \geq 40\ \text{dB?}\ \ \text{No}$$

However, if we predict that an SRT will be measured at about 80 dB HL in the right ear (based on the puretone average), then contralateral masking will be required because the estimated speech threshold of 80 dB HL equals or exceeds the best bone-conduction threshold of 15 dB HL in the nontest ear by 40 dB, our estimate of IA for speech:

$$\text{Presentation Level}_{\text{Test Ear}} - \text{Best BC}_{\text{Nontest Ear}} \geq \text{IA}$$

$$80\ \text{dB HL} - 15\ \text{dB HL} \geq 40\ \text{dB?}\ \ \text{Yes}$$

Stated differently, the difference between the predicted presentation level in the test ear and the best bone-conduction threshold in the nontest ear equals or exceeds our estimate of IA. It is important to note, however, that a decision about the need for contralateral masking during measurement of speech threshold must always take into account not only the presentation level at the measured SRT, but also all suprathreshold levels used during threshold measurement. This will be discussed further in the section addressing selection of masking levels during speech audiometry.

During our earlier discussion of the need for contralateral masking during puretone air-conduction audiometry, it was indicated that a correct decision about the need for contralateral masking can be made sometimes by simply comparing the air-conduction thresholds of the two ears. Similarly, a decision about the need for contralateral masking during measurement of speech thresholds can be often made by comparing speech thresholds in the two ears.

Contralateral masking is required during measurement of speech threshold when the speech threshold in the test ear ($ST_{Test\ Ear}$) equals or exceeds the speech threshold in the nontest ear ($ST_{Nontest\ Ear}$) by a conservative estimate of IA:

$$\text{ST}_{\text{Test Ear}} - \text{ST}_{\text{Nontest Ear}} \geq \text{IA}$$

Consider again the audiogram presented in Figure 6.6. Recall that we predicted that SRT would be measured at about 80 dB HL in the right ear. In this particular example, comparison of the two speech thresholds (i.e., the measured SRT of 20 dB HL in the left ear and the predicted SRT of 80 dB HL in the right ear) would lead us to a correct decision about the need for contralateral masking when measuring SRT in the poorer ear without the need to consider bone-conduction hearing sensitivity in the nontest ear. The difference between the predicted SRT in the right ear (80 dB HL) and the measured SRT in the left ear (20 dB HL) equals or exceeds 40 dB, our estimate of IA for speech:

$$\text{ST}_{\text{Test Ear}} - \text{ST}_{\text{Nontest Ear}} \geq \text{IA}$$

$$80\ \text{dB HL} - 20\ \text{dB HL} \geq 40\ \text{dB?}\ \ \text{Yes}$$

An alternative approach that can be used when making a decision about the need for contralateral masking during assessment of SRT involves measuring unmasked speech thresholds in both ears. Consider again the example presented in Figure 6.6. Assume that unmasked SRTs were measured at 65 and 20 dB HL in the right and left ears, respectively. Again there is an indication that contralateral masking will be required when measuring the SRT in the right ear. The presentation level of 65 dB HL (i.e., the unmasked SRT) in the test ear equals or exceeds the best bone-conduction threshold of 15 dB HL in the nontest ear by 40 dB, our estimate of IA for speech:

$$\text{Presentation Level}_{\text{Test Ear}} - \text{Best BC}_{\text{Nontest Ear}} \geq \text{IA}$$

$$65\ \text{dB HL} - 15\ \text{dB HL} \geq 40\ \text{dB?}\ \ \text{Yes}$$

Similarly, the difference between the unmasked SRT in the right ear (65 dB HL) and the measured SRT in the left ear (20 dB HL) equals or exceeds our estimate of IA (40 dB). Although this approach can sometimes provide the audiologist with a more accurate estimate of the patient's IA for speech (which may be useful when selecting appropriate masking levels), it often just increases the number of steps needed to establish the true SRT in the test ear.

The audiogram presented in Figure 6.7 illustrates the need for contralateral masking during assessment of suprathreshold speech recognition. Puretone testing reveals normal hearing through 1,000 Hz, steeply sloping to a severe-to-profound sensory/neural hearing loss in the high frequencies bilaterally. SRTs were measured at 0 dB HL in both ears, a finding consistent with the puretone findings. Contralateral masking was not required during puretone and speech threshold audiometry. Suprathreshold speech recognition will be assessed using the California Consonant Test (CCT). This is a closed-set word recognition test that is sensitive to the speech recognition difficulties of individuals with high-frequency hearing loss (Owens and Schubert, 1977). If we use the recommended sensation level (SL) of 50 dB (Schwartz and Surr, 1979), then presentation level for both ears will be 50 dB HL (i.e., 50 dB relative to the SRT of 0 dB HL).

Let us now consider the need for contralateral masking during assessment of suprathreshold speech recognition. We will consider the need for masking using two types of air-conduction transducers: Supra-aural and 3A insert earphones. The advantage of insert earphones will become apparent.

Let us assume that supra-aural earphones are being used during speech audiometry. Contralateral masking will be required when assessing suprathreshold speech recognition in both ears because the difference between the presentation level of 50 dB HL in the test ear and the best puretone bone-conduction threshold of 0 dB HL in the nontest ear equals or exceeds 40 dB, our conservative estimate of IA for speech:

$$\text{Presentation Level}_{\text{Test Ear}} - \text{Best BC}_{\text{Nontest Ear}} \geq \text{IA}$$

Right Ear 50 dB HL – 0 dB HL ≥ 40 dB? Yes

Left Ear 50 dB HL – 0 dB HL ≥ 40 dB? Yes

A different outcome results if we substitute 3A insert earphones for the supra-aural arrangement. Because of the greater IA offered by 3A insert earphones, contralateral masking will not be required when assessing suprathreshold speech recognition in either ear. Specifically, the difference between the presentation level of 50 dB HL in the test ear and the best puretone bone-conduction threshold of 0 dB HL in the nontest ear does not equal or exceed 60 dB, our conservative estimate of IA for speech:

$$\text{Presentation Level}_{\text{Test Ear}} - \text{Best BC}_{\text{Nontest Ear}} \geq \text{IA}$$

Right Ear 50 dB HL – 0 dB HL ≥ 60 dB? No

Left Ear 50 dB HL – 0 dB HL ≥ 60 dB? No

The example presented in Figure 6.7 illustrates two important concepts related to assessment of suprathreshold speech recognition. First, it should not be assumed that contralateral masking is never required when assessing individuals with symmetrical sensory/neural hearing loss. Second, the need for contralateral masking often can be eliminated by using an air-conduction transducer that provides greater IA (i.e., 3A insert earphone).

MASKING CONCEPTS

Before proceeding to a discussion of clinical masking procedures, a brief review of basic masking concepts, including masking noise selection and calibration, will be presented. Generally, masking relates to how sensitivity for one sound is affected by the presence of another sound. ANSI/ASA (2010) defines masking as follows:

> The process by which the threshold of hearing for one sound is raised by the presence of another (masking) sound. The amount by which the threshold of hearing for one sound is raised by the presence of another (masking) sound, expressed in decibels (p 7).

Consider the following example. Absolute threshold for a 1,000-Hz puretone stimulus is initially determined to be 40 dB HL. Another sound, white noise, is now presented simultaneously to the same ear. Absolute threshold for the 1,000-Hz signal is redetermined in the presence of the white noise and increases to 60 dB HL. Sensitivity to the puretone signal has been affected by the presence of the white noise. This increase in threshold of one sound in the presence of another is defined as masking. Because the puretone threshold was raised by 20 dB (i.e., a threshold shift of 20 dB), the white noise has produced 20 dB of masking.

There are two basic masking paradigms: ipsilateral and contralateral. In an ipsilateral masking paradigm, the test signal and the masker are presented to the same ear. In a contralateral masking paradigm, the test signal and masker are presented to opposite ears. Masking is used clinically whenever it is suspected that the *nontest* ear is participating in the evaluation of the test ear. Consequently, masking is always applied to the nontest or contralateral ear. Masking reduces sensitivity of the nontest ear to the test signal. The purpose of contralateral masking, therefore, is to raise the threshold of the nontest ear sufficiently so that its contribution to a response from the test ear is eliminated.

Masking Noise Selection

Standard diagnostic audiometers provide three types of masking stimuli: narrowband noise, speech spectrum noise, and white noise. Our clinical goal is to select a masker that is efficient (Hood, 1960). An efficient masker is one that produces a given effective level of masking with the least overall sound pressure level.

To better understand this concept of masker efficiency, let us review the classic masking experiment conducted by Fletcher (1940). White noise is a broadband stimulus that contains equal energy across a broad range of frequencies. Because of its broadband spectrum, it has the ability to mask puretone stimuli across a broad range of frequencies (Hawkins and Stevens, 1950). Fletcher addressed which frequency components of broadband noise contribute to the masking of a tone.

Fletcher (1940) conducted what is known as a centered masking experiment. Initially, a very narrow band of noise was centered around a puretone signal. The bandwidth of the noise was progressively widened, and the masking effect on the puretone signal was determined. Fletcher observed that the masked puretone threshold increased as the bandwidth of the masking noise was increased. However, once the noise band reached and then exceeded a "critical bandwidth," additional masking of the puretone signal did not occur.

This concept of the critical band as first described by Fletcher (1940) consists of two components:

1. When masking a puretone with broadband noise, the only components of the noise that have a masking effect

on the tone are those frequencies included within a narrow band centered around the frequency of the tone.
2. When a puretone is just audible in the presence of the noise, the total noise power present in the narrow band of frequencies is equal to the power of the tone.

The first component of the critical band concept has clinical implications when selecting an appropriate masker during puretone audiometry. The second component has relevance when calibrating the effective masking (EM) level of the masking stimulus.

White noise is adequate as a masker for puretone stimuli. However, it contains noise components that do not contribute to the effectiveness of the masker. The additional noise components outside the tone's critical band simply add to the overall level (and loudness) of the masking stimulus. Therefore, the most efficient masker for puretone stimuli is a narrow band of noise with a bandwidth slightly greater than the critical band surrounding the tone. It provides the greatest masking effect with the least overall intensity. Sanders and Rintelmann (1964) confirmed that narrowband noise was a far more efficient masker for puretone stimuli than white noise. For a given sound pressure level (50, 70, and 90 dB SPL), narrowband noise centered at the frequency of the puretone signal (ranging from 250 to 4,000 Hz) consistently produced a greater masking effect (about 10 to 20 dB) than white noise.

The masking noise typically used during puretone audiometry, therefore, is narrowband noise centered geometrically around the audiometric test frequency. ANSI/ASA (2010) specifies the band limits (i.e., the upper and lower cutoff frequencies) of narrowband masking noise. To minimize the perception of tonality that often is associated with very narrow bands of noise, the bands specified by ANSI/ASA are somewhat wider than the critical bands for EM. The goal is to avoid confusion of the masker with the signal.

Speech spectrum noise (i.e., weighted random noise for the masking of speech) is typically used as a masker during speech audiometry. Speech is a broadband stimulus that requires a broadband masker. Although white noise is an adequate masker, it is not the most efficient. Speech spectrum noise is white noise that has been filtered to simulate the long-term average spectrum of speech. The average spectrum of speech contains the greatest energy in the low frequencies with spectrum level decreasing as a function of increasing frequency (Dunn and White, 1940). Speech spectrum noise has a more limited bandwidth than white noise. It is a more efficient masker than white noise, producing a masking advantage of 8 dB (Konkle and Berry, 1983). ANSI/ASA (2010) specifies that the spectrum of weighted random noise for the masking of speech should have a sound pressure spectrum level that is constant from 100 to 1,000 Hz, decreasing at a rate of 12 dB per octave from 1,000 to 6,000 Hz.

Calibration of Effective Masking Level

When a masking noise is presented to the nontest ear, we are interested in how much masking is produced. Consequently, masking noise is calibrated in EM level (dB EM).

ANSI/ASA (2010) defines EM level for puretones as "the sound pressure level of a band of noise whose geometric center frequency coincides with that of a specific pure tone that masks the pure tone to 50% probability of detection" (p 7). (Reference EM levels, calculated by adding an appropriate correction value to the reference equivalent threshold sound pressure level [RETSPL] at each frequency, are provided in the current ANSI/ASA specification for audiometers.) It is also indicated that, in individuals with normal hearing, "the amount of effective masking…is equal to the number of decibels that a given band of noise shifts a pure-tone threshold…when the band of noise and the pure tone are presented simultaneously to the same ear" (ANSI/ASA, 2010, p 7).

Stated differently, effective masking (in dB EM) refers to

1. The HL (dB HL) to which puretone threshold is shifted by a given level of noise; and
2. The puretone threshold shift (in dB) relative to 0 dB HL provided by a given level of noise.

Although contralateral masking is used clinically during hearing assessment, the following examples of ipsilateral masking will facilitate an understanding of the concept of EM level.

Example 1: A puretone air-conduction threshold is measured at 0 dB HL in the right ear. A narrowband noise geometrically centered at the test frequency is presented to the same ear at 50 dB EM. This EM level of 50 dB will shift puretone threshold to 50 dB HL.

Example 2: A puretone air-conduction threshold is measured at 30 dB HL in the right ear. A narrowband noise geometrically centered at the test frequency is presented to the same ear at 50 dB EM. This EM level of 50 dB will shift puretone threshold to 50 dB HL.

These examples illustrate two important points. First, a given level of EM will shift all unmasked puretone thresholds to the same dB HL. Of course, if unmasked puretone threshold is greater than a particular level of EM, then no threshold shift will occur. For example, a masker of 50 dB EM will not have a masking effect if the unmasked puretone threshold is 70 dB HL. Second, EM refers to the amount of threshold shift only relative to 0 dB HL.

Speech spectrum noise is also calibrated in EM level. Just as HL for speech (dB HL) is specified relative to the SRT, EM level is also referenced to the SRT. Specifically, EM for speech refers to the dB HL to which the SRT is shifted by a given level of noise. ANSI/ASA (2010) defines EM level for speech as the "sound pressure level of a specified masking noise that masks a speech signal to 50% probability of

recognition" (p 8). (If the speech spectrum noise has spectral density characteristics as specified by ANSI/ASA and if the sound pressure level of the masker is equal to the RETSPL for speech, then the masker is calibrated in dB EM.) ANSI/ASA (2010) also states that in individuals with normal hearing, "the amount of effective masking…is equal to the number of decibels that a masking noise shifts a speech recognition threshold…when the masking noise and speech signal are presented simultaneously to the same ear" (p 8).

Consider the following example. SRT is measured at 0 dB HL. Speech spectrum noise is then presented to the same ear at 50 dB EM. This EM level of 50 dB will shift the SRT to 50 dB HL.

Calibration of masking noise in EM level greatly simplifies clinical masking procedures. When masking noise is calibrated in dB EM, then the decibel value indicated on the masking level control will indicate the masking effect produced in the nontest ear. This clearly facilitates the selection of appropriate masking levels during clinical testing.

CLINICAL MASKING PROCEDURES

All approaches to clinical masking address two basic questions. First, what is the minimum level of noise that is needed in the nontest ear to eliminate its response to the test signal? Stated differently, this is the *minimum masking level* that is needed to avoid *undermasking* (i.e., even with contralateral masking, the test signal continues to be perceived in the nontest ear). Second, what is the maximum level of noise that can be used in the nontest ear that will not change the true threshold or response in the test ear? Stated differently, this is the *maximum masking level* that can be used without *overmasking* (i.e., with contralateral masking, the true threshold or response in the test ear has been changed). Because of limited IA for air-conducted sound, the masking stimulus presented to the nontest ear can also cross over to the test ear and produce masking of the test signal (i.e., overmasking). Stated simply, the purpose of clinical masking is to make the test signal inaudible in the nontest ear without affecting the true response to the signal in the test ear. Therefore, the major goal of any clinical masking procedure is the avoidance of both undermasking and overmasking.

Studebaker (1979) has identified two major approaches to clinical masking: psychoacoustic and acoustic. Psychoacoustic procedures are "those based upon observed shifts in the measured threshold as a function of suprathreshold masker effective levels in the nontest ear" (Studebaker, 1979, p 82). These approaches are also identified as threshold shift or shadowing procedures. Acoustic procedures are "those based upon calculating the approximate acoustic levels of the test and masker signals in the two ears under any given set of conditions and on this basis deriving the required masking level" (Studebaker, 1979, p 82). These procedures are also referred to as calculation or formula methods. Psychoacoustic approaches are considered appropriate for threshold measurements, whereas acoustic methods are typically most efficient for suprathreshold measurements.

Puretone Audiometry

Formulas and equations have been presented for the calculation of minimum and maximum masking levels during puretone audiometry (Lidén et al., 1959; Martin, 1967, 1974; Studebaker, 1962, 1964). A brief discussion of these formulas will facilitate an understanding of the manner in which appropriate levels of masking are selected during puretone threshold testing.

Minimum Masking Level

Lidén et al. (1959) and Studebaker (1964) offered formulas for calculating minimum masking level during puretone air-conduction audiometry that include consideration of IA, HL of the test signal, and air-bone gaps in the nontest ear. Although this "formula" approach to calculating minimum masking level is necessary during administration of suprathreshold auditory tests (this approach will be discussed later in the section addressing masking in speech audiometry), it proves somewhat disadvantageous during threshold audiometry. First, it can be time consuming. Second, the clinician may not have all required information to accurately calculate minimum masking level at that point in time. (The reader is referred to Yacullo, 1996 for further discussion of the derivation of these equations and formulas.)

The simplified method described by Martin (1967, 1974) is recommended for clinical use. Martin has suggested that formulas are unnecessary during threshold audiometry and has simplified the calculation of minimum masking level. Specifically, the "initial" masking level (in dB EM) during air-conduction threshold testing is simply equal to air-conduction threshold (in dB HL) of the nontest ear (i.e., $AC_{Nontest\ Ear}$). It should be noted that the initial masking level is calculated in the same manner regardless of the air-conduction transducer being used (i.e., supra-aural earphone or 3A insert earphone).

The audiometric data presented in Figure 6.8 will be used to facilitate an understanding of the calculation of masking levels during puretone threshold audiometry. Audiometry was performed using supra-aural earphones. Unmasked air- and bone-conduction thresholds at 500 Hz are provided; masked air- and bone-conduction thresholds are also included for later discussion. Unmasked puretone air-conduction testing suggests that contralateral masking will be required only when measuring air-conduction threshold in the left ear. Specifically, the unmasked air-conduction threshold of 65 dB HL in the left ear equals or exceeds the threshold (both air and bone conduction) in the nontest ear by a conservative estimate of IA (i.e., 40 dB).

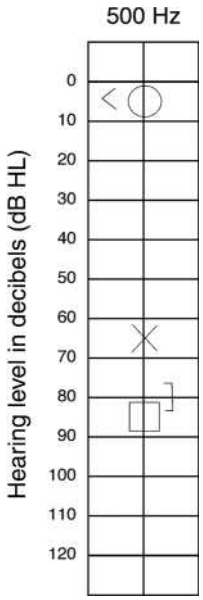


FIGURE 6.8 An example illustrating the calculation of initial and maximum masking levels during puretone threshold audiometry. See text for further discussion.

According to Martin (1967, 1974), the initial masking level (in dB EM) is equal to 5 dB EM (i.e., $AC_{Nontest\ Ear}$).

Martin (1967, 1974) explains the derivation of this simplified equation in the following way. A signal detected at threshold is assumed to have an SL of 0 dB, regardless of whether it is perceived in the test or nontest ear. Therefore, a cross-hearing response during puretone threshold testing theoretically represents a *threshold* response in the nontest ear. Given this assumption, the initial masking level required is one that will just mask a signal of 0 dB SL (i.e., threshold) in the nontest ear. Because of the manner in which masking stimuli are calibrated clinically (i.e., EM level, dB EM), a masker presented at a level (in dB EM) equal to the air-conduction threshold (in dB HL) in the nontest ear should just mask the threshold response in the nontest ear. Given the example presented in Figure 6.8, a masker level of 5 dB EM (which is equal to the air-conduction threshold in the right ear) should be sufficient to just mask a threshold response to the test signal in the right ear. Martin also indicates that the simplified approach will lead to the selection of the same masker level as when using the more complex formulas for calculating minimum masking level.

Martin (1974) recommends that approximately 10 dB should be added to the initial masking level to account for intersubject variability. Remember that dB EM refers to the HL (dB HL) to which threshold is shifted by a given level of noise. Calibration of EM is based on the averaged responses of a group of normal-hearing subjects. Therefore, a given EM level will not prove equally effective for all subjects. If masked thresholds are normally distributed around the average effective level and if the standard deviation of the distribution is about 5 dB, then Studebaker (1979) recommends that a safety factor of not less than 10 dB should be added to the calculated minimum masking level. Given this recommendation, Martin's simplified equation for initial masking level (in dB EM) during air-conduction threshold audiometry can be stated as follows:

$$\text{Initial Masking Level} = AC_{Nontest\ Ear} + 10\ \text{dB}$$

Considering again the example presented in Figure 6.8, the initial masking level is now calculated as 15 dB EM:

$$\begin{aligned}\text{Initial Masking Level} &= AC_{Nontest\ Ear} + 10\ \text{dB}\\ &= 5\ \text{dB HL} + 10\ \text{dB}\\ &= 15\ \text{dB EM}\end{aligned}$$

It is important to differentiate the terms *minimum masking level* and *initial masking level* during air-conduction threshold audiometry. Earlier in this discussion, a general definition of minimum masking level was provided. Minimum masking level was defined as the minimum level of noise needed in the nontest ear to eliminate its response to the test signal. Related to puretone threshold audiometry, a more specific definition of minimum masking level can be offered: Minimum masking level is the minimum level of noise needed to eliminate the contribution of the nontest ear *to establish the true or correct threshold in the test ear.* Initial masking level is simply the first level of noise introduced to the nontest ear. This initial level of masking is often not sufficient to establish the threshold in the test ear; higher levels of masking are often required. This concept will be addressed again in our discussion of the recommended clinical masking procedure during puretone threshold audiometry.

Lidén et al. (1959) and Studebaker (1964) also have offered formulas for minimum masking level during bone-conduction testing that are derived from the same theoretical constructs used during air-conduction testing (see Yacullo, 1996 for further discussion). Again, the formula approach during bone-conduction threshold audiometry is not clinically practical. The use of Martin's simplified approach is recommended. Specifically, initial masking level during bone-conduction audiometry is equal to the air-conduction threshold of the nontest ear. However, we will need to add the OE to the initial masking level to compensate for covering (i.e., occluding) the nontest ear with an earphone (Martin, 1967, 1974; Studebaker, 1964). Martin's simplified equation for initial masking level (in dB EM) during bone-conduction threshold testing can be stated as follows:

$$\text{Initial Masking Level} = AC_{Nontest\ Ear} + \text{OE} + 10\ \text{dB}$$

Bone-conduction thresholds are always obtained with the test ear unoccluded or uncovered. However, when an earphone covers or occludes the nontest ear during masked

bone-conduction audiometry, an OE can be created in the nontest ear. The nontest ear consequently can become more sensitive to bone-conducted sound for test frequencies below 2,000 Hz, particularly when using supra-aural earphones (Berger and Kerivan, 1983; Berrett, 1973; Dean and Martin, 2000; Dirks and Swindeman, 1967; Elpern and Naunton, 1963; Goldstein and Hayes, 1965; Hodgson and Tillman, 1966). During the application of contralateral masking, there is increased probability that the nontest ear will respond when obtaining a masked bone-conduction threshold in the test ear. Studebaker (1979) points out that the OE does not actually affect the hearing sensitivity of the occluded ear, but rather increases the sound pressure level of the signal reaching the cochlea. The reader is referred to Tonndorf (1968, 1972) for further discussion of the contribution of the external auditory meatus to bone-conduction thresholds.

There is evidence suggesting that the OE is decreased significantly when using deeply inserted insert earphones (Berger and Kerivan, 1983; Chaiklin, 1967; Dean and Martin, 2000). Berger and Kerivan (1983) and Dean and Martin (2000) studied the magnitude of the OE in normal-hearing subjects using E-A-R foam eartips and supra-aural earphones as occluding devices. Their overall results are remarkably similar. First, the average OEs in the low frequencies are greatly reduced when occluding the ear using an E-A-R foam eartip with deep insertion. Second, the advantage of a greatly reduced OE for the E-A-R foam eartip is lost when a partial or shallow insertion is used. Third, partial or shallow insertion of an E-A-R foam eartip yields average OEs that are similar to those measured with supra-aural earphones. Different theories have been offered to explain the reduced OE for an occluding device that is deeply inserted into the ear canal. The reader is referred to Berger and Kerivan (1983), Tonndorf (1972), and Yacullo (1996) for further discussion.

The clinician can use either individually determined (Dean and Martin, 2000; Martin et al., 1974) or fixed OE values (i.e., based on average data reported in the literature) when calculating initial masking level. Based on the largest average OEs reported in the literature (Berger and Kerivan, 1983; Berrett, 1973; Dean and Martin, 2000; Dirks and Swindeman, 1967; Elpern and Naunton, 1963; Goldstein and Hayes, 1965; Hodgson and Tillman, 1966), the following values are recommended for clinical use.

When using supra-aural earphones, the following fixed OE values are recommended: 30 dB at 250 Hz, 20 dB at 500 Hz, and 10 dB at 1,000 Hz. When using 3A insert earphones with deeply inserted foam eartips, the following values are recommended: 10 dB at 250 and 500 Hz and 0 dB at frequencies of 1,000 Hz or higher.

It should be noted that the OE is decreased or absent in ears with conductive hearing impairment (Martin et al., 1974; Studebaker, 1979). If the nontest ear exhibits a potential air-bone gap of 20 dB or more, then the OE should not be added to the initial masking level at that frequency.

Consider again the example presented in Figure 6.8. Assume that we have subsequently measured a masked air-conduction threshold of 85 dB HL in the left ear. A masked bone-conduction threshold will also be required in the left ear. Comparison of the unmasked bone-conduction threshold of 5 dB HL with the masked air-conduction threshold of 85 dB HL in the left ear suggests a potentially significant air-bone gap (i.e., ≥15 dB). Initial masking level is calculated in the same manner regardless of the air-conduction transducer used for the delivery of the masking stimulus. The only difference in calculation relates to applying a different correction factor for the OE when testing in the lower frequencies. Using the recommended fixed OE values for supra-aural earphones, initial masking level during bone-conduction testing at 500 Hz is calculated as follows:

$$\begin{aligned}\text{Initial Masking Level} &= \text{AC}_{\text{Nontest Ear}} + \text{OE} + 10\ \text{dB} \\ &= 5\ \text{dB HL} + 20\ \text{dB} + 10\ \text{dB} \\ &= 35\ \text{dB EM}\end{aligned}$$

In this particular example, it is appropriate to account for the OE because there is no evidence of a significant air-bone gap in the nontest (i.e., right) ear. The use of a supra-aural earphone for delivery of masking in the lower frequencies, however, will result in greater initial masking levels than when using a 3A insert because of a larger OE correction factor.

Maximum Masking Level

Maximum masking level refers to the maximum level of noise that can be used in the nontest ear that will not shift or change the true threshold in the test ear. Two factors influence maximum masking level during puretone audiometry: (1) The bone-conduction threshold of the test ear ($\text{BC}_{\text{Test Ear}}$) and (2) IA of the air-conducted masking stimulus (Lidén et al., 1959). Maximum masking level (M_{Max}), based on the original concept described by Lidén et al., can be summarized using the following equation:

$$M_{\text{Max}} = \text{BC}_{\text{Test Ear}} + \text{IA} - 5\,\text{dB}$$

If $\text{BC}_{\text{Test Ear}}$ + IA is just sufficient to produce overmasking, then clinically, we want to use a masking level that is somewhat less than the calculated value. Consequently, 5 dB is subtracted from the level that theoretically produces overmasking. Because we are concerned about an undesired masking effect in the test ear, bone-conduction sensitivity in that ear must be considered. As a result, overmasking is more of a potential problem when bone-conduction sensitivity is very good in the test ear. Overmasking, on the other hand, is generally not an issue when bone-conduction hearing sensitivity is poor. The poorer the bone-conduction hearing sensitivity is in the test ear, the greater the levels of masking that can be used without overmasking.

The following two points are important to remember. First, the equation for maximum masking level is the same for both air- and bone-conduction audiometry. Masking noise is always delivered through an air-conduction transducer (e.g., insert or supra-aural earphone) regardless of the transducer used for measuring puretone threshold (i.e., air- or bone-conduction transducer). Second, maximum masking level is generally higher when using 3A insert earphones because of increased IA, particularly in the lower frequencies.

Consider again the example presented in Figure 6.8. We will now calculate the maximum masking level that can be used during both masked air- and bone-conduction audiometry:

$$\begin{aligned} M_{\text{Max}} &= \text{BC}_{\text{Test Ear}} + \text{IA} - 5 \text{ dB} \\ &= 80 \text{ dB HL} + 60 \text{ dB} - 5 \text{ dB} \\ &= 135 \text{ dB EM} \end{aligned}$$

Rather than using the very conservative IA estimate of 40 dB when using supra-aural earphones, in this case, we will use the more accurate estimate of 60 dB. If the bone-conduction threshold in the right (i.e., nontest) ear is assumed to be 5 dB HL (i.e., the unmasked bone-conduction threshold) and the unmasked air-conduction threshold in the left ear is 65 dB HL, then there is evidence that IA is at least 60 dB. If 140 dB EM is just sufficient to produce overmasking (i.e., $\text{BC}_{\text{Test Ear}}$ + IA), then 135 dB EM is the maximum level of noise that can be used in the nontest ear that will not shift or change the true threshold in the test ear. It should be noted that 135 dB EM is a level that significantly exceeds the output limits for standard audiometers.

Generally, it is neither time efficient nor necessary to calculate maximum masking level during puretone threshold audiometry, particularly when using psychoacoustic or threshold shift masking procedures (which will be described shortly). In addition, the estimated maximum masking level is typically very conservative and not an accurate indication of the true maximum. In the above example, we calculated a relatively accurate estimate by using a more accurate value of IA (rather than the conservative value) and the actual bone-conduction threshold in the test ear (i.e., 80 dB HL). However, the true bone-conduction threshold (obtained with appropriate contralateral masking) in the test ear is typically not known when maximum masking level is estimated during both air- and bone-conduction threshold audiometry. Because only an unmasked bone-conduction threshold is available at the time that masking levels are determined, we are required to use the unmasked threshold as the estimate of bone-conduction hearing sensitivity in the test ear. Let us calculate again M_{Max} using the unmasked bone-conduction response as the estimate of bone-conduction threshold:

$$\begin{aligned} M_{\text{Max}} &= 5 \text{ dB HL} + 60 \text{ dB} - 5 \text{ dB} \\ &= 60 \text{ dB EM} \end{aligned}$$

Clearly in this case, our calculation based on the unmasked bone-conduction threshold (i.e., 60 dB EM) is an underestimate of the actual maximum level (i.e., 135 dB EM).

Whenever an unmasked bone-conduction threshold is used during determination of maximum masking, the resultant value is typically smaller than the masking level that will actually result in overmasking. Although the actual calculation of maximum masking level during puretone threshold audiometry is often of limited use, consideration of the maximum level of noise that can be used in the nontest ear can alert the audiologist to the possibility of overmasking, particularly in cases of conductive hearing loss when bone-conduction hearing sensitivity is very good.

Recommended Clinical Procedure

The most popular method for measuring masked puretone thresholds was first described by Hood in 1957 (Hood, 1960). The Hood method, also referred to as the plateau, threshold shift, or shadowing procedure, is a psychoacoustic technique that relies on observations about the relationship between masker level in the nontest ear and measured threshold in the test ear. Hood originally described a masking procedure that was applicable for measurement of masked bone-conduction thresholds. However, it proves equally effective for measurement of air-conduction thresholds as well.

The example presented in Figure 6.9 will help facilitate an understanding of the underlying concept of the threshold shift procedure. Unmasked puretone air-conduction thresholds, obtained using supra-aural earphones, were measured at 10 dB HL in the right ear and 60 dB HL in the left ear (Figure 6.9A). Contralateral masking will be required when testing the left ear because there is a difference between the test and nontest ears that equals or exceeds a conservative estimate of IA (i.e., 40 dB). An initial masking level of 20 dB EM (i.e., $\text{AC}_{\text{Nontest Ear}}$ + 10 dB) is now presented to the right ear, and puretone threshold is re-established. Recall that the purpose of contralateral masking is to raise the threshold of the nontest ear sufficiently to eliminate its contribution when measuring a response in the test ear. Assuming that overmasking is not occurring, then contralateral masking should have an effect only on the responsiveness of the nontest ear.

There are two possible outcomes when puretone threshold is re-established in the presence of contralateral masking: (1) No measured puretone threshold shift (e.g., puretone threshold remains constant at 60 dB HL; Figure 6.9B) or (2) a measured puretone threshold shift (e.g., puretone threshold shifts from 60 to 70 dB HL; Figure 6.9C). If contralateral masking in the nontest ear does not produce a masking effect, then it is concluded that the unmasked puretone threshold represents a response from the test ear. Conversely, if contralateral masking in the nontest ear does produce a

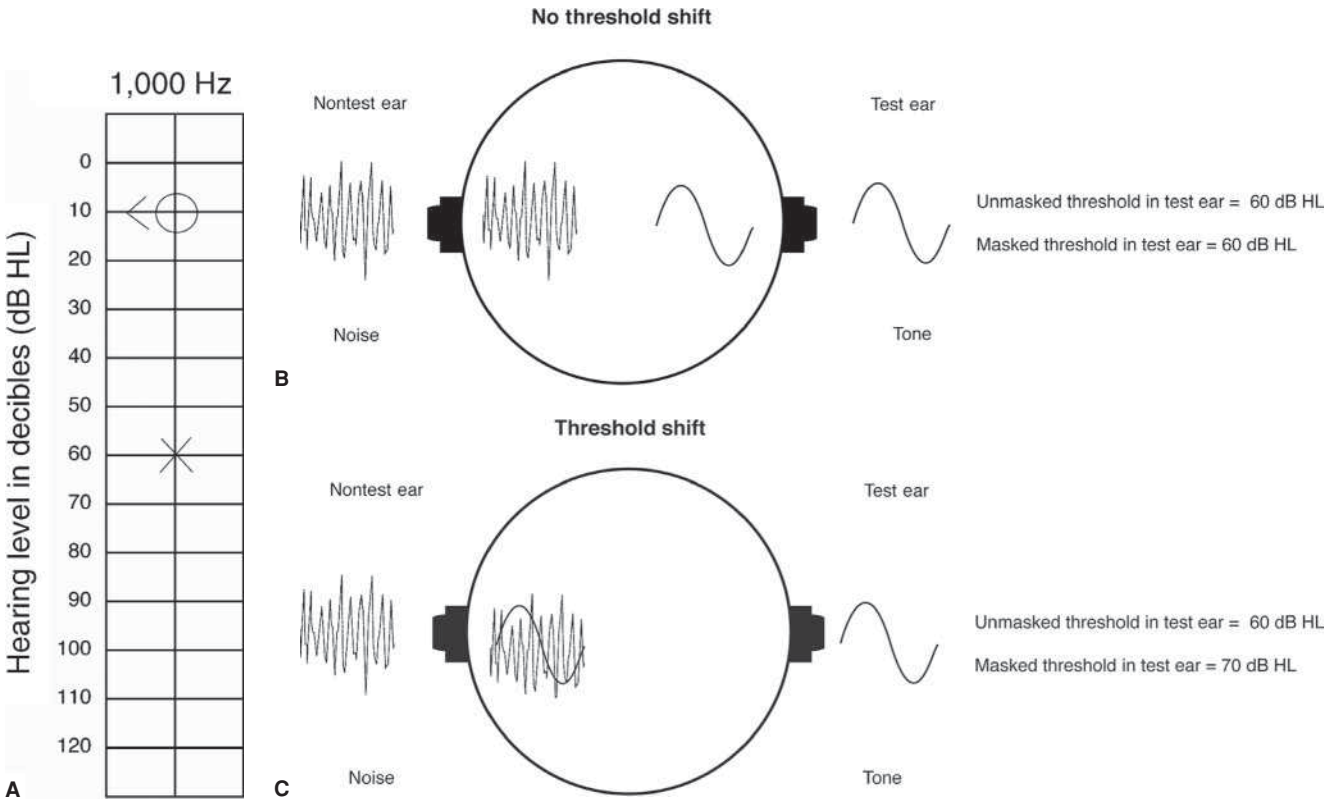


FIGURE 6.9 Example illustrating the underlying concept of the plateau or threshold shift masking procedure. See text for further discussion. [From Yacullo WS. [1996] *Clinical Masking Procedures*. 1st ed. Boston, MA: Allyn & Bacon, © 1996, p 69. Adapted by permission of Pearson Education, Inc., Upper Saddle River, NJ.]

masking effect, then it is concluded that the unmasked puretone threshold represents a response from the nontest ear. The underlying concept of the Hood procedure is that the introduction of masking to the nontest ear will produce a masking effect (i.e., a threshold shift) only if the nontest ear is contributing to the observed response. Decisions about which ear is contributing to the measured threshold are based on whether a threshold shift occurs when masking is introduced to the nontest ear.

Hood (1960) outlined two essential steps of the plateau masking procedure: (1) Demonstration of the shadowing effect and (2) identification of the changeover point. The hypothetical example presented in Figure 6.10 illustrates basic concepts of the plateau masking procedure. Puretone testing using supra-aural earphones reveals unmasked air-conduction thresholds of 0 dB HL in the right ear and 40 dB HL in the left ear (Figure 6.10A). Unmasked bone-conduction threshold is 0 dB HL. Because there is a 40-dB difference between ears, contralateral masking will be required when measuring air-conduction threshold in the left ear. (Masked air- and bone-conduction thresholds in the left ear are included for later discussion.)

The masking function presented in Figure 6.10B shows the relationship between measured puretone threshold (in dB HL) in the test ear and EM level (in dB EM) in the nontest ear. Masking noise is introduced at an initial masking level of 10 dB EM (i.e., $AC_{Nontest\ Ear}$ + 10 dB), and puretone threshold is re-established. Threshold shifts to 50 dB HL. When the masker level is raised sequentially to 20 and 30 dB EM, puretone threshold continues to shift by 10 dB. A shadowing effect has occurred because the masked puretone threshold "shadows" the threshold of the nontest or masked ear with each increment in EM level. Because a threshold shift occurs when masking level is raised, it is concluded that the masking noise and the tone are restricted to the nontest ear.

When the masker is raised from 30 to 100 dB EM, puretone threshold no longer shifts and remains stable at 70 dB HL. A plateau has been reached. Because there is no additional masking effect (i.e., a threshold shift) when masker level is increased, it is concluded that the nontest ear is no longer contributing to the observed response. Puretone threshold of the test ear (i.e., 70 dB HL) has been reached. Hood (1960) refers to the initial point on the masking function where puretone threshold remains stable with increasing masking level as the "changeover point." In this example, the changeover point of 30 dB EM also corresponds to minimum masking level, the minimum amount of noise required to establish the true threshold in the test ear. Masker levels that result in no threshold shift (i.e., the plateau) represent adequate masking (i.e., 30 through 100 dB EM). Masker

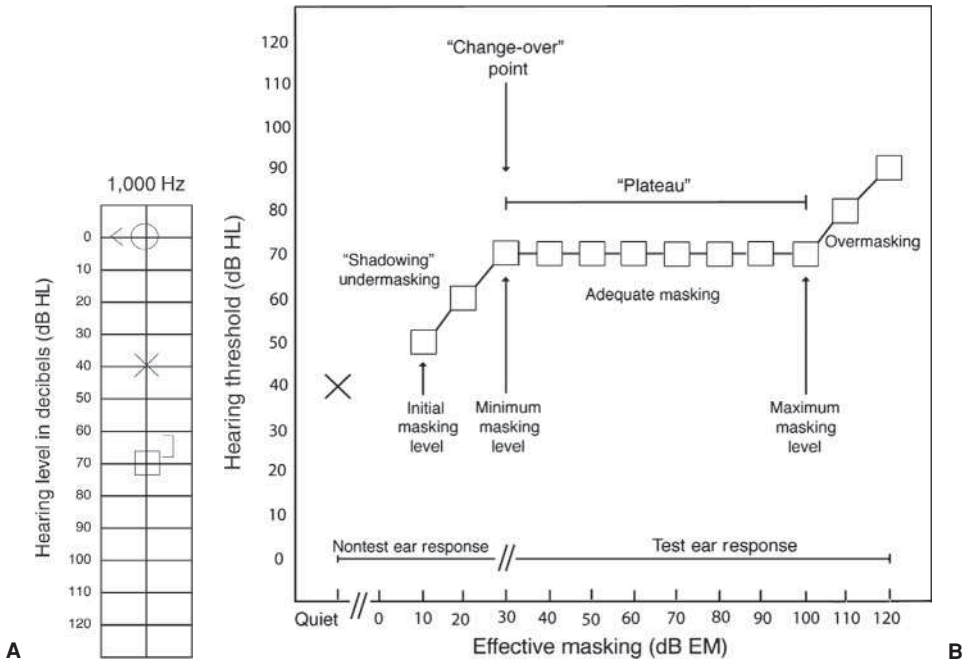


FIGURE 6.10 Hypothetical example illustrating the concepts of undermasking, adequate masking, and overmasking using the threshold shift or plateau masking procedure. See text for explanation. [From Yacullo WS. [1996] *Clinical Masking Procedures*. 1st ed. Boston, MA: Allyn & Bacon, © 1996, p 72. Adapted by permission of Pearson Education, Inc., Upper Saddle River, NJ.]

levels less than 30 dB EM represent undermasking. That is, there is insufficient masking to establish the true puretone threshold in the test ear.

When the masker level exceeds 100 dB EM (i.e., 110 and 120 dB EM), however, a puretone threshold shift with each increment in masking level is again observed. Overmasking is now occurring. The masking noise has reached the test ear through cross hearing, and a masking effect (i.e., a threshold shift) is observed in the test ear. Assuming that a masked bone-conduction threshold is measured subsequently in the left ear at 65 dB HL, then an estimate of maximum masking level is 100 dB EM ($BC_{Test\ Ear}$ + IA − 5 dB: 65 dB HL + 40 dB − 5 dB). Whereas the plateau and overmasking portions of the masking function represent responses from the test ear, the undermasking or shadowing portion represents responses from the nontest ear. It should be apparent from the masking function in Figure 6.10 that the width of the masking plateau is defined by the minimum and maximum masking levels.

The clinical goal of the plateau procedure is to establish the HL at which puretone threshold remains unchanged with increments in masking level. Two important variables that relate to the plateau procedure are (1) the magnitude of the masker increment and (2) the number of masker increments needed to establish a masking plateau. Although Hood (1960) originally recommended that masker level be changed in increments of 10 dB, others have suggested that the level should be a 5-dB step size (Martin, 1980; Silman and Silverman, 1991). Martin (1980) suggests that accuracy is increased somewhat by using a masker increment of 5 dB. It is somewhat arbitrary whether a 5- or 10-dB step size is used for increasing masker level. Either step size is acceptable. However, the smaller step size of 5 dB is strongly recommended whenever the masking plateau is narrow and there is increased risk of overmasking (i.e., cases of bilateral conductive hearing loss).

Hood (1960) did not specify the number of masker increments needed to establish a masking plateau. Clinically, it is neither time efficient nor necessary to measure the entire masking plateau. It is generally agreed that a masking "plateau" has been established when masker level can be increased over a range of at least 15 to 20 dB without shifting puretone threshold (Kaplan et al., 1993; Martin, 1980; Sanders, 1991; Silman and Silverman, 1991).

The recommended clinical procedure (Yacullo, 1996, 2004), based on the major components of Hood's shadowing technique, is summarized as follows:

1. Masking noise is introduced to the nontest ear at the initial masking level. Puretone threshold is then re-established.
2. Level of the tone or noise is increased subsequently by 5 dB. If there is a response to the tone in the presence of the noise, the level of the noise is increased by 5 dB. If there is no response to the tone in the presence of the noise, the level of the tone is increased in 5-dB steps until a response is obtained.

3. A plateau has been reached when the level of the noise can be increased over a range of 15 to 20 dB without shifting the threshold of the tone. This corresponds to a response to the tone at the same HL when the masker is increased in three to four consecutive levels.
4. Masked puretone threshold corresponds to the HL of the tone at which a masking plateau has been established.

If a 10-dB step size is used for increasing masking level, then the plateau corresponds to a range of 20 dB (i.e., a response to the tone at the same HL when the masker is increased in two consecutive levels).

The recommended procedure for establishing a masking plateau does not require that puretone threshold be formally established each time that the masking level is increased. This approach would significantly increase the time required to establish a masking plateau. Rather, the tone is presented once at the same HL as the previous response. If no response occurs, the tone is increased in 5-dB steps until audibility is achieved. However, the HL of the tone may be increased inappropriately because of a decision-making process based on a single response. This may lead to imprecision when measuring the masked threshold. Therefore, it is recommended that masked puretone threshold be re-established using a standardized threshold procedure (e.g., ASHA, 2005) in the presence of the final level of masking noise that resulted in a plateau. This sometimes leads to a 5-dB improvement in the masked puretone threshold. However, the decision to re-establish masked puretone threshold at the end of the plateau procedure will be influenced by time considerations.

Remember that the goal of the plateau procedure is to establish the HL at which puretone threshold remains unchanged with increments in masking level. Given this goal, there are three major outcomes that can result when measuring puretone threshold. These outcomes are illustrated in the three examples presented in Figure 6.11. In

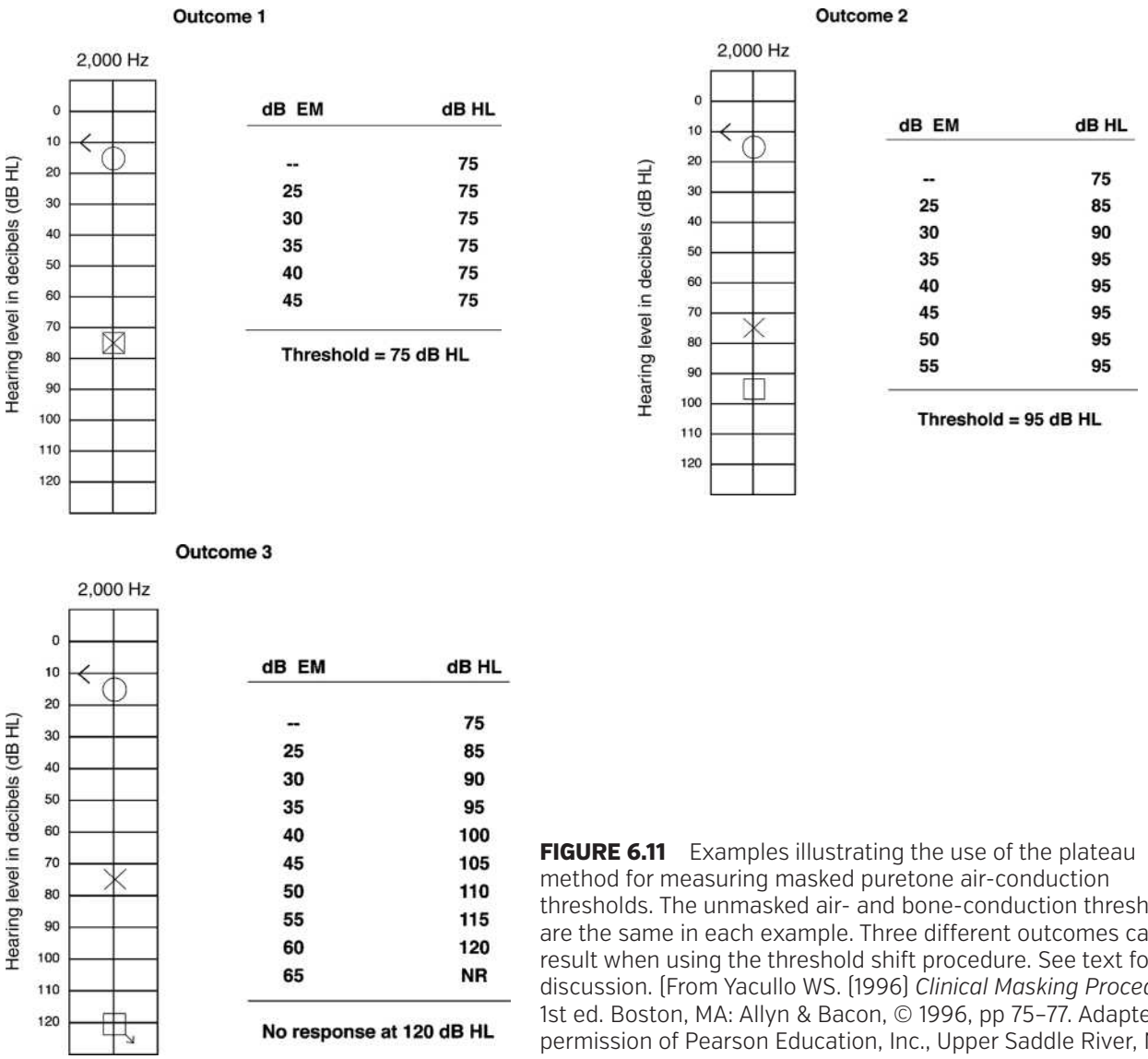


FIGURE 6.11 Examples illustrating the use of the plateau method for measuring masked puretone air-conduction thresholds. The unmasked air- and bone-conduction thresholds are the same in each example. Three different outcomes can result when using the threshold shift procedure. See text for discussion. [From Yacullo WS. (1996) *Clinical Masking Procedures*. 1st ed. Boston, MA: Allyn & Bacon, © 1996, pp 75–77. Adapted by permission of Pearson Education, Inc., Upper Saddle River, NJ.]

each example, the unmasked puretone thresholds at 2,000 Hz are the same. Unmasked puretone air-conduction thresholds, obtained using 3A insert earphones, were measured at 15 dB HL in the right ear and 75 dB HL in the left ear. Contralateral masking will be required when measuring air-conduction threshold in the left ear; an initial masking level of 25 dB EM is presented to the right ear, and puretone threshold is re-established.

In the first outcome, the unmasked puretone threshold of 75 dB HL remains unaffected with increasing masking level. The level of the noise was increased over a range of 20 dB without shifting the threshold of the tone. In this example, the initial masking level occurs at the masking plateau. Contralateral masking has confirmed that the unmasked puretone threshold represents a response from the test ear.

In the second outcome, the initial masking level produces a puretone threshold shift. A masking plateau is reached, however, when masking level is increased from 35 to 55 dB EM (i.e., a masking range of 20 dB). Because masked puretone threshold remains stable at 95 dB HL with increasing masking level, puretone threshold is recorded as 95 dB HL. Contralateral masking has confirmed that the unmasked puretone threshold represents a cross-hearing response from the nontest ear.

In the third outcome, the initial masking level again produces a puretone threshold shift. However, puretone threshold continues to shift to the output limits of the audiometer with increasing masking level. A plateau is not obtained. Therefore, it is concluded that there is no measurable hearing in the left ear. This conclusion is correct assuming that overmasking has not occurred.

Turner (2004a, 2004b) has described a masking method that can replace the plateau procedure in some masking situations. A disadvantage of the plateau method is that it can be time consuming. The "optimized" masking method described by Turner, which is based on the principles of the masking plateau, can reduce the number of masking levels required to reach the plateau and establish threshold. The method is optimized because it uses the maximum possible masking levels without overmasking. This is accomplished through the use of higher initial masking levels and maximum masker increments. However, there are some masking situations where the optimized approach is not appropriate. The reader is referred to the two articles by Turner (2004a, 2004b) for further discussion.

The Masking Dilemma

There are clinical situations where minimum masking levels can result in overmasking. Studebaker (1979) states that a "masking dilemma" results when the width of the masking plateau is very narrow or nonexistent. Remember that the width of the masking plateau is defined by minimum and maximum masking levels. Generally, a masking dilemma results whenever there is a significant hearing loss in the nontest ear and a conductive hearing loss in the test ear. The presence of significant hearing loss in the nontest ear requires higher initial masking levels; the presence of a conductive hearing loss in the test ear (i.e., normal bone-conduction hearing sensitivity) decreases the maximum masking level. The consequence of a reduced or nonexistent masking plateau is the inability to establish correct masked thresholds in the test ear.

The classic example of a masking dilemma is demonstrated with a bilateral, mild-to-moderate conductive hearing loss. The possibility for overmasking exists when measuring masked air- and bone-conduction thresholds in both ears. Naunton (1960) states that, in some cases of bilateral conductive hearing loss, it is not possible to mask the nontest ear without simultaneously producing a masking effect in the test ear.

One solution to the masking dilemma is the use of insert earphones (Coles and Priede, 1970; Hosford-Dunn et al., 1986; Studebaker, 1962, 1964). Recall that the use of 3A insert earphones significantly increases IA for air-conducted sound, particularly in the lower frequencies (Killion et al., 1985; Sklare and Denenberg, 1987). There are two advantages of using insert earphones in cases of bilateral conductive hearing loss. First, the need for masking during measurement of air-conduction thresholds is often eliminated because of greater IA for air-conducted sound. Second, the use of insert earphones reduces the probability of overmasking in cases where contralateral masking is required. The use of an air-conduction transducer with increased IA increases the range between the minimum and maximum masking levels, thereby increasing the width of the masking plateau and the range of permissible masking levels (Studebaker, 1962).

Central Masking

The introduction of contralateral masking can produce a small threshold shift in the test ear even when the masking level is insufficient to produce overmasking. Wegel and Lane (1924) referred to this phenomenon as central masking. It has been hypothesized that threshold shifts in the presence of low levels of masking are mediated through central nervous system processes (Lidén et al., 1959). Central masking has been reported to influence thresholds measured during both puretone and speech audiometry (Dirks and Malmquist, 1964; Lidén et al., 1959; Martin, 1966; Martin and DiGiovanni, 1979; Martin et al., 1965; Studebaker, 1962). Although the threshold shift produced by central masking is generally considered to be approximately 5 dB (Konkle and Berry, 1983; Martin, 1966), variable results have been reported across subjects and studies. There is also some indication that central masking effects increase with increasing masking level (Dirks and Malmquist, 1964; Martin et al., 1965; Studebaker, 1962).

There is currently no agreed upon procedure that accounts for central masking effects during threshold audiometry. However, it is generally not recommended that the effect of central masking be subtracted from masked thresholds. First, it is difficult to determine an appropriate correction factor given the variability of the central masking effect across subjects. Second, the typical central masking effect size of about 5 dB is considered to be within good test-retest reliability during threshold measurements. It is important to remember that the use of contralateral masking can somewhat influence the measured masked thresholds and should be taken into account when interpreting audiometric test results. For example, a difference of 5 dB between unmasked and masked thresholds is generally not considered significant. This difference may simply reflect (1) central masking effects and/or (2) normal variability related to test-retest reliability.

Masked Audiogram Interpretation

Unmasked and masked puretone thresholds are typically recorded on the same audiogram. Therefore, audiogram interpretation will involve consideration of both masked and unmasked responses. ASHA (1990) has published guidelines for audiometric symbols and procedures for graphic representation of frequency-specific audiometric findings. These guidelines have been followed throughout this chapter.

Figure 6.12 presents an audiogram in which contralateral masking was required when obtaining both air- and bone-conduction thresholds in the left ear. Air-conduction audiometry was performed using supra-aural earphones. Puretone testing reveals a mild conductive hearing loss of flat configuration in the right ear. Masked air- and bone-conduction responses indicate a severe-to-profound, sensory/neural hearing loss of gradually sloping configuration in the right ear.

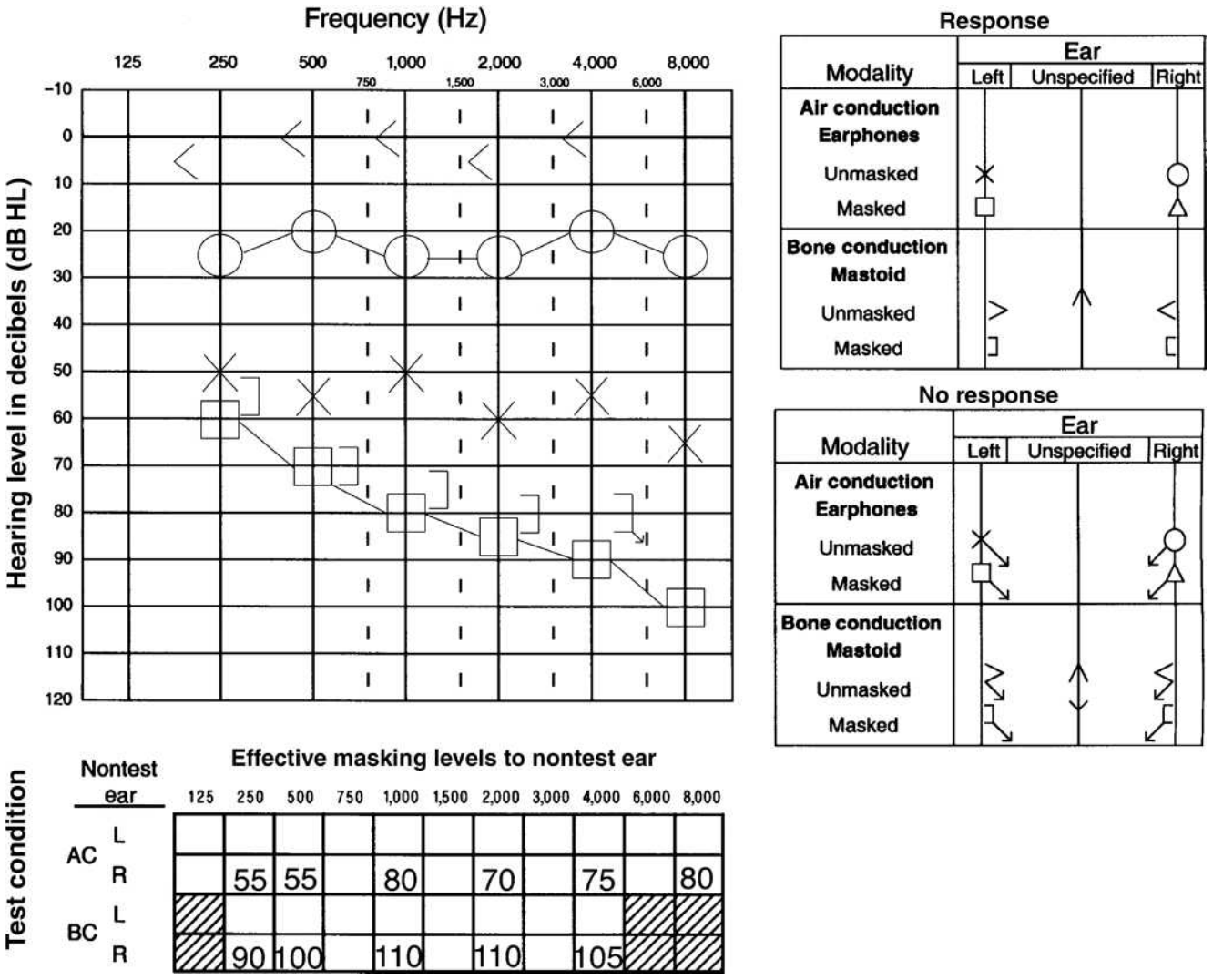


FIGURE 6.12 An example illustrating audiogram interpretation using unmasked and masked puretone thresholds.

It should be noted that the unmasked air-conduction thresholds in the left ear are not considered when interpreting hearing status. Because a significant threshold shift (i.e., >5 dB) occurred when contralateral masking was introduced to the nontest ear, the unmasked air-conduction responses in the left ear actually represent cross-hearing responses from the better (i.e., right) ear. In this case, the unmasked air-conduction thresholds should not be connected with lines. In cases where contralateral masking is required, it is acceptable to record only the masked thresholds (ASHA, 1990).

Although the results of unmasked bone-conduction audiometry suggested that masked bone-conduction thresholds were required in both ears because of potential air-bone gaps, contralateral masking was required only when testing the left ear. Whenever there is an asymmetrical hearing loss, it is traditional to first measure masked bone-conduction thresholds in the poorer ear. There is the assumption that the unmasked bone-conduction thresholds may more likely reflect hearing in the better ear. When masked bone-conduction thresholds were subsequently measured in the left ear, results suggested a sensory/neural hearing loss. Consequently, we can correctly assume that the unmasked responses are originating from the better (i.e., right) ear. Depending on the outcome when measuring masked bone-conduction thresholds in the poorer ear, it is not always necessary to also measure masked thresholds in the opposite ear. As the above example illustrates, ear-specific information can be inferred from unmasked bone-conduction responses in some cases.

It is traditional to record masking levels when obtaining masked air- and bone-conduction thresholds. Assuming that the clinician has used the recommended threshold shift (i.e., plateau) procedure, then a range of masking levels will be used when establishing threshold. ASHA (1990) recommends that the final level of masking used to obtain masked threshold should be recorded for the nontest ear. A table for recording EM levels to the nontest ear is typically provided on an audiogram form. Consider again the audiogram presented in Figure 6.12. For example, a masked puretone air-conduction threshold was measured at 85 dB HL at 2,000 Hz in the left ear; this threshold was obtained with a final masking level of 70 dB EM in the right ear.

Speech Audiometry

The speech audiometry test battery is traditionally composed of two major components: (1) Measures of hearing sensitivity for speech (i.e., speech threshold) and (2) measures of suprathreshold speech recognition. Although the psychoacoustic or threshold shift procedure proves efficient when measuring SDT, the acoustic masking procedure is the method of choice when assessing threshold and suprathreshold speech recognition.

Psychoacoustic Masking Procedures

Recall that the psychoacoustic or threshold shift masking procedures rely on the observation of shifts in the measured threshold in the test ear as a function of masking levels in the nontest ear. The plateau procedure can be applied easily during measurement of speech thresholds (Konkle and Berry, 1983; Studebaker, 1979). A major advantage of the plateau procedure is that information about bone-conduction hearing sensitivity in each ear is not required when selecting appropriate masking levels. Although the plateau procedure can be applied during measurement of both masked recognition and detection thresholds, it proves most efficient during measurement of SDT because of the nature of the response task (i.e., detection rather than recognition).

ASHA's most recent guidelines for determining threshold level for speech were published in 1988. Recommended procedures for measuring both detection and recognition thresholds are described. Given that determination of SDT involves a detection task that is similar to the one used in puretone threshold audiometry, ASHA recommends using a test procedure that follows published guidelines for measuring puretone threshold (e.g., ASHA, 2005). Therefore, the plateau masking procedure recommended earlier for use during puretone threshold audiometry can be used equally effectively when measuring masked SDT.

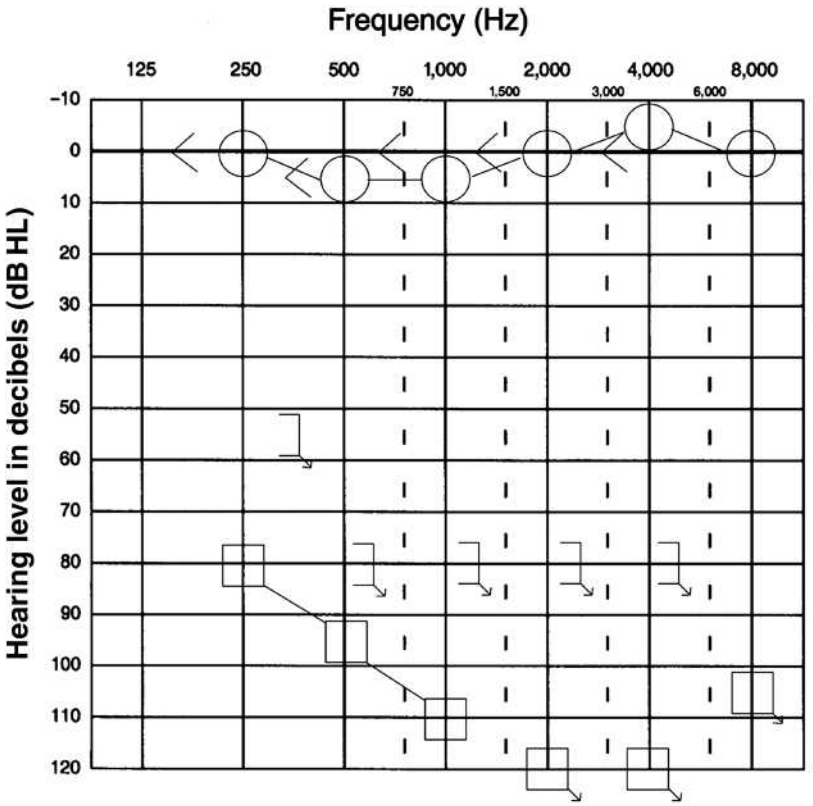
Consider the example presented in Figure 6.13. Audiometry was performed using 3A insert earphones. puretone testing reveals normal hearing in the right ear. There is a profound sensory/neural hearing loss of fragmentary configuration in the left ear. (Contralateral masking was required during measurement of air- and bone-conduction thresholds in the left ear.) An SRT of 5 dB HL was measured in the right ear, a finding that supports the puretone results. When spondaic words were presented at suprathreshold levels in the left ear, the patient was not able to correctly recognize any words. Consequently, a decision was made to measure an SDT.

An unmasked SDT is measured at 75 dB HL in the left ear. Because the difference between the unmasked SDT in the test ear (i.e., 75 dB HL) and the SRT in the nontest ear (i.e., 5 dB HL) clearly exceeds our conservative estimate of IA for speech (i.e., 60 dB) when using 3A insert earphones, contralateral masking will be required.

Using the recommended plateau masking procedure, speech spectrum noise is introduced to the nontest ear at an initial masking level, that is, an EM level (in dB EM) equal to the speech threshold of the nontest ear ($SRT_{Nontest\ Ear}$) plus a 10-dB safety factor:

$$\text{Initial Masking Level} = SRT_{Nontest\ Ear} + 10\ \text{dB}$$

In this example, initial masking level is equal to 15 dB EM. SDT is then re-established in the nontest ear in the presence of the initial masking level. Depending on the patient's response to the speech in the presence of the noise, the level



Response

Modality	Ear: Left	Unspecified	Right
Air conduction Earphones			
Unmasked	×		○
Masked	□		△
Bone conduction Mastoid			
Unmasked	>	^	<
Masked	⌋		⌊

No response

Modality	Ear: Left	Unspecified	Right
Air conduction Earphones			
Unmasked	× ↘		↙ ○
Masked	□ ↘		↙ △
Bone conduction Mastoid			
Unmasked	> ↘	^ ↓	↙ <
Masked	⌋ ↘		↙ ⌊

Test condition

Nontest ear

Effective masking levels to nontest ear

		125	250	500	750	1,000	1,500	2,000	3,000	4,000	6,000	8,000
AC	L											
	R		30	35		50		50		40		35
BC	L											
	R		70	105		100		90		90		

Abbreviations

AC	Air conduction
BC	Bone conduction
CNT	Could not test
DNT	Did not test
EM	Effective masking
FA	Fletcher average
LDL	Loudness discomfort level
MCL	Most comfortable loudness
NR	No response
PTA	Puretone average
SDT	Speech detection threshold
SL	Sensation level
SRT	Speech recognition threshold

Speech audiometry

	PTA	SRT	SDT	MCL / LDL	Speech recognition
					Test:
					SL % / SL %
Right ear	3	5			
Effective masking in left ear		dB EM	dB EM		dB EM / dB EM
Left ear	FA= 103	CNT	90		
Effective masking in right ear		dB EM	40 dB EM		dB EM / dB EM

Comments:

FIGURE 6.13 An example illustrating the use of the threshold shift masking procedure for determining speech detection threshold.

of the speech or noise is increased by 5 dB until a masking plateau has been reached. Remember that it is acceptable to use a 10-dB masker increment when establishing a masking plateau. In this particular case, the risk of overmasking is essentially nonexistent because of the poor bone-conduction hearing sensitivity in the test ear (and the use of an air-conduction transducer with increased IA for presenting the masking noise). Therefore, the use of a 10-dB masker increment can be easily justified. Masked SDT is subsequently measured at 90 dB HL (using 40 dB EM).

Although the plateau masking procedure can be used during assessment of masked SRT, it can prove very time consuming. Recall that only a single detection response to speech is required when measuring masked SDT before making a decision about increasing the level of the speech or masker. The use of the plateau procedure for measuring masked SRT, however, requires that threshold be re-established (i.e., 50% correct recognition of spondaic words) at each masking level until a plateau is reached. The acoustic method proves to be the method of choice when measuring masked SRT because of its greater time efficiency (Konkle and Berry, 1983; Studebaker, 1979) and will be discussed in the following section.

Acoustic Masking Procedures

Recall that acoustic masking procedures are based on calculating the estimated acoustic levels of the test and masking stimuli in the two ears during a test condition and, on this basis, selecting an appropriate masking level. A major disadvantage of the acoustic or formula approach is that the application requires knowledge about air-bone gaps in both test and nontest ears (Konkle and Berry, 1983; Studebaker, 1979). Knowledge about air-bone gaps in the nontest ear is required to calculate minimum masking level. Information about bone-conduction hearing sensitivity in the test ear is required to calculate maximum masking level. Assuming that complete puretone threshold data are available before performing speech audiometry, however, formula approaches for calculating required masking levels prove very effective during measurement of both threshold and suprathreshold speech recognition.

The underlying concepts of minimum and maximum masking levels for speech are similar to those offered earlier for puretone stimuli. Minimum masking level for speech (M_{Min}), originally described by Lidén et al. (1959), can be defined using the following equation:

$$M_{Min} = \text{Presentation Level}_{\text{Test Ear}} - \text{IA} + \text{Largest Air-Bone Gap}_{\text{Nontest Ear}}$$

Presentation $\text{Level}_{\text{Test Ear}}$ represents the HL (dB HL) of the speech signal at the test ear, IA is equal to IA for speech, and Largest Air-Bone $\text{Gap}_{\text{Nontest Ear}}$ represents the largest air-bone gap in the nontest ear in the 250- to 4,000-Hz frequency range. Presentation $\text{Level}_{\text{Test Ear}}$ – IA, an estimate of the HL of the speech signal reaching the test ear, represents the minimum masking level required. The presence of air-bone gaps in the nontest (i.e., masked) ear, however, will reduce the effectiveness of the masker. Consequently, minimum masking level must be increased by the size of the air-bone gap.

Lidén et al. (1959) recommended that the *average* air-bone gap in the nontest ear, calculated using frequencies of 500, 1,000, and 2,000 Hz, be accounted for when determining the minimum masking level. Coles and Priede (1975) suggested a more conservative approach and recommended that the largest air-bone gap at any frequency in the range from 250 to 4,000 Hz be considered. Remember that speech is a broadband signal. Therefore, bone-conduction hearing sensitivity across a range of frequencies in the nontest ear must be considered. There is the assumption that the largest air-bone gap will have the greatest effect on masking level. Following the conservative recommendation of Coles and Priede (1975), it is recommended that the largest air-bone gap across the frequency range in the nontest ear be accounted for when determining minimum masking level.

Maximum masking level (M_{Max}) for speech, originally described by Lidén et al. (1959), can be defined using the following equation:

$$M_{Max} = \text{Best BC}_{\text{Test Ear}} + \text{IA} - 5\,\text{dB}$$

Best $BC_{\text{Test Ear}}$ represents the best bone-conduction threshold in the test ear in the frequency range from 250 to 4,000 Hz, and IA is equal to IA for speech. If Best $BC_{\text{Test Ear}}$ + IA represents a level that will just produce overmasking in the test ear, then a slightly lower masking level should be used clinically. Consequently, a value of 5 dB is subtracted from the calculated level.

Lidén et al. (1959) originally recommended that the *average* puretone bone-conduction threshold in the test ear, again calculated using frequencies of 500, 1,000, and 2,000 Hz, should be accounted for when estimating maximum masking level. However, a more conservative approach includes consideration of the *best* bone-conduction threshold in the test ear over a wider range of frequencies (i.e., 250 to 4,000 Hz). There is the assumption that the best bone-conduction threshold in the test ear is the most susceptible to the effects of overmasking.

The optimal masking level during speech audiometry is one that occurs above the minimum and below the maximum masking levels (Konkle and Berry, 1983; Lidén et al., 1959; Studebaker, 1979). Minimum and maximum masking levels represent, respectively, the lower and upper limits of the masking plateau. Studebaker (1979) states that a major goal of the acoustic or formula approach is to apply rules that will place the masking level at approximately the middle of the range of correct values (i.e., the middle of the

masking plateau). This concept was originally discussed by Luscher and König in 1955 (cited by Studebaker, 1979).

Studebaker (1962) first described an equation for calculating midmasking level during puretone bone-conduction audiometry. The basic concepts underlying the midplateau procedure, however, can be easily applied during speech audiometry. Yacullo (1999) states that a simple approach to calculating the midmasking level (M_{Mid}) involves determining the arithmetic mean of the minimum and maximum masking levels:

$$M_{Mid} = (M_{Min} + M_{Max})/2$$

For example, if M_{Min} is equal to 40 dB EM and M_{Max} is equal to 80 dB EM, then M_{Mid}, the masking level that falls at midplateau, is 60 dB EM. When a masking level falls at the middle of the acceptable masking range (i.e., midmasking level), then the risk of undermasking and overmasking is minimized (Studebaker, 1962). It should be noted that midplateau actually represents a range of values surrounding the midmasking level. Consequently, there is some flexibility in using a somewhat higher or lower masking level.

Yacullo (1999) states that there are two major advantages of the midplateau masking procedure. First, IA is eliminated as a source of error when determining an appropriate masking level. Masking levels are often determined using very conservative estimates of IA. However, IA has equal yet opposite effects on minimum and maximum masking levels. Although the value of IA used for determining minimum and maximum masking levels will influence the width of the masking plateau, the midmasking level remains the same.

Second, midmasking level can be determined for both threshold and suprathreshold speech measures by using the same formula approach (Konkle and Berry, 1983). The midplateau procedure avoids a potential problem during measurement of suprathreshold speech recognition that is related to calibration of EM level and percent correct response criterion. Recall that EM level for speech is specified relative to the SRT (i.e., 50% correct recognition of spondaic words) (ANSI/ASA, 2010). Suprathreshold speech recognition performance, however, can range from 0% to 100%. Konkle and Berry (1983) indicate that a major advantage of the midplateau procedure is that the middle of the masking plateau (i.e., the optimal masking level) is not influenced by different listener response criteria used during assessment of threshold and suprathreshold speech recognition. The reader is referred to Konkle and Berry (1983) and Studebaker (1979) for more detailed discussion.

Studebaker (1979) has described a somewhat different acoustic masking procedure for use during speech audiometry that is consistent with the goal of selecting a masking level that occurs at midplateau. Specifically, the recommended masking level is equal to the presentation level of the speech signal in dB HL at the test ear, adjusted appropriately for air-bone gaps in the test and nontest ears. In cases where there are no air-bone gaps in either ear, the selected masking level is simply equal to the HL of the speech signal. To avoid the use of very high levels of contralateral masking that can sometimes result, Studebaker indicates that it is permissible to reduce the masking level by 20 to 25 dB below the presentation level of the speech signal. The reader is referred to Studebaker (1979) for a more comprehensive discussion.

According to the results of a survey of audiometric practices in the United States, many audiologists "base their masking level for word-recognition testing on the stimulus level presented to the test ear and subtract a set amount, such as 20 dB" (Martin et al., 1998, p 100). Although selection of a masking level that is equal to the presentation level at the test ear minus 20 dB may appear somewhat arbitrary, it can actually be supported by sound theoretical constructs.

Yacullo (1996, 1999) has described a simplified approach, based on the underlying concepts of both the midplateau and Studebaker acoustic procedures, that can be used when selecting contralateral masking levels during speech audiometry. Although this approach was originally described for use during assessment of suprathreshold speech recognition (Yacullo, 1996), it also proves equally effective during measurement of SRT (Yacullo, 1999). Stated simply, EM level is equal to the presentation level of the speech signal in dB HL at the test ear minus 20 dB:

$$\text{dB EM} = \text{Presentation Level}_{\text{Test Ear}} - 20 \text{ dB}$$

Given two prerequisite conditions (which will be discussed shortly), the selected masking level will fall approximately at midplateau. Unfortunately, inappropriate use of this simplified approach can result in undermasking or overmasking.

Jerger and associates (Jerger and Jerger, 1971; Jerger et al., 1966) appear to be the first to report the use of a masking procedure that involved presenting contralateral masking noise at a level 20 dB less than the presentation level of the speech signal at the test ear. Specifically, it was reported that "whenever the speech level to the test ear was sufficiently intense that the signal might conceivably cross over and be heard on the nontest ear, the latter was masked by white noise at a level 20 dB less than the speech presentation level on the test ear" (Jerger and Jerger, 1971, p 574). It should be noted, however, that Jerger and associates used white noise as a contralateral masker rather than the typically used speech spectrum noise. In addition, the white noise was not calibrated in EM level for speech.

More recently, Hannley (1986) and Gelfand (2009) have discussed briefly the simplified approach to masking. Gelfand indicates, however, that using an EM level equal to the HL of the speech signal at the test ear minus 20 dB generally proves most effective in cases of sensory/neural hearing loss. In fact, the desired outcome may not occur when

there are significant air-bone gaps in the nontest ear (e.g., conductive hearing loss).

Yacullo (1999) states that the simplified masking procedure when used appropriately can significantly reduce the calculations required for the determination of optimal (i.e., midmasking) masking level. Specifically, the method proves effective given the following two conditions: (1) There are no significant air-bone gaps (i.e., ≥15 dB) in either ear and (2) speech is presented at a moderate SL (i.e., 30 to 40 dB SL) relative to the measured or estimated SRT. If these two prerequisites are met, then the selected masking level should occur approximately at midplateau.

Acoustic masking procedures are recommended when assessing threshold and suprathreshold speech recognition. The following two examples help illustrate the use of the midplateau masking procedure, as well as the simplified approach when applicable, for measurement of suprathreshold speech recognition and SRT.

The example presented in Figure 6.14 illustrates the use of the midplateau masking procedure during assessment of suprathreshold speech recognition. Puretone testing reveals a mild, sensory/neural hearing loss of flat configuration in the right ear. There is a moderate-to-severe, sensory/neural hearing loss of gradually sloping configuration in the left ear. SRTs were measured at 35 dB HL in the right ear and 55 dB HL in the left ear, findings that support the puretone results. Suprathreshold speech recognition will be assessed at 40 dB SL using Central Institute for the Deaf (CID) W-22 monosyllabic word lists.

Let us first consider the situation where supra-aural earphones are being used during audiometry. Contralateral masking will be required only when measuring suprathreshold speech recognition in the left ear. Specifically, the presentation level of 95 dB HL (i.e., SRT of 55 dB HL + 40 dB SL) exceeds the best bone-conduction threshold of 30 dB HL in the nontest ear by a conservative estimate of IA for speech (i.e., 40 dB):

$$\text{Presentation Level}_{\text{Test Ear}} - \text{Best BC}_{\text{Nontest Ear}} \geq \text{IA}$$

$$95 \text{ dB HL} - 30 \text{ dB HL} \geq 40 \text{ dB}$$

$$65 \text{ dB HL} \geq 40 \text{ dB}$$

We will use the midplateau masking procedure to select an appropriate contralateral masking level. Remember that the midplateau masking procedure involves a three-step process: Calculation of (1) minimum masking level (M_{Min}), (2) maximum masking level (M_{Max}), and (3) midmasking level (M_{Mid}):

$$\begin{aligned} M_{\text{Min}} &= \text{Presentation Level}_{\text{Test Ear}} - \text{IA} \\ &\quad + \text{Largest Air-Bone Gap}_{\text{Nontest Ear}} \\ &= 95 \text{ dB HL} - 40 \text{ dB} + 5 \text{ dB} \\ &= 60 \text{ dB EM} \end{aligned}$$

$$\begin{aligned} M_{\text{Max}} &= \text{Best BC}_{\text{Test Ear}} + \text{IA} - 5 \text{ dB} \\ &= 45 \text{ dB HL} + 40 \text{ dB} - 5 \text{ dB} \\ &= 80 \text{ dB EM} \end{aligned}$$

$$\begin{aligned} M_{\text{Mid}} &= (M_{\text{Min}} + M_{\text{Max}})/2 \\ &= (60 + 80)/2 \\ &= 70 \text{ dB EM} \end{aligned}$$

An EM level of 70 dB is appropriate for three reasons. First, it occurs at midplateau. Second, it occurs at least 10 dB above the calculated minimum. Remember that a safety factor of at least 10 dB or greater should be added to the calculated minimum value to account for intersubject variability with respect to masker effectiveness (Martin, 1974; Studebaker, 1979). Finally, it does not exceed the calculated maximum masking level.

It should be noted that the width of the masking plateau is typically underestimated when a conservative estimate of IA is used for determining the minimum and maximum masking levels. If IA is actually greater than the conservative estimate of 40 dB, then the width of the masking plateau will increase. For example, if this patient actually exhibits IA for speech of 55 dB (rather than the conservative estimate of 40 dB), then the minimum level will be decreased and the maximum level will be increased by the same amount (i.e., 15 dB). Although the width of the masking plateau increases, the midmasking level remains the same. As stated earlier, a major advantage of the midplateau method is that IA is eliminated as a source of error when selecting an appropriate masking level.

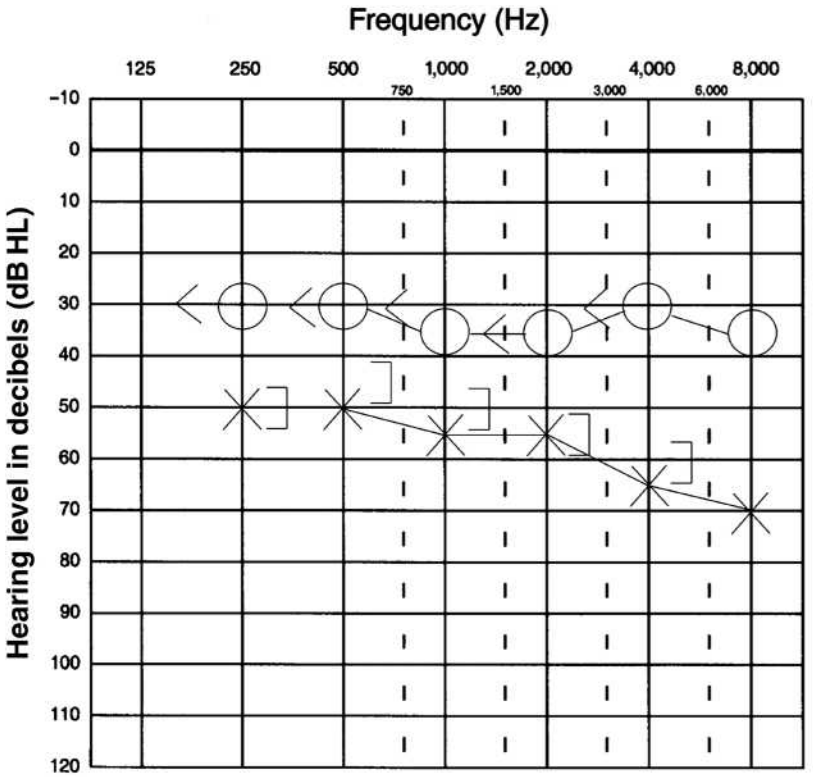
We now will take another look at the example presented in Figure 6.14 and substitute 3A insert earphones for the supra-aural arrangement. Contralateral masking will also be required when assessing suprathreshold speech recognition in the left ear. The presentation level of 95 dB HL (i.e., SRT of 55 dB HL + 40 dB SL) exceeds the best bone-conduction threshold of 30 dB HL in the nontest ear by a conservative estimate of IA for speech (i.e., 60 dB). We will again use the midplateau masking procedure to select an appropriate level of contralateral masking. The calculations are the same for both supra-aural and 3A insert earphones with the exception that an IA value of 60 dB will be substituted in our equations for minimum and maximum masking levels. Masking levels for use with insert earphones are summarized below:

$$M_{\text{Min}} = 40 \text{ dB EM}$$

$$M_{\text{Max}} = 100 \text{ dB EM}$$

$$M_{\text{Mid}} = 70 \text{ dB EM}$$

It should be apparent that an increase in IA has equal yet opposite effects on the minimum and maximum



Response

Modality	Ear: Left	Unspecified	Right
Air conduction Earphones			
Unmasked	×		○
Masked	□		△
Bone conduction Mastoid			
Unmasked	>	^	<
Masked	⌋		⌊

No response

Modality	Ear: Left	Unspecified	Right
Air conduction Earphones			
Unmasked	×		○
Masked	□		△
Bone conduction Mastoid			
Unmasked	>	^	<
Masked	⌋		⌊

Effective masking levels to nontest ear

Test condition

Nontest ear		125	250	500	750	1,000	1,500	2,000	3,000	4,000	6,000	8,000
AC	L											
	R											
BC	L											
	R		90	75		85		75		80		

Abbreviations

AC	Air conduction
BC	Bone conduction
CNT	Could not test
DNT	Did not test
EM	Effective masking
FA	Fletcher average
LDL	Loudness discomfort level
MCL	Most comfortable loudness
NR	No response
PTA	Puretone average
SDT	Speech detection threshold
SL	Sensation level
SRT	Speech recognition threshold

Speech audiometry

	PTA	SRT	SDT	MCL / LDL	Speech recognition
					Test: W-22
					SL / % ; SL / %
Right ear	33	35			40
Effective masking in left ear		dB EM	dB EM		dB EM ; dB EM
Left ear	55	55			40
Effective masking in right ear		dB EM	dB EM		dB EM ; dB EM

Comments:

FIGURE 6.14 An example illustrating the use of the midplateau and simplified masking procedures during assessment of suprathreshold speech recognition.

masking levels. Because IA is increased by 20 dB when using insert earphones, the width of the masking plateau is increased by 40 dB. The midmasking level, however, remains the same.

We now will take one final look at the example presented in Figure 6.14 and consider the use of the simplified masking approach. Because the two prerequisite conditions are met, the simplified approach should result in the selection of an optimal masking level (i.e., midmasking level). Recall that EM level is simply calculated as the presentation level of the speech signal in dB HL at the test ear minus 20 dB. The same equation will be applicable when using any earphone (i.e., 3A and supra-aural):

$$\begin{aligned} \text{dB EM} &= \text{Presentation Level}_{\text{Test Ear}} - 20 \text{ dB} \\ &= 95 \text{ dB HL} - 20 \text{ dB} \\ &= 75 \text{ dB} \end{aligned}$$

It should be noted that the masking level of 75 dB EM calculated using the simplified approach is in good agreement (i.e., ±5 dB) with the value (i.e., 70 dB EM) determined using the midplateau procedure. Yacullo (1999) indicates that there are two major advantages to using the simplified masking approach with 3A insert earphones, which are the result of a wider masking plateau. First, there is greater flexibility in deviating somewhat from the calculated midmasking level while still remaining within an acceptable range of midplateau. It should be noted that the midplateau actually represents a small range of values surrounding the midmasking level. This range of acceptable values essentially increases when using 3A insert earphones. The use of the simplified masking approach can sometimes result in the selection of high masking levels, even though overmasking is not occurring. Consequently, the audiologist can justify subtracting a value of greater than 20 dB (e.g., 25 or 30 dB) from the presentation level at the test ear when using insert earphones. In the example presented in Figure 6.14, an EM level equal to the presentation level in the test ear minus 25 or 30 dB (e.g., 65 or 70 dB EM) would still result in an acceptable masking level that falls within the vicinity of midplateau, yet clearly exceeds the minimum level by a sizeable amount.

Second, there is greater flexibility in deviating slightly from the recommended prerequisite conditions (i.e., no air-bone gaps in either ear, use of moderate SLs) while still remaining within an acceptable range of midplateau and without significantly increasing the potential for overmasking. Consequently, there is greater margin for error when selecting an appropriate level of masking.

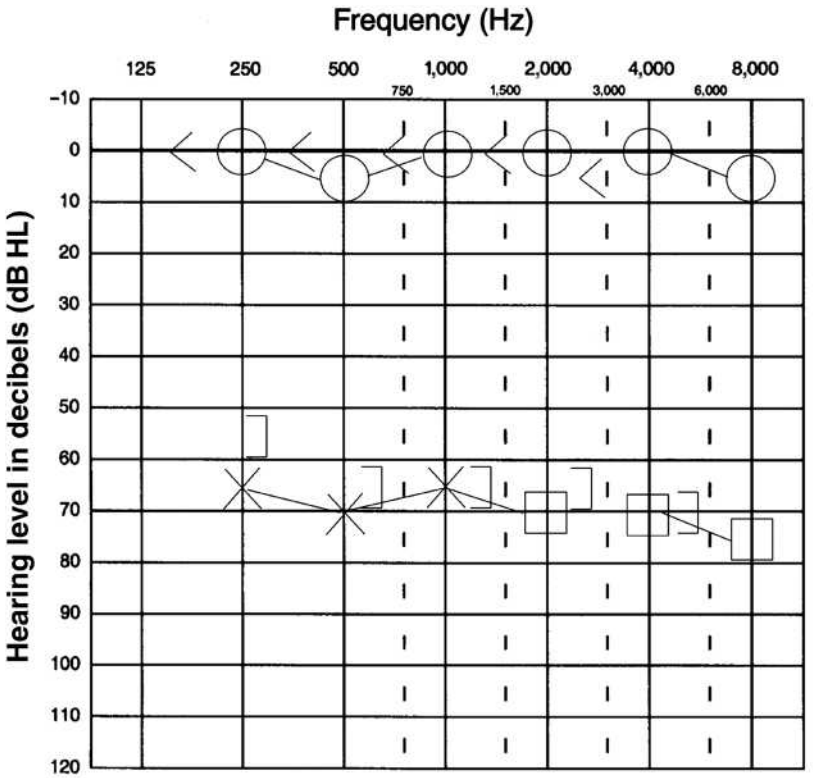
The example presented in Figure 6.15 illustrates the application of the midplateau and simplified masking procedures during measurement of SRT. Audiometry was performed using 3A insert earphones. Puretone testing reveals normal hearing in the right ear. There is a severe sensory/neural hearing loss of flat configuration in the left ear. Based on the puretone findings, it is predicted that SRTs will be measured at approximately 0 dB HL in the right ear and 70 dB HL in the left ear. Prior to measurement of speech thresholds, we can accurately predict whether contralateral masking will be required. Contralateral masking will be required only when measuring SRT in the left ear because the estimated speech threshold of 70 dB HL exceeds the best bone-conduction threshold of 0 dB HL in the nontest ear by a conservative estimate of IA for speech (i.e., 60 dB). An unmasked SRT is subsequently measured in the left ear at 0 dB HL, a finding consistent with the puretone results.

It has already been demonstrated that the simplified masking procedure proves very effective during assessment of suprathreshold speech recognition. However, it can also be applied effectively during measurement of SRT. When selecting an appropriate contralateral masking level when measuring SRT, it is important to consider not only the HL at which the speech threshold is finally established, but also the highest presentation levels used throughout the threshold procedure. Regardless of the SRT procedure used, spondaic words are presented typically at both suprathreshold and threshold levels.

For example, ASHA (1988) recommends a descending threshold technique for measuring SRT that is based on the earlier work of others (Hirsh et al., 1952; Hudgins et al., 1947; Tillman and Olsen, 1973; Wilson et al., 1973). The initial phase involves familiarizing the patient with the spondaic words at a comfortable, suprathreshold HL. (Familiarization with test words is strongly recommended regardless of the SRT procedure.) The preliminary phase involves setting the HL to 30 to 40 dB above the estimated or predicted SRT before descending in 10-dB decrements until two words are missed. In fact, an HL of 30 to 40 dB above the predicted SRT typically results in a comfortable listening level for most patients during the familiarization phase. The test phase involves initially presenting test words at HLs approximately 10 dB higher than the calculated SRT. The calculation of threshold, based on a statistical precedent, takes into account the patient's responses at higher HLs.

Consider again the example presented in Figure 6.15. If the ASHA-recommended procedure is used to measure SRT, then the highest HLs employed (during the familiarization and preliminary phases) will be about 30 to 40 dB above the estimated SRT. In this example, we will use a moderate SL of 30 dB above the estimated SRT (i.e., 70 dB HL + 30 dB SL = 100 dB HL) during the familiarization and preliminary phases.

The use of the simplified approach to selecting an appropriate level of contralateral masking should prove effective in this case because both prerequisite conditions have been met. First, there are no significant air-bone gaps in either ear. Second, speech is presented at a moderate SL (i.e., 30 dB) when the highest HLs are used during the test procedure (i.e., familiarization and preliminary phases). Assuming that 100 dB HL is the highest presentation level



Response

Modality	Ear: Left	Unspecified	Right
Air conduction Earphones			
Unmasked	×		○
Masked	□		△
Bone conduction Mastoid			
Unmasked	>	^	<
Masked	⌋		⌊

No response

Modality	Ear: Left	Unspecified	Right
Air conduction Earphones			
Unmasked	×		○
Masked	□		△
Bone conduction Mastoid			
Unmasked	>	^	<
Masked	⌋		⌊

Test condition

Effective masking levels to nontest ear

Nontest ear		125	250	500	750	1,000	1,500	2,000	3,000	4,000	6,000	8,000
AC	L											
	R							35		30		35
BC	L											
	R		90	95		85		85		90		

Abbreviations

AC	Air conduction
BC	Bone conduction
CNT	Could not test
DNT	Did not test
EM	Effective masking
FA	Fletcher average
LDL	Loudness discomfort level
MCL	Most comfortable loudness
NR	No response
PTA	Puretone average
SDT	Speech detection threshold
SL	Sensation level
SRT	Speech recognition threshold

Speech audiometry

	PTA	SRT	SDT	MCL / LDL	Speech recognition
					Test:
					SL / % ; SL / %
Right ear	2	0			
Effective masking in left ear		dB EM	dB EM		dB EM ; dB EM
Left ear	68	70?			
Effective masking in right ear		dB EM	dB EM		dB EM ; dB EM

Comments:

FIGURE 6.15 An example illustrating the use of the midplateau and simplified masking procedures during assessment of speech recognition threshold.

used during our test procedure, then EM level in the nontest ear is calculated as follows:

$$\begin{aligned}\text{dB EM} &= \text{Presentation Level}_{\text{Test Ear}} - 20\ \text{dB}\\ &= 100\ \text{dB HL} - 20\ \text{dB}\\ &= 80\ \text{dB}\end{aligned}$$

We can verify the appropriateness of the selected masking level by using the midplateau method:

$$\begin{aligned}M_{\text{Min}} &= \text{Presentation Level}_{\text{Test Ear}} - \text{IA}\\ &\quad + \text{Largest Air-Bone Gap}_{\text{Nontest Ear}}\\ &= 100\ \text{dB HL} - 60\ \text{dB} + 0\ \text{dB}\\ &= 40\ \text{dB EM}\end{aligned}$$

$$\begin{aligned}M_{\text{Max}} &= \text{Best BC}_{\text{Test Ear}} + \text{IA} - 5\ \text{dB}\\ &= 55\ \text{dB HL} + 60\ \text{dB} - 5\ \text{dB}\\ &= 110\ \text{dB EM}\end{aligned}$$

$$\begin{aligned}M_{\text{Mid}} &= (M_{\text{Min}} + M_{\text{Max}})/2\\ &= (40 + 110)/2\\ &= 75\ \text{dB EM}\end{aligned}$$

The masking level of 80 dB EM selected using the simplified approach is in good agreement (i.e., ±5 dB) with the value determined using the midplateau approach (i.e., 75 dB EM). Although spondees will be presented at lower HLs when measuring the SRT, it is not necessary to decrease the original level of masking. First, the selected masking level is appropriate for the highest HLs used during all phases of threshold determination. Second, the selected masking level does not exceed the maximum masking level (i.e., overmasking will not occur).

It can be argued that the simplified approach (as well as the midplateau method) can result in the use of unnecessarily high masking levels during measurement of SRT. As was discussed earlier, the midplateau represents a range of masking levels. The audiologist can justify subtracting a value of greater than 20 dB (e.g., 25 or 30 dB) from the presentation level at the test ear, particularly when using insert earphones. In this example, a masking level of 70 or 75 dB EM (rather than the original level of 80 dB EM) still falls within an acceptable range of midplateau, while still occurring significantly higher than the minimum.

Yacullo (1999) states that the simplified masking approach during speech audiometry has wide applicability. First, it can be used with a large and diverse patient population, including those with normal hearing and sensory/neural hearing loss. Second, it can be used equally effectively when using either supra-aural or insert earphones. Third, the procedure can be used in clinical situations where moderate SLs are used. For example, the majority of audiologists in the United States continue to administer suprathreshold word recognition tests at a specified SL referenced to the SRT (Martin and Morris, 1989; Martin et al., 1994, 1998), typically 30 or 40 dB (Martin and Morris, 1989; Martin et al., 1994). Finally, it can be applied effectively during both threshold and suprathreshold measures of speech recognition.

Direct calculation of midmasking level is strongly recommended in cases where there is potential risk of overmasking. Yacullo (1999) states that any factor that increases minimum masking level or decreases maximum masking level will reduce the width of the masking plateau and increase the probability of overmasking. For example, the presence of significant air-bone gaps in the nontest ear and/or the use of higher SLs (i.e., ≥50 dB) will increase minimum masking level. The presence of significant air-bone gaps in the test ear will decrease maximum masking level. In cases where the masking plateau is either very narrow or nonexistent (e.g., unilateral or bilateral conductive hearing loss), knowledge about minimum and maximum masking levels will allow the clinician to make well-informed decisions about appropriate contralateral masking levels.

FOOD FOR THOUGHT

1. Discuss how IA influences decisions about the need for contralateral masking during puretone and speech audiometry.
2. Discuss how the OE influences measured IA for air-conduction transducers (i.e., supra-aural and 3A insert earphones with deeply inserted foam eartips). How does the OE influence contralateral masking levels during bone-conduction audiometry?
3. The plateau masking procedure originally was described by Hood as a method for contralateral masking during puretone threshold audiometry. Discuss how the underlying principles of the masking plateau are applied to procedures for contralateral masking during speech audiometry.

KEY REFERENCES

thePoint A full list of references for this chapter can be found at http://thePoint.lww.com. Below are the key references for this chapter.

American National Standards Institute/Acoustical Society of America (ANSI/ASA). (2010) *American National Standard Specification for Audiometers.* ANSI/ASA 3.6–2010. New York: ANSI/ASA.

American Speech-Language-Hearing Association. (1988) Determining threshold level for speech [Guidelines]. Available online at: www.asha.org/policy.

American Speech-Language-Hearing Association. (1990) Audiometric symbols [Guidelines]. Available online at: www.asha.org/policy.

American Speech-Language-Hearing Association. (2005) Guidelines for manual pure-tone threshold audiometry [Guidelines]. Available online at: www.asha.org/policy.

Beattie RC, Svihovec DV, Edgerton BJ. (1978) Comparison of speech detection and spondee thresholds for half- versus full-list intelligibility scores with MLV and taped presentations of NU-6. *J Am Audiol Soc.* 3, 267–272.

Berger EH, Kerivan JE. (1983) Influence of physiological noise and the occlusion effect on the measurement of real-ear attenuation at threshold. *J Acoust Soc Am.* 74, 81–94.

Berrett MV. (1973) Some relations between interaural attenuation and the occlusion effect. *Unpublished doctoral dissertation.* Iowa City, IA: University of Iowa.

Blackwell KL, Oyler RF, Seyfried DN. (1991) A clinical comparison of Grason Stadler inserts earphones and TDH-50P standard earphones. *Ear Hear.* 12, 361–362.

Chaiklin JB. (1959) The relation among three selected auditory speech thresholds. *J Speech Hear Res.* 2, 237–243.

Chaiklin JB. (1967) Interaural attenuation and cross-hearing in air-conduction audiometry. *J Aud Res.* 7, 413–424.

Coles RRA, Priede VM. (1970) On the misdiagnosis resulting from incorrect use of masking. *J Laryngol Otol.* 84, 41–63.

Coles RRA, Priede VM. (1975) Masking of the non-test ear in speech audiometry. *J Laryngol Otol.* 89, 217–226.

Dean MS, Martin FN. (2000) Insert earphone depth and the occlusion effect. *Am J Audiol.* 9, 131–134.

Dirks DD. (1994) Bone-conduction threshold testing. In: Katz J, ed. *Handbook of Clinical Audiology.* 4th ed. Baltimore, MD: Williams & Wilkins; pp 132–146.

Dirks DD, Malmquist C. (1964) Changes in bone-conduction thresholds produced by masking in the non-test ear. *J Speech Hear Res.* 7, 271–278.

Dirks DD, Swindeman JG. (1967) The variability of occluded and unoccluded bone-conduction thresholds. *J Speech Hear Res.* 10, 232–249.

Dunn HK, White SD. (1940) Statistical measurements on conversational speech. *J Acoust Soc Am.* 11, 278–288.

E-A-R Auditory Systems. (1997) *Instructions for the Use of E-A-RTONE 3 A Insert Earphones.* Revised ed. Indianapolis, IN: E-A-R Auditory Systems.

E-A-R Auditory Systems. (2000a) *Instructions for the Use of E-A-RTONE 5 A Insert Earphones.* Indianapolis, IN: E-A-R Auditory Systems.

E-A-R Auditory Systems. (2000b) *Introducing the New E-A-RTONE® 5 A Insert Earphone [Brochure].* Indianapolis, IN: E-A-R Auditory Systems.

Elpern BS, Naunton RF. (1963) The stability of the occlusion effect. *Arch Otolaryngol.* 77, 376–382.

Feldman AS. (1963) Maximum air-conduction hearing loss. *J Speech Hear Disord.* 6, 157–163.

Fletcher H. (1940) Auditory patterns. *Rev Mod Phys.* 12, 47–65.

Frank T, Vavrek MJ. (1992) Reference threshold levels for an ER-3 A insert earphone. *J Am Acad Audiol.* 3, 51–58.

Gelfand SA. (2009) *Essentials of Audiology.* 3rd ed. New York: Thieme Medical Publishers, Inc.

Goldstein DP, Hayes CS. (1965) The occlusion effect in bone-conduction hearing. *J Speech Hear Res.* 8, 137–148.

Hannley M. (1986) *Basic Principles of Auditory Assessment.* Needham Heights, MA: Allyn & Bacon.

Hawkins JE, Stevens SS. (1950) Masking of pure tones and of speech by white noise. *J Acoust Soc Am.* 22, 6–13.

Hirsh IJ, Davis H, Silverman SR, Reynolds EG, Eldert E, Benson RW. (1952) Development of materials for speech audiometry. *J Speech Hear Disord.* 17, 321–337.

Hodgson W, Tillman T. (1966) Reliability of bone conduction occlusion effects in normals. *J Aud Res.* 6, 141–151.

Hood JD. (1960) The principles and practice of bone-conduction audiometry. *Laryngoscope.* 70, 1211–1228.

Hosford-Dunn H, Kuklinski AL, Raggio M, Haggerty HS. (1986) Solving audiometric masking dilemmas with an insert masker. *Arch Otolaryngol Head Neck Surg.* 112, 92–95.

Hudgins CV, Hawkins JE Jr., Karlin JE, Stevens SS. (1947) The development of recorded auditory tests for measuring hearing loss for speech. *Laryngoscope.* 57, 57–89.

Jerger J, Jerger S. (1971) Diagnostic significance of PB word functions. *Arch Otolaryngol.* 93, 573–580.

Jerger J, Jerger S, Ainsworth J, Caram P. (1966) Recovery of auditory function after surgical removal of cerebellar tumor. *J Speech Hear Disord.* 31, 377–382.

Kaplan H, Gladstone VS, Lloyd LL. (1993) *Audiometric Interpretation.* 2nd ed. Needham Heights, MA: Allyn & Bacon.

Killion MC. (1984) New insert earphones for audiometry. *Hear Instrum.* 35, 28, 46.

Killion MC, Villchur E. (1989) Comments on "Earphones in audiometry" [Zwislocki et al., J. Acoust. Soc. Am. 83, 1688–1689]. *J Acoust Soc Am.* 85, 1775–1778.

Killion MC, Wilber LA, Gudmundsen GI. (1985) Insert earphones for more interaural attenuation. *Hear Instrum.* 36, 34, 36.

Konkle DF, Berry GA. (1983) Masking in speech audiometry. In: Konkle DF, Rintelmann WF, eds. *Principles of Speech Audiometry.* Baltimore, MD: University Park Press; pp 285–319.

Lidén G, Nilsson G, Anderson H. (1959) Masking in clinical audiometry. *Acta Otolaryngol.* 50, 125–136.

Littler TS, Knight JJ, Strange PH. (1952) Hearing by bone conduction and the use of bone-conduction hearing aids. *Proc R Soc Med.* 45, 783–790.

Martin FN. (1966) Speech audiometry and clinical masking. *J Aud Res.* 6, 199–203.

Martin FN. (1967) A simplified method for clinical masking. *J Aud Res.* 7, 59–62.

Martin FN. (1974) Minimum effective masking levels in threshold audiometry. *J Speech Hear Disord.* 39, 280–285.

Martin FN. (1980) The masking plateau revisited. *Ear Hear.* 1, 112–116.

Martin FN, Armstrong TW, Champlin CA. (1994) A survey of audiological practices in the United States in 1992. *Am J Audiol.* 3, 20–26.

Martin FN, Bailey HAT, Pappas JJ. (1965) The effect of central masking on threshold for speech. *J Aud Res.* 5, 293–296.

Martin FN, Blythe ME. (1977) On the cross hearing of spondaic words. *J Aud Res.* 17, 221–224.

Martin FN, Butler EC, Burns P. (1974) Audiometric Bing test for determination of minimum masking levels for bone conduction tests. *J Speech Hear Disord.* 39, 148–152.

Martin FN, Champlin CA, Chambers JA. (1998) Seventh survey of audiometric practices in the United States. *J Am Acad Audiol.* 9, 95–104.

Martin FN, DiGiovanni D. (1979) Central masking effects on spondee threshold as a function of masker sensation level and masker sound pressure level. *J Am Audiol Soc.* 4, 141–146.

Martin FN, Morris LJ. (1989) Current audiologic practices in the United States. *Hear J.* 42, 25–44.

Naunton RF. (1960) A masking dilemma in bilateral conduction deafness. *Arch Otolaryngol.* 72, 753–757.

Olsen WO, Matkin ND. (1991) Speech audiometry. In: Rintelmann WR, ed. *Hearing Assessment.* Needham Heights, MA: Allyn & Bacon; pp 39–140.

Owens E, Schubert ED. (1977) Development of the California Consonant Test. *J Speech Hear Res.* 20, 463–474.

Sanders JW. (1972) Masking. In: Katz J, ed. *Handbook of Clinical Audiology.* Baltimore, MD: Williams & Wilkins; pp 111–142.

Sanders JW. (1991) Clinical masking. In: Rintelmann WF, ed. *Hearing Assessment.* Needham Heights, MA: Allyn & Bacon; pp 141–178.

Sanders JW, Rintelmann WF. (1964) Masking in audiometry. *Arch Otolaryngol.* 80, 541–556.

Schwartz DM, Surr R. (1979) Three experiments on the California Consonant Test. *J Speech Hear Disord.* 64, 61–72.

Silman S, Silverman CA. (1991) *Auditory Diagnosis: Principles and Applications.* San Diego, CA: Academic Press.

Sklare DA, Denenberg LJ. (1987) Interaural attenuation for Tubephone insert earphones. *Ear Hear.* 8, 298–300.

Smith BL, Markides A. (1981) Interaural attenuation for pure tones and speech. *Br J Audiol.* 15, 49–54.

Snyder JM. (1973) Interaural attenuation characteristics in audiometry. *Laryngoscope.* 73, 1847–1855.

Studebaker GA. (1962) On masking in bone-conduction testing. *J Speech Hear Res.* 5, 215–227.

Studebaker GA. (1964) Clinical masking of air- and bone-conducted stimuli. *J Speech Hear Disord.* 29, 23–35.

Studebaker GA. (1967a) Clinical masking of the non-test ear. *J Speech Hear Disord.* 32, 360–371.

Studebaker GA. (1967b) Intertest variability and the air-bone gap. *J Speech Hear Disord.* 32, 82–86.

Studebaker GA. (1979) Clinical masking. In: Rintelmann WF, ed. *Hearing Assessment.* Baltimore, MD: University Park Press; pp 51–100.

Thurlow WR, Silverman SR, Davis H, Walsh TE. (1948) A statistical study of auditory tests in relation to the fenestration operation. *Laryngoscope.* 58, 43–66.

Tillman TW, Olsen WO. (1973) Speech audiometry. In: Jerger J, ed. *Modern Developments in Audiology.* 2nd ed. New York: Academic Press; pp 37–74.

Tonndorf J. (1968) A new concept of bone conduction. *Arch Otolaryngol.* 87, 49–54.

Tonndorf J. (1972) Bone conduction. In: Tobias JV, ed. *Foundations of Modern Auditory Theory.* Vol II. New York: Academic Press; pp 197–237.

Turner RG. (2004a) Masking redux I: An optimized masking method. *J Am Acad Audiol.* 15, 17–28.

Turner RG. (2004b) Masking redux II: A recommended masking protocol. *J Am Acad Audiol.* 15, 29–46.

Van Campen LE, Sammeth CA, Peek BF. (1990) Interaural attenuation using Etymotic ER-3 A insert earphones in auditory brain stem response testing. *Ear Hear.* 11, 66–69.

Wegel RL, Lane GI. (1924) The auditory masking of one pure tone by another and its probable relation to the dynamics of the inner ear. *Phys Rev.* 23, 266–285.

Wilson R, Morgan D, Dirks D. (1973) A proposed SRT procedure and its statistical precedent. *J Speech Hear Disord.* 38, 184–191.

Yacullo WS. (1996) *Clinical Masking Procedures.* Boston, MA: Allyn & Bacon.

Yacullo WS. (1999) Clinical masking in speech audiometry: A simplified approach. *Am J Audiol.* 8, 106–116.

Yacullo WS. (2004) Clinical masking. In: Kent RD, ed. *The MIT Encyclopedia of Communication Disorders.* Cambridge, MA: MIT Press; pp 500–504.

Zwislocki J. (1953) Acoustic attenuation between the ears. *J Acoust Soc Am.* 25, 752–759.

Zwislocki J, Kruger B, Miller JD, Niemoeller AR, Shaw EA, Studebaker G. (1988) Earphones in audiometry. *J Acoust Soc Am.* 83, 1688–1689.

CHAPTER 7

Case History

Douglas L. Beck

INTRODUCTION

Audiologists must critically and judiciously gather and examine all information related to sound perception, tinnitus, hearing, hearing loss, listening (in quiet and noise), dizziness, balance problems, and birth history (for newborns and infants). The audiologist creates and interprets anatomic and physiologic information within the context of a case history, to render an appropriate diagnosis. Audiologists are charged with the responsibility of diagnosing and "nonmedically" treating hearing loss. Traditional wisdom reveals two key ingredients to a correct differential diagnosis: An excellent case history and a thorough physical examination. Given these two key ingredients, the differential diagnosis "emerges" to the trained professional as the only clear answer (i.e., a single differential diagnosis) or potentially a series of equally plausible diagnoses emerge, indicating multiple remaining questions and avenues to be explored and resolved. Indeed, case history gathering is an important skill which facilitates the correct differential diagnosis if the clinician obtains relevant and focused information. Obtaining and using the case history requires skill, patience, practice, and knowledge.

In general, if you're not looking for something, you won't find it. However, simply looking for something doesn't mean you will find it. For example, if you're looking for zebras in a cow pasture, you probably won't find them. Of course this doesn't mean zebras don't exist. However, it may indicate zebras generally don't hang out in cow pastures. To find the correct solution to a given problem, we must pose the right question, formulate reasonable options and alternatives, and ultimately, choose the most probable alternative.

When gathering and assembling case histories, healthcare professionals must narrow the focus and filter the information available quickly and efficiently while pulling together what is most important. The case history questions should be reasonable, and result driven, allowing for an evidence-based outcome.

Across healthcare disciplines (including audiology), the method of choice for obtaining the case history is the "medical model." The medical model efficiently directs the professional to the "chief complaint" (CC) and helps organize information into a rational hierarchy with the most important or likely concerns at the forefront.

Researchers have designed decision trees and analysis weightings and other complex models which are powerful and accurate and, theoretically, will assist in finding the correct diagnosis. However, when the audiologist is working with the patient, assembling the case history is essentially a person-to-person event. Frankly, having good people skills and adjusting our approach (i.e., course corrections) to the client we are addressing, matter a great deal.

CASE HISTORY TOOLS

There are three primary tools used to create a case history: Interviews, questionnaires, and the subjective, objective, assessment, and plan (SOAP) format. These three tools are often used in tandem, but can certainly be used as preferred by the professional.

The audiologist, as a licensed or regulated healthcare professional, has a legal obligation to the patient's health and well-being. The audiologist must be aware of the warning signs of dangerous and treatable medical and surgical conditions and should refer to the appropriate professional when "red flags" are noticed. Red flags include a sudden hearing loss, ear pain, draining or bleeding ears, unilateral symptoms of hearing loss or tinnitus, conductive hearing loss, dizziness, and other referral criteria. Assembling the case history provides an opportunity to identify red flags while considering a multitude of diagnostic and treatment alternatives.

Interview Techniques

Of course, there is no "one correct way" to interview patients. Flexibility is the key, as professionals, patients, work settings, and the particulars of each situation vary. Nonetheless, it is always a good idea to proceed in an orderly and professional manner. Interviews should be patient centered, friendly, and private, in accordance with applicable laws, rules, and regulations.

While gathering the case history, ascertaining an "index of suspicion" regarding the CC is an important part of the interview. If the index of suspicion for the item highest on our list is low, we generally need to look for more probable alternatives. If the index of suspicion is high, we ask further questions to confirm or refute our suspicions.

For example, a patient presenting with a fluctuating low-frequency sensory/neural hearing loss (SNHL) and tinnitus in the same ear, with aural fullness and occasional vertigo, has a low index of suspicion for otosclerosis, but has a reasonably high index of suspicion for Ménière's disease. The high index of suspicion for Ménière's disease would lead us to ask probing questions to note whether the presenting symptomatology is in agreement with a Ménière's diagnosis or would lead us in another direction, such as an acoustic neuroma.

The competent professional understands the probabilities of certain things occurring and the related signs and symptoms of each. Although Ménière's disease is a relatively rare disorder, occurring in less than 1% of the general population, it is a common diagnosis for patients with the symptoms described earlier. Of course, we do not make a medical diagnosis of a disease. However, if the data come together with what we know about the patient the audiologist might include "Consistent with Ménière's disease," in the report.

Three scenarios follow to illustrate the interview technique.

Scenario One

Review any/all assembled paperwork (chart, lab notes, test results, history, etc.) before meeting the patient for the initial consultation. Shake hands and greet the patient, their spouse, significant other, family, and so on, and always introduce yourself. This is an amazingly simple protocol, but it is often overlooked, and when it is overlooked, it sets an unprofessional tone for the rest of the encounter. I usually say, "Good morning. My name is Dr. Beck, I'm an audiologist. Please come in Mr. Smith."

After exchanging greetings and after sitting down in the office, inquire as to why the patient scheduled today's visit.

"Thanks for coming in today Mr. Smith. What brings you to the office?"

Mr. Smith: "I would like a comprehensive audiometric evaluation to confirm my bilateral sensory/neural noise-induced hearing loss that my otolaryngologist diagnosed last week. I am very interested in acquiring two digital hearing aids, and by the way, I am wealthy and do not have insurance. I pay cash, and money is no object. I want to hear everything as best I can."

Because this patient has already been seen and diagnosed by the ear, nose, and throat (ENT) specialist, the index of suspicion for some other disease process or a medical/surgical issue is extremely low.

Scenario Two

Mr. Smith: "Well doc, you know how it is. My wife always complains I have the TV up too loud and it drives her outta the room. I like to be able to hear the darn thing so I keep it a little loud. The same thing happens with the car radio when we're driving to the store. When she sets the volume, I just hear noise and can't tell anything about what they're saying. When I was a boy, I could hear a pin drop from 40 paces."

"I understand. How long have you been playing the TV and radio louder than your wife likes it?"

"Let's see, I started working at the steel fabrication factory 14 years ago, and my son was born 8 years ago…. so yeah, it's been at least 8 or 10 years. When I let her set the volume, I can hear the voices, but I really can't understand what they're saying. That drives me nuts. I told her and I'm telling you too, I ain't gonna wear no hearing aids."

Given the information presented in this scenario, one can make several, reasonable, assumptions. We could assume that Mr. Smith has a noise-induced SNHL, likely impacting 4,000 Hz, and because he cannot hear the consonant sounds (high frequencies), he cannot clearly understand the words spoken to him. We might also assume that Mr. Smith is not going to wear hearing aids and that there is little we can do to assist. However, there are other options and protocols to employ:

"Mr. Smith, have you had a hearing test before?"

"Not since the Army, back some 20 years ago."

"Do both ears seem about the same, or is one ear better than the other?"

"The left ear is terrible—can't hear thunder with that one."

"I see. Do you have any ear pain?"

"None at all. My ears feel fine."

"Okay then. May I take a look?"

"Sure, help yourself."

At this point, the audiologist has a rather low index of suspicion for a tumor, such as an acoustic neuroma, because they occur in about 0.00001% of the population, but a higher index of suspicion for more likely possibilities, including a unilateral sudden sensory/neural loss that went undiagnosed (or maybe Mr. Smith works with his left ear toward a loud machine while wearing hearing protection only in the right ear, or perhaps he experienced head trauma on the left or an explosion near his left side during boot camp; there are lots of possibilities). The examination of the pinna, concha, ear canal, and tympanic membranes is normal. The audiologist says, "Okay, your ears look fine," and the interview continues to determine which diagnosis has the highest index of suspicion.

"Mr. Smith, let me make sure I understand… the right ear is the better ear and the left ear has been bad for a long time. Have you ever had the left ear checked?"

"Yes. I had the doctor look at it a year or two ago when it went bad. He put me on antibiotics and that was the end of it. It didn't get better though, so I left it alone.

"Okay. What about drainage, anything coming out of your ears?"

"No sir."

"Any dizziness or spinning sensations?"

"Not any more. Well, maybe a little. When my left ear was going bad, I had some dizziness, but the doctor looked at it and put me on antibiotics, and the dizziness got better after a while."

"So the dizziness started and the left ear went bad all about a year or two ago?"

"That's right."

"Okay, very good. Are you on any medications?"

"Just a cholesterol pill and a baby aspirin, that's about it."

"Okay, and one last thing I'd like to ask you before we do the hearing test—do you have any ringing or buzzing noises in your ears?"

"Yeah, the darn left ear can't hear anything, but it sure makes a racket. Kinda like a "shhhhh" noise going on in there. Keeps me up at night sometimes."

The audiologist does a comprehensive audiometric evaluation and determines the following audiometric profile:

Right ear: Normal peripheral hearing. Tympanogram normal (type A), ipsilateral reflexes within normal limits (WNL). Word recognition score (WRS) = 96%. Speech reception threshold (SRT) = 15 dB HL.

Left ear: Flat 85 dB sensory/neural (SN) loss. Tympanogram normal (type A), ipsilateral reflexes absent @105 dB stimulus level. WRS = 8%, SRT = SAT (speech awareness threshold used because speech understanding was extremely poor) = 80 dB HL.

The index of suspicion for a left retrocochlear disorder is very high at this point. The case history supports this possibility, and the test results indicate a possible retrocochlear diagnosis for the left ear.

The audiologist refers the patient to an otolaryngologist (preferably an otologist or neurotologist) based on the high index of suspicion for a retrocochlear hearing loss. The otologist meets with and interviews the patient and refers the patient for a magnetic resonance imaging (MRI) study with contrast (gadolinium). A 3-cm vestibular schwannoma (acoustic neuroma) is diagnosed. Mr. Smith is scheduled for surgery 3 weeks later, and the tumor is removed via the translabyrinthine approach.

Scenario Three

Mr. Smith: "Let's see, I started working at this really noisy factory 14 years ago, and my son was born 8 years ago… so yeah, it's been at least 8 or 10 years. When my wife sets the TV, it sounds like everyone is mumbling; I can hear the voices, but I really can't understand what they're saying. That drives me nuts. I told her and I'm telling you too, I ain't gonna wear no hearing aids."

Given the information presented above, one can make several assumptions. We could assume Mr. Smith has a noise-induced SNHL, impacting frequencies around 4,000 Hz, and because of the reduced amplitude and distortion affecting mostly the high-frequency consonant sounds, he cannot clearly hear the words spoken to him. We can also be comfortable in thinking that Mr. Smith is not going to wear hearing aids, which reduces what we can do to assist him. However, there are other options and protocols to explore.

"Mr. Smith, have you had a hearing test before?"

"Not since the Army, back some 20 years ago."

"Do both ears seem about the same, or is one ear better than the other?"

"They're just about the same"

"I see. Any ear pain?"

"None at all. My ears feel fine."

"That's good. May I take a look?"

"Sure doc, knock yourself out."

The pinna, concha, ear canal, and tympanic membranes are normal in appearance. The audiologist says, "Your ears look fine," and the interview continues.

"Okay, what about drainage? Is there anything coming out of your ears?"

"No sir."

"Any dizziness or spinning sensations"

"Nope."

"Very good. Are you taking any medications?"

"Just a cholesterol pill and a baby aspirin, that's about it."

"The last question I'd like to ask you before we do the hearing test is do you have any ringing or buzzing noises in your ears?"

"Yeah…maybe a little when it's really quiet, nothing that really bothers me though."

The audiologist does a comprehensive audiometric evaluation and determines the following audiometric profile:

Right ear: Moderate high-frequency sensory/neural hearing loss. Tympanogram normal (type A), ipsilateral reflexes are within normal limits (WNL). WRS = 96%. SRT = 45 dB HL.

Left ear: Moderate high-frequency sensory/neural hearing loss. Tympanogram normal (type A), ipsilateral reflexes are WNL. WRS = 92%. SRT = 45 dB HL.

"Mr. Smith, I'd like to review the results of today's tests with you. Would you like to have your wife join us while I review the results?"

"Sure, that would be great. She's in the waiting room."

"Hi Mrs. Smith, please join us while I review the results of today's examination. This way, the two of you will have the chance to learn about the results, and I can address your questions."

In this third scenario, the index of suspicion for a noise-induced hearing loss is high, and there are no red flags and no indications of a medical or surgical problem. In essence, the same patient, in three different scenarios, has three separate sets of circumstances, each of which are typically revealed through an interview-based case history, which is more or less driven by the index of suspicion.

Questionnaires

Another very useful and efficient case history tool is the health questionnaire. A well-designed questionnaire is highly focused, simple, takes just a few minutes to fill out, and quickly directs the professional to the area(s) of greatest concern. Questionnaires regarding hearing health care can be presented to patients verbally or written. Written questionnaires are available in electronic and paper-based formats.

However, it is my personal preference to not have patients fill in downloadable, lengthy questionnaires at home. It is terrifically advantageous for the audiologist to spend the time working through a well-designed questionnaire with the patient, to establish rapport and trust and to allow the patient to tell their story. We learn much more about the patient and their situation when we put in the time to ask questions and listen to the patient and then write chart notes reflecting that conversation. Time spent asking questions and listening to and then questioning and clarifying their response is time well spent.

Verbal Presentations

Remember, if you are giving a patient a verbal presentation of a hearing questionnaire, there is already a reasonable index of suspicion for hearing loss. Therefore, sit about 3 ft away from the patient in a well-lit room, face the patient, be sure there is no background noise or visual distractions, and maintain the patient's full attention.

Pencil and Paper Presentations

Keep in mind that, because the majority of patients seen by audiologists are over 55 years of age, large font, dark print, and maximal contrast between the printed words and the background page are preferred and appreciated. Black print on a white background will be the easiest to read. Another important consideration is to use and/or design questionnaires that are easily assessed and tabulated, so the professional can scan the page to find the "positive" results, which will need to be considered.

In 2005, the Centers for Medicare and Medicaid Services (CMS) added a new benefit under Medicare Part B that will likely increase the quantity of pencil and paper–based hearing and balance screenings offered to patients. This benefit is "bundled" within the "Welcome to Medicare" examination. The examination has seven screening sections for physicians, nurses, or nurse practitioners to employ when addressing new patients. Importantly, the Medicare rules state that the screening tests must be in the form of questions or questionnaires and that the selected screening tests must be recognized by a national medical professional organization.

In addition to a wealth of other tests, the American Academy of Audiology (AAA) and the American Speech-Language-Hearing Association (ASHA) have recommended that the following questionnaire be used for this purpose: Hearing Handicap Inventory for the Elderly—Screening Version (HHIE-S; Ventry and Weinstein, 1982). There is likely to be greater popularity for screening tests. Therefore, audiologists should be familiar with the above-noted questionnaires and their format, scoring, and importance.

Subjective, Objective, Assessment, and Plan

Another way to gather useful case history information quickly is to use the standard subjective, objective, assessment, and plan (SOAP) format. The SOAP format is essentially a "medical model" case history–gathering tool. There are many variations on the SOAP format used by clinics, medical schools, and, often, military healthcare facilities.

Critics believe the SOAP format is impersonal and does not recognize the patient as a whole person. In addition, the SOAP format tends to treat the person as if he or she was the disease/disorder/problem, and it calls for the use of jargon and related abbreviations. Although jargon is commonly used in health professions, it can vary from location to location, and it may be nearly impossible for many well-educated colleagues to interpret. In the following examples, abbreviations will be used along with their explanations, which will immediately follow in parenthesis.

Subjective

The subjective section provides a brief subjective history, often focusing on the CC as well as other clinical observations. The patient's medical and audiology history would be placed in this section. Other entries in this section would be notes the patient/relative/friends offer regarding pain or

discomfort and related miscellaneous symptoms. An example follows:

Pt (patient) is 56-year-old, Caucasian, divorced female.
NKA (no known allergies).
Pt has one adult daughter (age 26 years).
Pt has +BP (high blood pressure) that has been under control via meds for 3 years. Pt takes daily multivitamin.
No other known medical issues.
Pt consumes ETOH (alcohol) daily (one glass), stopped smoking 15 years ago.
Previous surgery: C-section 26 years ago. Ingrown toenail (left big toe) operated on 22 years ago.
Today CC: Hearing loss AD (right ear) × 1 mo (1 month duration) with tinnitus, no spinning/vertigo, no complaints AS (left ear).
Pt presents for AE (audiometric evaluation).

Objective

In medical charts, the objective section often includes measures of temperature, blood pressure, skin color, swelling, and other "objective" data that can be obtained in the office easily and quickly. This section is where the audiologist would write the "objective" test results. An example follows:

Puretones:
65 dB HL SNHL (sensory/neural hearing loss) AD (right)
AS (left) WNL (within normal limits)
SRT (speech reception threshold):
70 dB HL AD, 15 dB HL AS
SAT (speech awareness threshold):
60 dB HL AD
15 dB HL AS
WRS (word recognition score):
24% AD at SAT plus 35 dB with contralateral masking
100% AS
OAEs (otoacoustic emissions):
AD ABS (absent)
AS WNL
Tympanograms:
WNL AU (within normal limits, both ears)

Assessment

The assessment section is where the physician or audiologist would make a statement about the probable "working" diagnosis, or the final diagnosis, and prognosis. For example,

Pt presents with probable AD SNHL (right sensory/neural hearing loss), possibly from untreated sudden SNHL, possibly retrocochlear?

Plan

The plan is the "plan" as of this moment, moving forward. The physician may write the recommended prescriptions or may order blood tests, lab work, or radiology tests, as needed. The audiologist might write

Refer pt to ENT for AD asymmetric, SNHL to R/O (rule out) retrocochlear origin or other medical/surgical concerns. Assuming medical/surgical is R/O, proceed with hearing aid evaluation AD.

Although the SOAP format is a quick and an efficient way to gather the history and related information, it may ignore more global problems, while attending primarily to the CC.

SUMMARY

Gathering an efficient and thorough case history requires understanding, patience, and knowledge of hearing, hearing loss, and related disorders. Although there are options regarding the preferred method with which to gather a case history, there is no alternative to accuracy.

Whichever protocol(s) is (are) chosen, the clinician has the responsibility of assembling the information in a meaningful and relevant way to maximally address the needs, concerns, and well-being of the patient.

FOOD FOR THOUGHT

1. As we move forward, of course the electronic medical record (EMR) will play a more prominent role in medicine, audiology and will increasingly impact the case history. Although the EMR will eventually be standardized and comprehensive (at least we can hope!). Do you believe a better "Case History" will come about using a one-on-one dialog, rather than a checklist approach?
2. Do you feel that the human side (i.e., information, emotions and the relationship between the professional and the patient) will greatly impact the type of case history we use?
3. Although the standardized EMR will (eventually) enable the gathering and analysis of comprehensive and objective data, do you feel that this will reduce the value of dialog between the patient and the professional? That is, how important is the patient's story more-or-less in their own words for the professional?

REFERENCE

Ventry IM, Weinstein BE. (1982) The hearing handicap inventory for the elderly: A new tool. *Ear Hear.* 3, 128–134.

CHAPTER 8

Diagnostic Audiology

Brian M. Kreisman, Jennifer L. Smart, and Andrew B. John

INTRODUCTION

Diagnostic audiology is the use of audiologic tests to determine the location of a problem in the auditory system and, in many cases, further insights about the disorder. Diagnostic audiology can be likened to crime shows you may watch on television. Each test serves as a clue that points toward a diagnosis of the patient's hearing disorder. However, if individual tests (or clues) are examined without taking other evidence into consideration, a wrong conclusion might be made. For audiologists to make correct diagnoses, an audiologic test battery is used. A test battery is a series or combination of tests used to assess the auditory system. For most of the examples in this chapter, we will limit discussion of diagnostic audiology to tests that are commonly performed in an audiologic clinic, including puretone air-conduction and bone-conduction testing, speech testing, tympanometry, acoustic reflex thresholds (ARTs, also called middle-ear muscle reflexes [MEMRs]), and otoacoustic emissions (OAEs). These tests are discussed more fully in other chapters in this textbook and we refer you to these chapters for specific test procedures and norms (see Table 8.1); however, it is important to understand how to utilize these tests synergistically to arrive at an accurate diagnosis for each patient. Audiometric tests are used in conjunction with one another to help reinforce or, alternatively, rule out the diagnosis of a particular type of hearing loss or the site of lesion. The test battery is useful for determining some, but not all, auditory disorders.

Following a brief introduction to the cross-check principles employed by audiologists, this chapter will utilize a case study format. Finally, we will address the limitations of the test battery and discuss situations when referrals for other testing are indicated.

TABLE 8.1

Audiology Procedures Discussed in This Chapter

Test	Chapter
Puretone testing (air and bone conduction)	3
Speech testing (quiet)	5
Speech-in-noise testing	5
Tympanometry	9
Acoustic reflex thresholds	10
Otoacoustic emissions	19

CROSS-CHECKING TEST RESULTS

The major reason that an audiologist uses a diagnostic battery is to be able to check the results of individual tests with each other. The idea that "the results of a single test are cross-checked by an independent test measure" is referred to as the cross-check principle (Jerger and Hayes, 1976, p. 614). Since the cross-check principle was first proposed, many manuscripts have revisited the concept as new diagnostic tests have been developed and different test batteries have been proposed to diagnose specific disorders. The goal of comparing the results of two or more tests is to increase the rate of correct identification of disorders (hit rate) and to decrease the rate of diagnosing a disorder when no disorder exists (false alarm rate) (Turner, 2003).

Cross-checks for Puretone Air Conduction

If you only obtained puretone air-conduction thresholds then you would not be able to accurately diagnose the type of hearing loss. Air-conduction audiometry is normally cross checked with bone-conduction audiometry or tympanometry to rule out a conductive component of the hearing loss. If a difference greater than 10 dB exists between the air-conduction and bone-conduction thresholds at the same frequency, a conductive component is indicated. Similarly, air-conduction thresholds for an ear may be within normal limits; however, if a tympanogram for that ear falls outside of the norms for middle-ear pressure and compliance (e.g., Jerger Type B or Type C), a conductive component may be present. ARTs can reveal more information about the type of loss based on the pattern of responses obtained, thus serving as an additional cross-check for puretone air conduction.

Cross-checks for Puretone Audiometry

When puretone audiometry (air- and bone-conduction testing) suggests a significant air–bone gap, tympanometry and ARTs can be used to reinforce the diagnosis of the conductive element and to contribute to a specific diagnosis. OAEs also can be used as a cross-check of puretone audiometry. OAEs are used to assess the health of the outer hair cells of the cochlea, but their measurement may be affected by disorders in the conductive pathway. An audiologist might use OAEs as a cross-check to aid in potentially ruling out a nonorganic hearing loss, to verify outer hair cell function and the degree of cochlear hearing loss, and to further assist with the diagnosis of conductive components, auditory neuropathy spectrum disorder (ANSD), and other retrocochlear disorders. In addition, ARTs have been used to cross check puretone audiometry (Jerger et al., 1974), although other objective tests, such as tone-burst–stimulated auditory brainstem response (ABR), are considered to be better procedures for estimating hearing thresholds. Acoustic reflexes can be used to help identify the presence of hearing loss in young children as well as in adults with language and/or cognitive issues that may reduce the validity and reliability of behavioral measures (Hall, 2010). Acoustic reflexes can also be used to determine site of lesion within the auditory pathway, specifically in differentiating between cochlear and retrocochlear pathologies.

Cross-check for Puretone Average

A puretone average (PTA) is usually calculated as the average of the air-conduction thresholds at 500, 1,000, and 2,000 Hz for each ear. Normally, the PTA should agree with the speech recognition threshold (SRT), meaning that the PTA and SRT should be within 10 dB of one another in the same ear. One instance in which the audiometric thresholds may cause the PTA to be greater than the SRT by 10 dB is when the audiogram configuration is sharply sloping or sharply rising. In such instances, it is preferable to use a two-frequency PTA by averaging the two lowest (e.g., best) thresholds at 500, 1,000, and 2,000 Hz. The two-frequency PTA should then be in agreement with the SRT. Another instance in which the PTA and SRT may disagree is if a person is malingering or intentionally exaggerating a hearing loss. Outside of these special circumstances, we would expect SRTs and PTAs to be highly correlated (except when language or foreign language is a major factor). This allows us to use the SRT to validate the PTA (American Speech-Language-Hearing Association, 1988).

Considerations for Assessing Speech Understanding

One additional step that audiologists may take to address a patient's complaint of not being able to understand speech in noisy environments is to administer a speech-in-noise test in addition to the word recognition testing in quiet. Although this is technically not a cross-check, the addition of a speech-in-noise test, especially with sentence stimuli, will provide a more realistic test environment to evaluate a common patient complaint. The puretone audiogram does not necessarily correlate with the amount of difficulty a listener will have in noise (Killion and Niquette, 2000). In addition, when word recognition testing is performed in quiet at a single speech presentation level, no guarantee exists that the test is measuring the patient's maximum speech understanding (Wiley et al., 1995).

Cross-check Considerations for Pediatric Testing

For children, it is imperative that the audiologist utilize the cross-check principle. The behavioral responses obtained via behavioral observation audiometry (BOA) or visual reinforcement audiometry (VRA) are considered to be accurate reflections of a child's true thresholds when these tests are conducted carefully (Madell and Flexer, 2008). However, because children often do not respond as consistently or as quickly as adults, it is possible that a child's behavioral responses may still be elevated compared to actual thresholds. As a result, the audiologist may judge the child's responses as unreliable (Baldwin et al., 2010). Regardless of the judged reliability of such measures, audiologists should use objective tests such as OAEs and tympanometry as cross-checks for pediatric behavioral responses (Baldwin et al., 2010; Littman et al., 1998; Madell and Flexer, 2008). In addition, OAEs and acoustic reflexes have been shown to be good cross-checks for ABR in young children (Berlin et al., 2010; Stach et al., 1993). The Joint Committee on Infant Hearing Position Statement (JCIH; American Academy of Pediatrics, 2007) also recommends that electrophysiological measures be employed as a cross-check for behavioral response audiometry for children younger than 6 months chronological age. The statement further stresses the importance of obtaining behavioral thresholds as soon as possible using the most age-appropriate method "to cross check and augment physiologic findings" (American Academy of Pediatrics, 2007, p. 910).

Electrophysiological Tests as Cross-checks

Although beyond the scope of this chapter, it should be noted that certain electrophysiological tests can be used to cross check behavioral measures, as well as to cross check each other and to help confirm diagnoses of certain disorders (Bachmann and Hall, 1998; Berlin et al., 2010; Gravel, 2002; Hall and Bondurant, 2009; Stapells, 2011). For example, Berlin et al. (2010) discussed the use of cross-checking

TABLE 8.2

Summary of Cross-checks Used in Diagnostic Audiology

Test	Test	Cross-check
Air conduction	Bone conduction	Rule out conductive component (air–bone gap)
Puretone audiometry	Tympanometry	Helps to verify/rule out middle-ear pathology (air–bone gaps); rule out nonorganic hearing loss
Puretone audiometry	Otoacoustic emissions	Helps to verify/rule out middle-ear pathology; helps to confirm outer hair cell function; rule out nonorganic hearing loss
Puretone audiometry	Acoustic reflexes	Helps to determine site of lesion (e.g., differentiate cochlear from retrocochlear hearing loss); helps to determine degree of hearing loss and rule out nonorganic hearing loss
Puretone average	Speech recognition threshold	Verify performance on both measures (SRT should correlate with PTA)
Speech in quiet (WRS)	Speech-in-noise tests	Compare speech perception in quiet (normal audiologic testing) to noise (more realistic test that addresses many patient complaints of not understanding in noise)
BOA	Electrophysiological measures	Better estimate/confirmation of true thresholds
VRA	Electrophysiological measures	Better estimate of true thresholds (if VRA responses unreliable)

Note: Tests do not need to be administered in this order.

test results to diagnose ANSD: "…the presence of a [cochlear microphonic] or reversing waves at the beginning of the trace does NOT make a diagnosis of ANSD…without the cross-check of middle-ear muscle reflexes (MEMR), OAEs, and an ABR latency-intensity function" (p. 32). For further information about these tests, the reader is referred to the chapters that discuss electrophysiological tests in the text. Table 8.2 summarizes many of the cross-check tests that are used in audiology.

Order of Tests Administered

Although we acknowledge that there is considerable variability in test protocols across clinics, we recommend that testing begin with the objective tests unless contraindicated. At least two major advantages can be found for testing objective measures first. The first advantage is that the audiologist will have a good idea of the type and degree of hearing loss before beginning the subjective tests. The second advantage is the reduced risk for misdiagnosis of disorders such as ANSD and vestibular schwannoma, as well as failure to detect a patient who is malingering. One caveat needs to be discussed with conducting objective tests first. With rising costs of health care we need to be cautious that we are doing tests that are necessary. It is possible that, if the patient's hearing is completely normal (with no listening complaints) or the patient is profoundly deaf (with previous documentation to support the initial diagnosis), tests such as OAEs and acoustic reflexes will be unlikely to add further information about the patient (but we would recommend that everyone have tympanometry for the reasons previously discussed). We think that a nonorganic component is more likely to be present during subjective testing and may not be discovered until cross-checked with objective tests. For these reasons, we recommend objective testing first. A suggested testing order is shown in Figure 8.1. Nevertheless, it should be noted that some audiologists advocate giving puretone and speech tests first when the patient may be more alert and can actively respond to these tests and then relax during the objective tests.

Beyond the Test Battery

Although the use of a test battery is important, it is also vital for the audiologist to remember the case history and the patient complaints. In some ways, one may consider this patient-reported information to be a cross-check of the test battery itself. The case studies presented below demonstrate examples of diagnostic audiology in action.

CASE STUDIES

The importance of objective testing in conjunction with subjective tests can be seen through the use of case examples. The following cases are a range of examples that highlight the use and benefit of incorporating the cross-check principle into your clinical practice. The cases will be presented with a brief history and test results. A discussion of potential difficulties and challenges in interpreting the audiologic data is incorporated within each case. Although

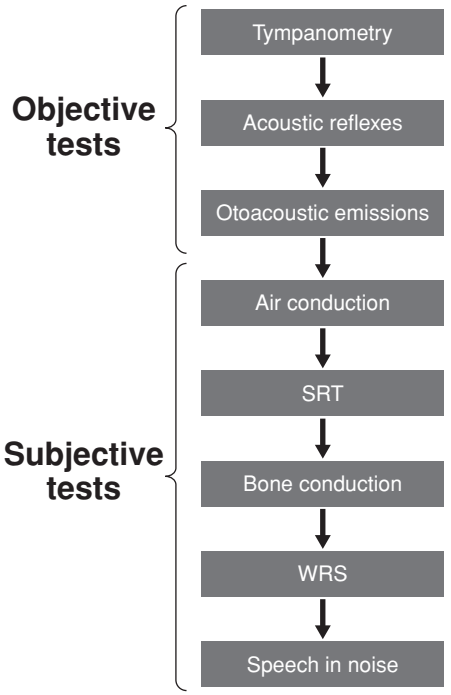


FIGURE 8.1 A suggested comprehensive diagnostic test battery.

there is an ideal order to the test sequence as noted above, the order of tests often varies because of tester preference, patient complaint, age of patient, and so on. Therefore, the test order in the cases below varies to better represent what may be actually done in a clinical setting or what a student clinician may see from his/her supervisors. For the sake of simplicity, all of the cases represent testing with standard audiologic procedures on adults. As you read through each case, we encourage you to make a decision tree based on the order of tests presented in the case and then think about how you might decide to change the test order. It is important to review your clinical decision making periodically to ensure that your practice is evidence based.

Case 1

Case History

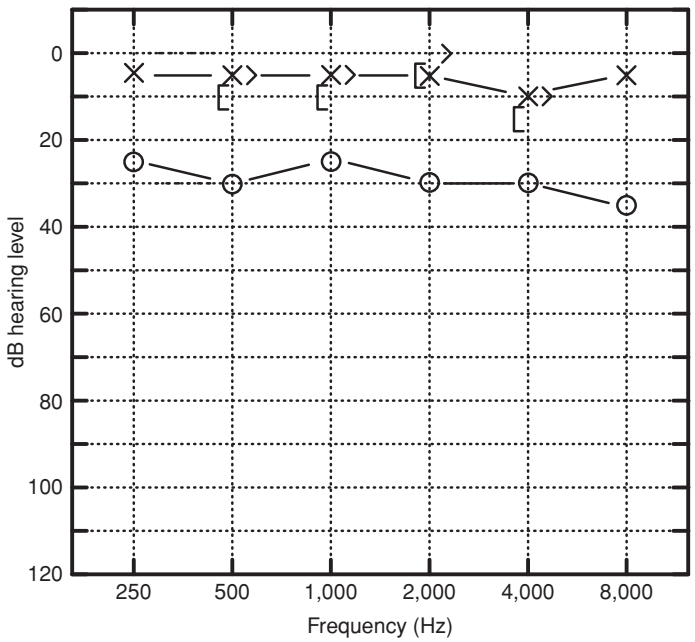
Mr. Ang Kim, age 36, is being seen today after he failed the hearing screening at his company's health fair. His medical history is generally unremarkable, though he reports that he is just getting over a sinus infection and recently underwent surgery for a slipped disc in his back. You have back-to-back patients today and because there is nothing remarkable in his history you decide to do a quick audiogram and send him on his way. Results from otoscopy, puretone, and speech audiometry are shown in Table 8.3 and Figure 8.2.

With subjective information alone this audiogram could indicate many things. For example, you may inaccurately diagnose Mr. Kim with a collapsed ear canal, an impacted cerumen plug, or a perforated tympanic membrane without additional tests to cross check your findings.

TABLE 8.3

Otoscopy Findings for Case 1

Right Ear	Left Ear
Stenotic ear canal, could not visualize tympanic membrane	Stenotic ear canal, could not visualize tympanic membrane



Transducer	Supra-aural headphones	
Reliability	Good	
Results:		
	Right	**Left**
PTA	28 dB HL	5 dB HL
SRT	25 dB HL	5 dB HL
WRS	92% @ 65 dB HL	100% @ 45 dB HL

FIGURE 8.2 Puretone and speech audiometry results for case 1.

TABLE 8.4

Tympanometry Results (226-Hz Probe Tone) for Case 1

	Right	Left
ECV	0.8 mL	0.7 mL
Compliance	NP	0.6 mL
Middle-ear pressure	NP	–50 daPa

TABLE 8.6

Transient-evoked Otoacoustic Emission Results for Case 1

Ear	1,000 Hz	1,400 Hz	2,000 Hz	2,800 Hz	4,000 Hz
Right	Absent	Absent	Absent	Absent	Absent
Left	10.5 dB	10.3 dB	11.4 dB	14.9 dB	13.9 dB

TABLE 8.5

Acoustic Reflexes Results (in dB HL) for Case 1

	Ipsilateral			Contralateral		
Stimulus Ear	**500 Hz**	**1,000 Hz**	**2,000 Hz**	**500 Hz**	**1,000 Hz**	**2,000 Hz**
Right	NR	NR	NR	110 dB	110 dB	105 dB
Left	85 dB	80 dB	85 dB	110 dB	105 dB	105 dB

NR, no response to maximum presentation (110 dB HL).

Despite your busy schedule, you decide you need more information to make an accurate diagnosis, so you perform objective testing to cross check your subjective results. The results from immittance testing and OAE testing are shown in Tables 8.4–8.6.

With this information, you have several different tests to confirm your finding of a conductive hearing loss. The Type B tympanogram in the right ear reveals normal ear canal volume but no mobility. The normal ear canal volume suggests that the TM is not perforated and there is no cerumen plug. The pattern of the ARTs is consistent with a right conductive pathology. TEOAEs in the right ear are absent which is expected with a conductive pathology.

The combination of the subjective *and* objective test results correctly leads you to suspect otitis media with effusion and would require a referral for Mr. Kim to a physician. In this case, you are able to make an appropriate referral based on the information you obtained from a test battery incorporating both objective and subjective measures.

Case 2

Case History

Mrs. Edith Jones, age 77, is being seen today for a hearing test. She does not perceive a listening difficulty but her husband was recently fit with hearing aids and insisted she have her hearing checked too. Her medical history is remarkable for high blood pressure and type 2 diabetes which are both controlled by medication.

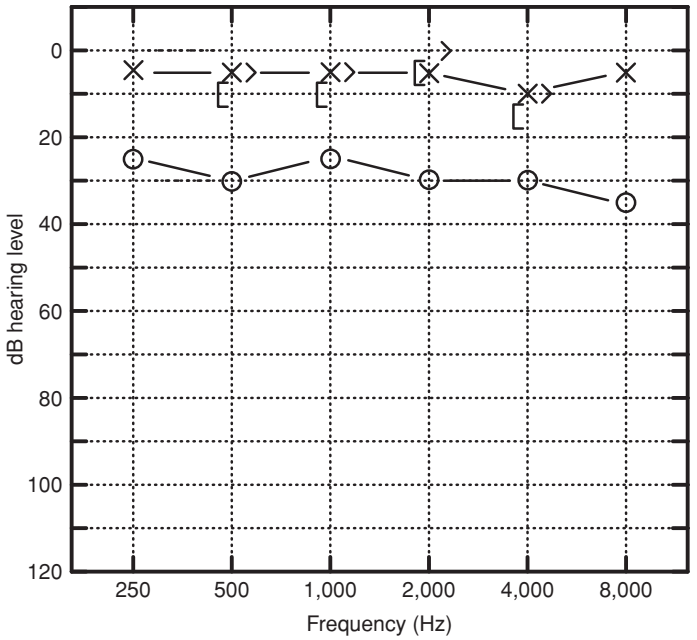
You conduct a basic audiometric evaluation on Mrs. Jones. Results for otoscopy are displayed in Table 8.7 and puretone and speech audiometry results are shown in Figure 8.3.

If you decide not to proceed with further tests to cross check your results, you might diagnose this patient with normal hearing in the right ear and a mild conductive hearing loss in the left ear. You might then refer Mrs. Jones to an Ear Nose and Throat physician who would order more tests.

Instead, you decide to proceed and include additional tests in your battery that would provide a cross-check. We will review those results next (see Tables 8.8–8.10).

These results suggest that Mrs. Jones has normal hearing that contradicts your puretone findings. Normal results on tympanometry, ARTs, and TEOAEs are not consistent with a mild conductive hearing loss. With this information you review the patient's case history and puretone findings again and realize that the apparent conductive hearing loss in the right ear is likely the result of a collapsing ear canal. It is not uncommon for the pressure of the supra-aural headphones to cause the canal to collapse, particularly in older patients for whom the cartilage supporting the ear canal is soft. To confirm this finding you decide to retest Mrs. Jones with insert earphones. When you repeat your audiogram using the insert earphones, you measure Mrs. Jones's right-ear air-conduction thresholds at 5 or 10 dB HL for all frequencies tested. You are able to report to Mrs. Jones that her hearing appears to be normal!

Both cases 1 and 2 highlight the importance of using objective test results in conjunction with subjective test results to avoid misdiagnosis. Both audiograms revealed similar test results but very different actual diagnoses, which were only confirmed with the use of objective testing.



Transducer	Supra-aural headphones	
Reliability	Good	
Results:		
	Right	Left
PTA	28 dB HL	5 dB HL
SRT	30 dB HL	10 dB HL
WRS	96% @ 70 dB HL	100% @ 50 dB HL

FIGURE 8.3 Puretone and speech audiometry results for case 2.

TABLE 8.7

Otoscopy Findings for Case 2

Right Ear	Left Ear
Stenotic ear canal, could not tympanic membrane	Stenotic ear canal, could not tympanic membrane

TABLE 8.8

Tympanometry Results (226-Hz Probe Tone) for Case 2

	Right	Left
ECV	1.3 mL	1.4 mL
Compliance	0.7 mL	0.8 mL
Middle-ear pressure	0 daPa	–5 daPa

TABLE 8.9

Acoustic Reflexes Results (in dB HL) for Case 2

	Ipsilateral			Contralateral		
Stimulus Ear	500 Hz	1,000 Hz	2,000 Hz	500 Hz	1,000 Hz	2,000 Hz
Right	85 dB	85 dB	80 dB	90 dB	95 dB	95 dB
Left	85 dB	80 dB	85 dB	95 dB	90 dB	90 dB

TABLE 8.10

Transient-evoked Otoacoustic Emission Results for Case 2

Ear	1,000 Hz	1,400 Hz	2,000 Hz	2,800 Hz	4,000 Hz
Right	8.9 dB	9.1 dB	12.3 dB	10.4 dB	7.3 dB
Left	9.9 dB	10.4 dB	10.5 dB	9.7 dB	6.1 dB

TABLE 8.11

Otoscopy Findings for Case 3

Right Ear	Left Ear
Clear ear canal; intact tympanic membrane	Clear ear canal; intact tympanic membrane

TABLE 8.12

Tympanometry Results (226-Hz Probe Tone) for Case 3

	Right	Left
ECV	1.0 mL	1.1 mL
Compliance	0.5 mL	0.6 mL
Middle-ear pressure	5 daPa	–20 daPa

Case 3

You receive the following case, accompanied by a patient-signed medical information release, via fax. A recently graduated audiologist at a practice across town just finished testing Mr. Smith and would like a second opinion.

Case History

Mr. Aaron Smith, 49, reports that he can no longer hear out of his left ear. He works in construction and reports that a transformer overloaded at his work site yesterday, resulting in a loud explosion which he believes caused his hearing loss. Mr. Smith reported that his hearing was normal prior to the explosion. He denies any aural fullness, tinnitus, or dizziness. His medical history is unremarkable and he denies any other injuries as a result of the explosion. Results of the audiologic testing are shown in Tables 8.11–8.13 and Figure 8.4.

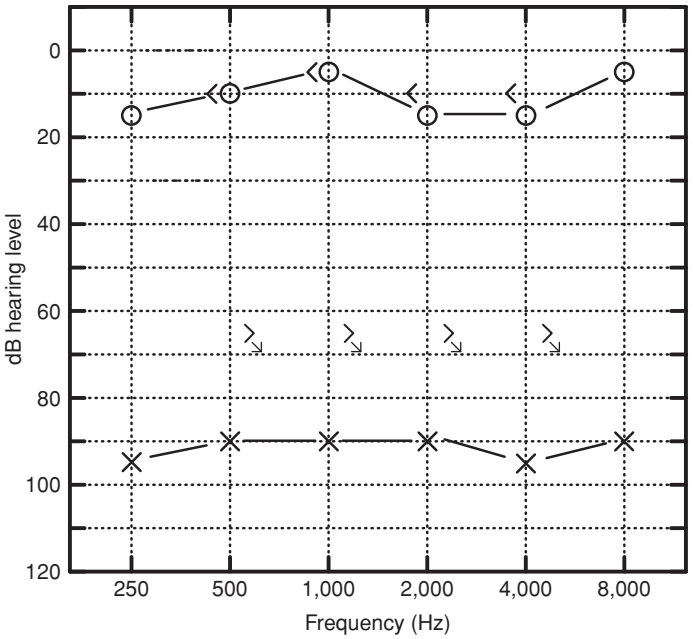
You call the audiologist right away and review your concerns with her. Both the air-conduction and bone-conduction thresholds for the left ear need to be masked. Cross-hearing should have occurred before those thresholds were obtained. Furthermore, you would not expect to obtain no response for bone-conduction testing with the bone oscillator on the left side when the hearing in the right ear is evidently normal. You also note that the PTA and the SRT are not in agreement for the left ear. ARTs are better than you would expect them to be (given the puretone thresholds for the left ear). A patient with hearing thresholds at 90 dB HL would be expected to have ARTs in the range of 95 to 125 dB HL at 500 Hz and 100 to 125 dB HL at 1,000 and 2,000 Hz (Gelfand et al., 1990). Lastly, the WRS was only obtained at 20 dB SL in the left ear, yet Mr. Smith's WRS is 76%, which is better than expected. According to Dubno et al. (1995), a patient with a PTA of 90 dB HL would have an expected WRS of less than 24%. You suggest to the other audiologist that obtaining TEOAEs would further assist in this diagnosis. The audiologist performs TEOAEs (see Table 8.14) to confirm the suspected diagnosis and faxes the results to you.

Based on the pattern of test results, your suspected diagnosis is nonorganic hearing loss. Let us review the facts. First, the patient's left ear thresholds are elevated above where cross-hearing should have occurred. Second, the objective test results (tympanometry, ARTs, and OAEs) reveal no conductive component and suggest that outer hair cells are functioning normally. However, the puretone and speech audiometry results suggest a severe-to-profound unilateral hearing loss in the left ear, which is inconsistent with the objective results. Several cross-checks identified inconsistencies (e.g., ARTs and puretones; PTA–SRT agreement; puretone thresholds and OAEs). At this point, you could suggest that the audiologist reinstruct the patient and then retest the left ear, masking appropriately. If the thresholds for the left ear are still elevated, a Stenger test could be performed to confirm the accuracy of the left puretone thresholds. If the Stenger test result is positive (i.e., the patient does not respond to the stimulus), this would be additional evidence that the apparent hearing loss is nonorganic. This case highlights the importance of a high-quality diagnostic battery (including masking where appropriate) and use of cross-checks to confirm our test results.

TABLE 8.13

Acoustic Reflexes Results (in dB HL) for Case 3

	Ipsilateral			Contralateral		
Stimulus Ear	**500 Hz**	**1,000 Hz**	**2,000 Hz**	**500 Hz**	**1,000 Hz**	**2,000 Hz**
Right	80 dB	85 dB	85 dB	85 dB	90 dB	90 dB
Left	85 dB	80 dB	85 dB	90 dB	85 dB	90 dB



Transducer	Insert earphones	
Reliability	Good	
Results:		
	Right	Left
PTA	10 dB HL	90 dB HL
SRT	10 dB HL	75 dB HL
WRS	100% @ 50 dB HL	76% @ 95 dB HL

FIGURE 8.4 Puretone and speech audiometry results for case 3.

TABLE 8.14

Transient-evoked Otoacoustic Emission Results for Case 3

Ear	1,000 Hz	1,400 Hz	2,000 Hz	2,800 Hz	4,000 Hz
Right	9.1 dB	12.7 dB	9.2 dB	10.1 dB	12.4 dB
Left	10.5 dB	10.3 dB	11.4 dB	14.9 dB	13.9 dB

Case 4

Case History

Ms. Ashley Jackson, age 27, has had hearing problems all of her life. She has been told by her audiologist that she has only a mild sensory/neural hearing loss. Her doctor always tells her that her hearing is really very good. She tried hearing aids a few years ago but she says that they did not help at all. Unfortunately, Ms. Jackson cannot hold a job because of her hearing difficulties. Her bosses always cite miscommunication problems as the reason for her dismissal. Ms. Jackson is here today to see if her hearing has changed. Tables 8.15 and 8.16 show otoscopy and tympanometry results. Figure 8.5 shows puretone and speech audiometry results.

Ms. Jackson's puretone results appear to be consistent with the previous hearing tests in her medical record. There are some red flags that warrant additional testing, though. First, her reports of listening difficulties and communication problems in her case history suggest that she may have more than a mild sensory/neural hearing loss. Additionally, her word recognition scores are poorer than expected given her puretone thresholds. You would expect a patient with PTAs in this range to have WRS of 68% or better (Dubno et al., 1995). The next tests that should be performed are ARTs and OAEs. Tables 8.17 and 8.18 show the results of those tests.

Now that you have completed your testing, you cross check your test results. First, the patient's ARTs are not consistent with her puretone thresholds. With a mild sensory/neural hearing loss you would expect acoustic reflexes to be present (Gelfand et al., 1990). The patient's TEOAEs are present and robust which would not be expected based on Ms. Jackson's puretone thresholds. These findings in conjunction with the poor WRS indicate a need for additional testing such as an ABR. You suspect that the patient has ANSD. Results of the ABR and a medical evaluation may help to confirm or rule out your suspected diagnosis. Without the addition of both ARTs and OAEs to the test battery, Ms. Jackson's disorder might have been missed again. The recommendations for patients with ANSD or other retrocochlear pathologies are often very different from the recommendations for those who have a peripheral hearing loss. Misidentification of the site of lesion for a hearing loss like

TABLE 8.15

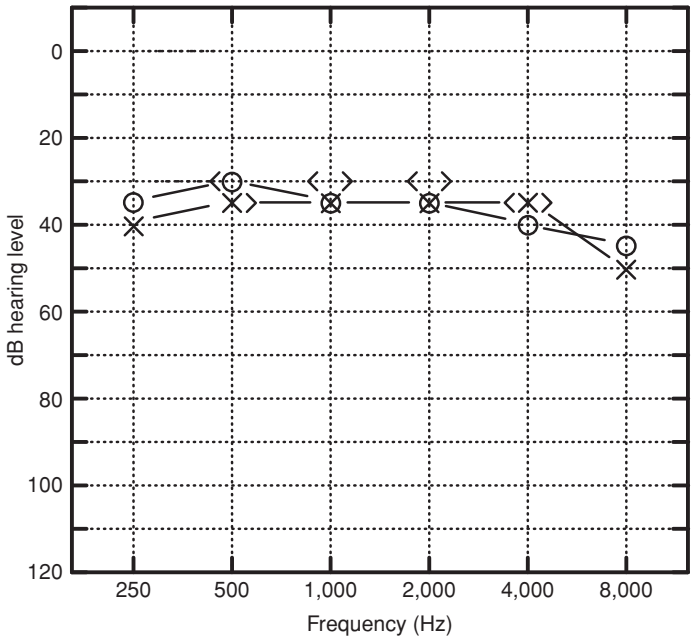
Otoscopy Findings for Case 4

Right Ear	Left Ear
Clear ear canal; intact tympanic membrane	Clear ear canal; intact tympanic membrane

TABLE 8.16

Tympanometry Results (226-Hz Probe Tone) for Case 4

	Right	Left
ECV	1.3 mL	1.4 mL
Compliance	0.7 mL	0.5 mL
Middle-ear pressure	20 daPa	–10 daPa



Transducer	Insert earphones	
Reliability	Good	
Results:		
	Right	Left
PTA	33 dB HL	35 dB HL
SRT	30 dB HL	35 dB HL
WRS	48% @ dB HL	52% @ 75 dB HL

FIGURE 8.5 Puretone and speech audiometry results for case 4.

TABLE 8.17

Acoustic Reflexes Results (in dB HL) for Case 4

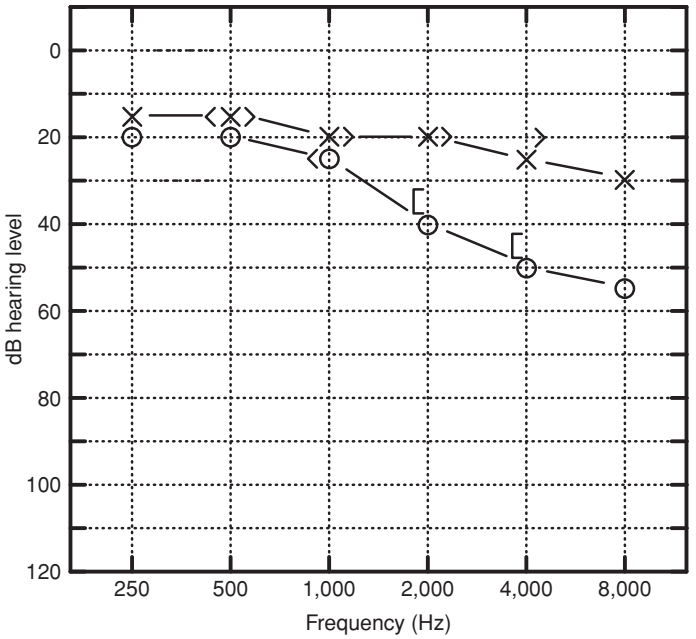
	Ipsilateral			Contralateral		
Stimulus Ear	500 Hz	1,000 Hz	2,000 Hz	500 Hz	1,000 Hz	2,000 Hz
Right	NR	NR	NR	NR	NR	NR
Left	NR	NR	NR	NR	NR	NR

NR, no response to maximum presentation (110 dB HL).

TABLE 8.18

Transient-evoked Otoacoustic Emission Results for Case 4

Ear	1,000 Hz	1,400 Hz	2,000 Hz	2,800 Hz	4,000 Hz
Right	20.1 dB	22.9 dB	19.5 dB	18.4 dB	19.3 dB
Left	22.5 dB	20.6 dB	20.1 dB	22.9 dB	20.3 dB



Transducer	Insert earphones	
Reliability	Good	
Results:		
	Right	Left
PTA	28 dB HL	18 dB HL
SRT	30 dB HL	20 dB HL
WRS	40% @ 70 dB HL	85% @ 60 dB HL
	10% @ 85 dB HL	

FIGURE 8.6 Puretone and speech audiometry results for case 5.

Ms. Jackson's might result in delayed or inappropriate rehabilitation recommendations. Unfortunately, ANSD can be missed easily in clinical practice if the audiologist does not perform a thorough test battery (Smart and Kelly, 2008).

Case 5

Case History

Mr. Don Warner, age 58, is being seen today with his primary complaint being a constant ringing in his right ear. He notes that the ringing has been present off and on for over 3 years but it has become more bothersome recently. When asked about his hearing he admits that he has difficulty understanding what others are saying in noisy places. He denies aural fullness and dizziness. He plays tennis regularly and generally is in great health. Results from your testing are shown in Tables 8.19–8.22 and Figure 8.6.

The order of test administration is important. Because you performed immittance testing and TEOAEs first, you knew that the patient's tympanic membrane is mobile, that the ART pattern is abnormal in the right ear, and that the outer hair cells of the right ear do not appear to be functioning normally. You were able to obtain this information before the patient provided any information through subjective testing. The patient's asymmetry in high-frequency audiometric thresholds and poor WRS in the right ear combined with the objective results suggest a retrocochlear pathology and warrant an ENT referral for additional testing. The patient's report of unilateral tinnitus, the abnormal ART pattern, the asymmetry in puretone thresholds, and the apparent rollover in the patient's right-ear word recognition are all suggestive of retrocochlear dysfunction. Taken in isolation, each might be sufficient for you to make a referral for a medical evaluation. However, having consistent results from several tests allows you to be more confident in your recommendation and provide the ENT with as much information as possible.

TABLE 8.19

Otoscopy Findings for Case 5

Right Ear	Left Ear
Clear ear canal; intact tympanic membrane	Clear ear canal; intact tympanic membrane

Case 6

Case History

Mr. José Gonzalez, age 24, was seen today for an audiologic evaluation. He was just hired as a clerk for a federal judge and therefore has to undergo a rigorous physical examination, including a hearing test. Mr. Gonzalez denies any hearing difficulties, tinnitus, dizziness, or aural fullness. He reports that he is in great health and is currently training for a marathon.

TABLE 8.20

Tympanometry Results (226-Hz Probe Tone) for Case 5

	Right	Left
ECV	1.6 mL	1.8 mL
Compliance	0.7 mL	0.9 mL
Middle-ear pressure	0 daPa	–10 daPa

TABLE 8.21

Acoustic Reflexes Results (in dB HL) for Case 5

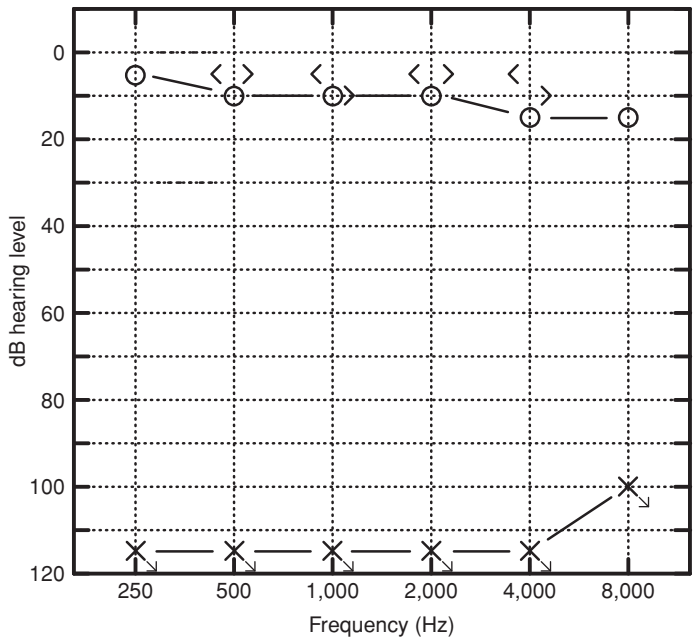
	Ipsilateral			Contralateral		
Stimulus Ear	**500 Hz**	**1,000 Hz**	**2,000 Hz**	**500 Hz**	**1,000 Hz**	**2,000 Hz**
Right	105 dB	110 dB	110 dB	110 dB	NR	NR
Left	85 dB	90 dB	85 dB	95 dB	95 dB	95 dB

NR, no response to maximum presentation (110 dB HL).

TABLE 8.22

Transient-evoked Otoacoustic Emission results for Case 5

Ear	1,000 Hz	1,400 Hz	2,000 Hz	2,800 Hz	4,000 Hz
Right	Absent	Absent	Absent	Absent	Absent
Left	8.0 dB	9.3 dB	9.1 dB	6.2 dB	6.1 dB



Transducer	Supra-aural headphones	
Reliability	?	
Results:		
	Right	**Left**
PTA	10 dB HL	NR
SRT	5 dB HL	NR
WRS	100% @ 45 dB HL	CNT

FIGURE 8.7 Puretone and speech audiometry results for case 6.

TABLE 8.23

Otoscopy Findings for Case 6

Right Ear	Left Ear
Clear ear canal; intact tympanic membrane	Clear ear canal; intact tympanic membrane

TABLE 8.24

Tympanometry Results (226-Hz Probe Tone) for Case 6

	Right	Left
ECV	1.5 mL	1.7 mL
Compliance	0.9 mL	0.7 mL
Middle-ear pressure	–10 daPa	–15 daPa

TABLE 8.25

Acoustic Reflexes Results (in dB HL) for Case 6

	Ipsilateral			Contralateral		
Stimulus Ear	**500 Hz**	**1,000 Hz**	**2,000 Hz**	**500 Hz**	**1,000 Hz**	**2,000 Hz**
Right	85 dB	85 dB	90 dB	95 dB	90 dB	95 dB
Left	85 dB	90 dB	85 dB	90 dB	95 dB	95 dB

TABLE 8.26

Transient-evoked Otoacoustic Emission Results for Case 6

Ear	1,000 Hz	1,400 Hz	2,000 Hz	2,800 Hz	4,000 Hz
Right	12.3 dB	14.6 dB	10.2 dB	11.1 dB	8.9 dB
Left	13.5 dB	12.8 dB	12.4 dB	10.1 dB	9.9 dB

Based on the testing you have completed thus far you would expect this patient has normal hearing. His case history and all objective tests suggest hearing within normal limits. You proceed with your puretone and speech testing.

Results from your testing are shown in Tables 8.23–8.26 and Figure 8.7.

The puretone and speech audiometry results are surprising because they conflict with the results from the objective tests. Specifically, ARTs and TEOAEs within normal limits are not consistent with a measured profound hearing loss in the left ear. Your first thought is nonorganic hearing loss. You decide to reinstruct Mr. Gonzalez and retest the left ear air-conduction thresholds. This time you tell Mr. Gonzalez that you are going to play some more beeps in his left ear and all he has to do is press the button when he hears the tone. He nods and appears to understand the instructions. You begin retesting at 1,000 Hz and Mr. Gonzalez does not respond at the maximum limits of the audiometer. As you enter the booth to reinstruct again, Mr. Gonzalez informs you that he never heard a beep and has been waiting for you to present the tone. In fact, he notes that he has not heard anything from the left earphone. You check the headphone jack connections and find that the left headphone jack is unplugged. After you plug in the jack and retest Mr. Gonzalez's left ear, you obtain thresholds within normal limits. It is important to note that the patient history and the objective test results were not consistent with the subjective test results. Although having a well-constructed test battery is important, you also want to be flexible with your test order and be vigilant to notice inconsistencies between test results as you go. This flexibility would allow you to notice the unplugged headphone jack sooner and save time and frustration for you and Mr. Gonzalez.

LIMITATIONS OF THE AUDIOLOGIC TEST BATTERY

The combination of well-validated test measures, precise patient instruction, careful scoring, and application of the cross-check principle should result in accurate diagnostic and rehabilitative decisions for most patients. It is important to remember, however, that real-world patients usually do not present as textbook cases. The case studies contained in this chapter and the diagnostic criteria published in the audiologic test literature should be treated as guidelines rather than absolute rules. High-quality diagnosis depends on both the construction of a high-quality test battery and skill in interpreting ambiguous or seemingly contradictory test results. A good rule for daily practice is this: When test results seem in disagreement, first check the tester (rule out the clinician's own mistakes); then, check the equipment (rule out malfunction or equipment performing out of calibration); and finally, check the patient (rule out patient error or pseudohypacusis).

MAKING REFERRALS

No audiologist is an island. A team approach to the treatment of hearing and balance disorders, particularly in pediatric patients, is often indicated. Appropriate treatment of a patient seen for audiologic evaluation may require consultation with specialists including (but not limited to) allergists, endocrinologists, neurologists, occupational therapists, ophthalmologists, psychiatrists, rheumatologists, and speech-language pathologists. Referral of pediatric patients with hearing loss to an ophthalmologist is particularly important; approximately 50% of children born with

severe-to-profound hearing loss also have abnormalities of vision (American Academy of Pediatrics, 2007).

Referral for Medical Otolaryngologic Evaluation

The most common referral made by audiologists is to a medical doctor. Sending a patient to an otolaryngologist, primary care physician, or pediatrician is indicated if the audiologic evaluation reveals evidence of an underlying medical condition. Symptoms may include ear pain, bleeding or drainage from the ear (otorrhea), tympanometric abnormality without known etiology, or physical abnormality observed during otoscopy. Patients who report frequent ear infections, fluctuating or sudden hearing loss, or balance disturbance should also be seen by a medical professional (see Table 8.27). Newly identified hearing loss is also reason for referral. Although some audiologists undertake cerumen management in their own practice, many others prefer to refer to an otolaryngologist or the patient's primary care physician for removal of impacted wax. Children who exhibit a previously undiagnosed hearing loss or who exhibit delays in speech or language development should be seen by a pediatric otolaryngologist or developmental pediatrician prior to any audiologic management.

With respect to the audiologic test battery, disagreement among objective and subjective test measures which cannot be resolved as tester, equipment, or patient error is indicative of need for medical referral. Abnormally poor speech scores relative to the audiogram, asymmetric hearing loss, and reports of aural fullness and/or tinnitus are other signs of possible serious ear disease which should be evaluated by a physician.

Referral for Auditory Processing Evaluation

Disagreement between objective and subjective hearing tests may be reason to refer a patient for an evaluation of auditory processing. Patients with apparently normal peripheral auditory function may still have difficulty processing complex signals such as speech. These individuals often report that they can hear well, but have difficulty understanding what others are saying, particularly in the presence of noise. Tests of speech perception in noise such as the Bamford–Kowal–Bench Speech-in-Noise Test (BKB-SIN; Etymōtic Research, 2005), Quick Speech-in-Noise Test (QuickSIN; Etymōtic Research, 2001), and Hearing in Noise Test (HINT; Nilsson et al., 1994) may help to confirm this difficulty. If performance on speech-in-noise tests is poor, particularly if

TABLE 8.27

Seven Signs of Serious Ear Disease

Sign	Possible Etiologies
Ear pain (otalgia) or sensation of fullness	Otalgia may be a result of disease of the ear (e.g., otitis media, otitis externa) or may be referred pain resulting from illness in the head or neck (e.g., temporomandibular joint dysfunction, tumors of the upper digestive tract)
Discharge (otorrhea) or bleeding from the ear	Otorrhea and bleeding may result from chronic otitis media, otitis externa, cholesteatoma, and other disorders of the temporal bone. Bleeding from the ear may also be a sign of traumatic injury to the ear or temporal bone tumor
Sudden or progressive sensory/neural hearing loss, even with recovery	Sudden sensory/neural hearing loss may result from viral infection, ischemic event, trauma, or VIII nerve pathology. Progressive hearing loss is associated with immune disorders and viral or bacterial infections. Fluctuating hearing loss is commonly noted in patients with Ménière's disease
Asymmetric hearing between the ears or tinnitus	Asymmetric hearing loss and/or unilateral tinnitus may be a result of a tumor on the VIII nerve
Hearing loss following injury, exposure to loud sound, or air travel	Blunt or penetrating trauma to the head and barotrauma may result in hearing loss that is conductive (disruption of tympanic membrane and/or ossicular chain) or sensory/neural (disruption of cochlear membranes). Noise-induced sensory/neural hearing loss may be seen after isolated intense sound events (explosions, gunfire) or repeated exposure to loud noise
Slow or abnormal speech development in children	Delayed speech and language development in children is often a result of inability to hear certain sounds of speech. This may result from conductive hearing loss (usually related to otitis media) or permanent sensory/neural loss
Balance disturbance or dizziness	Balance disturbance may be a result of otologic (e.g., Ménière's disease, perilymph fistula) or neurologic disease (e.g., stroke, demyelinating disease)

Adapted from Hall JW III, Mueller HG. (1997) *Audiologists' Desk Reference*. Vol I. San Diego, CA: Singular.

the audiogram is normal or suggests good hearing sensitivity, auditory testing should be performed. Parental concerns about a child's ability to process speech in noisy or reverberant places may also indicate need for APD evaluation. Auditory processing evaluation and rehabilitation are described in Chapters 27 to 30 of this textbook.

Referral for Vestibular Evaluation

Formal vestibular evaluation may be indicated by patient history or results of a doctor's physical evaluation. The symptoms of vestibular dysfunction are often obvious to the patient, but he or she may not realize that they are a relevant part of the audiologic case history. Therefore, it is important for any audiologist's case history form to include questions specifically asking about vertigo or balance problems to elicit this information. Reports of dizziness (particularly recent dizziness), vertigo, or feelings of spinning suggest need for evaluation by a vestibular specialist and/or otolaryngologist. Reports of imbalance (as opposed to vertigo) are also reason for medical evaluation, but may require treatment by a physical therapist rather than an audiologist. Other specific indicators for vestibular testing include history of exposure to ototoxins (particularly vestibulotoxins, such as aminoglycoside antibiotics), bacterial meningitis, or perilymph fistula. Patients with genetic conditions such as Pendred syndrome, Usher syndrome, and CHARGE syndrome are also candidates for vestibular referral. Pediatric patients presenting with inner ear dysplasia of unknown origin or delays in motor or balance skills should also be referred. Vestibular evaluation (Chapter 21) and rehabilitation (Chapter 22) are discussed in detail later in this textbook.

Referral for Genetic Evaluation

Roughly 0.1% to 0.3% of children are born with some hearing loss, and about half of these cases appear to be related to some genetic cause. Of these genetic cases, about 30% are syndromic, meaning that they can be related to sets of clinically recognizable features or symptoms known to co-occur. The remaining 70% of genetic hearing loss cases are characterized by hearing loss in isolation and are referred to as nonsyndromic. Approximately 50% of cases of autosomal recessive nonsyndromic hearing loss are due to mutation in gap junction beta-2 (*GJB2*), the gene that encodes the gap junction protein connexin 26 (CX26) (Kelsell et al., 1997). Hearing loss resulting from connexin 26 mutation is typically present from birth and can range in severity from moderate to profound. More than 90 mutations of *GJB2* have been identified.

Testing for *GJB2* mutation is an emergent field in early hearing loss identification. Quick and low-cost screening methods have been developed and are available through many medical centers and genetic testing service providers. It should be noted that many patients and parents may be hesitant to undergo formal genetic testing because of fears that their health insurance costs may increase if a genetic predisposition to disease is found. For these patients, a consultation with a genetic counselor may be preferable to a referral to a medical geneticist.

SUMMARY

A well-constructed and consistently administered test battery provides the foundation for high-quality audiologic diagnosis and care. The case studies contained within this chapter are intended to underscore the importance of the diagnostic battery in terms of test selection, test order, and use of cross-checks. When test discrepancies cannot be resolved or a patient presents with complaints or symptoms outside of the audiologist's scope of practice, a referral to an appropriate specialist is indicated. Consultation with other specialists can also help the audiologist form a more complete picture of a patient's hearing health, increasing the likelihood of success in audiologic or vestibular rehabilitation.

FOOD FOR THOUGHT

1. For each of the cases presented in this chapter, what are some ways that the test order may have affected your thought process regarding the potential diagnoses?
2. How might you modify the order that you administer tests in your test battery, or change particular tests, based on individual patient factors such as age or cognitive ability?
3. Imagine that you notice a colleague in your clinic administering tests in an order that you think is unhelpful or omitting tests that would help to differentiate between possible diagnoses. What might you say or do to suggest a different test battery or test sequence? What evidence might you use to support your suggestion?

REFERENCES

American Academy of Pediatrics, Joint Committee on Infant Hearing. (2007) Year 2007 position statement: Principles and guidelines for early hearing detection and intervention programs. *Pediatrics.* 120, 898–921.

American Speech-Language-Hearing Association. (1988) Determining threshold level for speech [Guidelines]. Available online at: www.asha.org/policy.

Bachmann KR, Hall JW III. (1998) Pediatric auditory brainstem response assessment: The cross-check principle twenty years later. *Semin Hear.* 19 (1), 41–60.

Baldwin SM, Gajewski BJ, Widen JE. (2010) An evaluation of the cross-check principle using visual reinforcement audiometry, otoacoustic emissions, and tympanometry. *J Am Acad Audiol.* 21, 187–196.

Berlin CI, Hood LJ, Morlet T, Wilensky D, Li L, Mattingly KR, Taylor-Jenafreau J, Keats BJB, St. John P, Montgomery E, Shallop JK, Russell BA, Frisch SA. (2010) Multi-site diagnosis

and management of 260 patients with auditory neuropathy/dys-synchrony (auditory neuropathy spectrum disorder). *Int J Audiol.* 49, 30–43.

Dubno JR, Lee F, Klein AJ, Matthews LJ, Lam CF. (1995) Confidence limits for maximum word-recognition scores. *J Speech Hear Res.* 38, 490–502.

Etymōtic Research. (2001) *Quick Speech-in-Noise Test.* Elk Grove Village, IL: Etymōtic Research.

Etymōtic Research. (2005) *BKB-SIN™ Speech-in-Noise Test.* Elk Grove Village, IL: Etymōtic Research.

Gelfand SA, Schwander T, Silman S. (1990) Acoustic reflex thresholds in normal and cochlear-impaired ears: Effects of no-response rates on 90th percentiles in a large sample. *J Speech Hear Disord.* 55, 198–205.

Gravel JS. (2002) Potential pitfalls in the audiological assessment of infants and young children. In: Seewald RC, Gravel JS, eds. *A Sound Foundation through Early Amplification 2001: Proceedings of the Second International Conference.* Chicago, IL: Phonak AG; pp 85–101.

Hall JW III. (2010) Aural immittance measures are more useful now than ever. *Hear J.* 63 (4), 10, 12, 14–15.

Hall JW III, Bondurant LM. (2009) Neuro-diagnostic paediatric audiology. In: Newton VE, ed. *Audiological Medicine.* 2nd ed. West Sussex, UK: John Wiley & Sons; pp 72–89.

Hall JW III, Mueller HG. (1997) *Audiologists' Desk Reference.* Vol I. San Diego, CA: Singular.

Jerger J, Burney B, Mauldin L, Crump B. (1974) Predicting hearing loss from the acoustic reflex. *J Speech Hear Disord.* 39, 11–22.

Jerger JF, Hayes D. (1976) The cross-check principle in pediatric audiometry. *Arch Otolaryngol.* 102, 614–620.

Kelsell DP, Dunlop J, Stevens HP, Lench NJ, Liang JN, Parry G, Mueller RF, Leigh IM. (1997) Connexin 26 mutations in hereditary non-syndromic sensorineural deafness. *Nature.* 387 (6628), 80–83.

Killion MC, Niquette PA. (2000) What can the puretone audiogram tell us about a patient's SNR loss? *Hear J.* 53, 46–53.

Littman TA, Magruder A, Strother DE. (1998) Monitoring and predicting ototoxic damage using distortion-product otoacoustic emissions: pediatric case study. *J Am Acad Audiol.* 9, 257–262.

Madell JR, Flexer CA. (2008) *Pediatric Audiology: Diagnosis, Technology, and Management.* New York, NY: Thieme.

Nilsson M, Soli S, Sullivan JA. (1994) Development of the Hearing in Noise Test for the measurement of speech reception thresholds in quiet and in noise. *J Acoust Soc Am.* 95, 1085–1099.

Smart JL, Kelly AS. (2008) Searching for an answer: auditory neuropathy/auditory dys-synchrony, a 20 year long mystery solved. *N Z Audiol Soc Bull.* 18, 25–33.

Stach BA, Wolf SJ, Bland L. (1993) Otoacoustic emissions as a cross-check in pediatric hearing assessment: Case report. *J Am Acad Audiol.* 4, 392–398.

Stapells DR. (2011) Frequency-specific threshold assessment in young infants using the transient ABR and the brainstem ASSR. In: Seewald RC, Tharpe AM, eds. *Comprehensive Handbook of Pediatric Audiology.* San Diego, CA: Plural; pp 409–448.

Turner RG. (2003) Double checking the cross-check principle. *J Am Acad Audiol.* 14, 269–277.

Wiley TL, Stoppenbach DT, Feldhake LJ, Moss KA, Thordardottir ET. (1995) Otoacoustic emissions as a cross-check in pediatric hearing assessment: case report. *Am J Audiol.* 4, 26–34.

SECTION II

Physiological Principles and Measures

CHAPTER 9

Tympanometry and Wideband Acoustic Immittance

Lisa L. Hunter and Chris A. Sanford

INTRODUCTION

Tympanometry is one of the most frequently performed and important components of the basic audiologic evaluation. Tympanometry measures how the middle-ear system responds to sound energy and how it reacts dynamically to changes in atmospheric pressure. Because sounds must be transmitted from a low-impedance air medium in the ear canal to a higher impedance fluid medium (the labyrinth) for humans to hear, it is important to understand whether the middle ear is providing effective sound transmission. In fact, without the middle ear, humans would only be able to hear very loud sounds via bone conduction, since soft to moderate sounds would be reflected back by the tissues and bone of the head.

The middle ear acts as an "impedance matching" system, allowing sounds to be transmitted more effectively through an elaborate system of levers via the ossicles and by concentrating sound pressure, since the round window area is smaller than the tympanic membrane (TM). However, not all sounds are transmitted equally by the middle ear. Low-frequency sounds below 1,000 Hz and high-frequency sounds above 4,000 Hz are transmitted less efficiently. Thus, the filtering of sounds by the middle-ear system largely determines our hearing sensitivity for different frequencies. The middle ear transmits the most energy to the cochlea in the frequency range of 1,000 to 4,000 Hz and is matched to the frequency region in which the majority of speech cues are carried.

This chapter provides a review of principles of tympanometry, discussion of single-frequency tympanometry across the age span from infancy to adults, principles of multiple frequencies and subcomponents of tympanometry, new advances in wideband (WB) tympanometry, and applications of tympanometry in cases of middle-ear pathology. The overall goal of this chapter is to provide a foundation for graduate students in audiology to understand the principles and applications of basic and advanced tympanometry measures and to provide the practicing clinician with an update on newer measures and recent clinical research evidence for putting these advances into everyday practice.

OVERVIEW OF TYMPANOMETRY

Tympanometry is an objective, physiological measure of acoustic admittance of the middle ear as a function of air pressure in a sealed ear canal. Normally, our ears operate most efficiently at atmospheric or ambient pressure. Clinically, it is of interest to measure middle-ear function at greater and lesser pressures compared to ambient pressure for diagnostic purposes because many conditions can affect pressure within the middle ear. When pressure is varied over a range of positive to negative pressure compared to atmospheric pressure, the effect on middle-ear function can be observed graphically. Increases or decreases in air pressure cause the TM and ossicular chain to stiffen, and this change can be seen as a decrease in admittance of sound energy to the middle ear, as shown in Figure 9.1. This figure also illustrates the effect of varying pressure in the ear canal on distension or contraction of the ear canal and TM. The most efficient operating point in terms of ear canal air pressure is observed as a peak in the tympanogram. The most common tympanometric measurement is peak height or "static admittance," which is a measure of the amount of acoustic energy that flows into the middle-ear system.

To obtain a tympanogram, a calibrated probe stimulus (either puretones or WB clicks) is presented to the outer ear canal with a microphone. Air pressure in the ear canal is varied above and below atmospheric (ambient) pressure, which causes the TM and ossicular chain to stiffen. As the air pressure is increased or decreased in the ear canal, the admittance flowing into the middle ear is decreased, so more sound pressure remains in the ear canal. At the microphone, this is read as an increase in probe sound pressure level. If you perform tympanometry in your own ear, listen carefully as the air pressure is varied—both positive and negative. You will hear a decrease in sound intensity as the pressure is increased or decreased, and you should hear an increase in sound intensity at the peak of the tympanogram, where admittance is greatest.

A normal tympanogram has a single clearly defined peak occurring near atmospheric pressure, as in Figure 9.2, type A. Problems in the middle ear cause alterations in the shape of the tympanogram. For example, the most common pathology that affects tympanometry is fluid in the middle ear space, or otitis media with effusion (OME). This condition

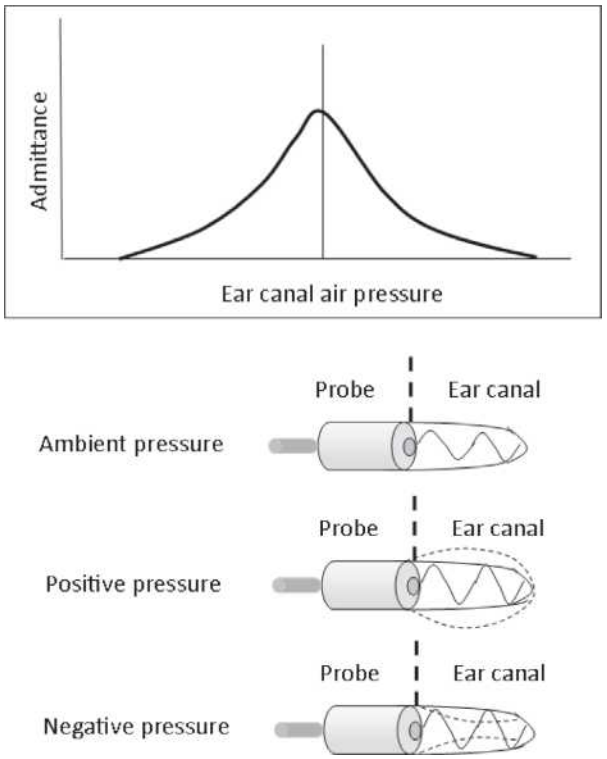


FIGURE 9.1 Effects of air pressure on the external ear canal and tympanic membrane as pressure is varied from ambient, to positive, and then to negative pressure. As the pressure is varied in the sealed ear canal, admittance is plotted as a function of ear canal air pressure on a tympanogram (*upper panel*). The *lower panels* show effects of inducing positive and negative pressure in the ear canal on the external walls, ear canal volume, and volume of probe tone sound pressure level in the ear canal.

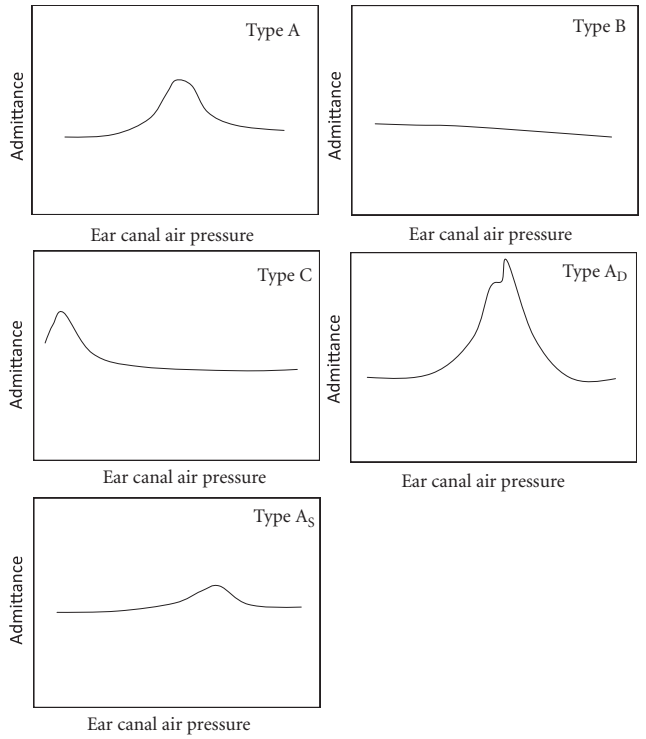


FIGURE 9.2 Lidén–Jerger classification scheme for tympanometric shapes, based on qualitative analysis of the height and location of the primary peak. (Modified from Lidén G. (1969) The scope and application of current audiometric tests. *J Laryngol Otol.* 83, 507–520 and Jerger J. (1970) Clinical experience with impedance audiometry. *Arch Otolaryngol.* 92, 311–324).

results in an increase in impedance (decreased admittance), which broadens or flattens the tympanogram, as shown in Figure 9.2, represented by type B. In severe cases, the tympanogram will be completely flat across all air pressures, indicating that the TM is stiffened and unable to transmit sound energy effectively at any air pressure. Another common condition, Eustachian tube (ET) dysfunction, causes middle-ear pressure to be decreased relative to atmospheric pressure, shown as type C. In ET dysfunction, the most effective energy transfer point is usually more negative, which shifts the peak of the tympanogram to the left. Rarely, positive pressure can be present in the middle-ear space, usually due to acute otitis media (AOM). In these cases, the tympanogram peak will be shifted to the right. In cases of a thinned tympanic membrane (TM) or ossicular discontinuity, decreased stiffness or an increase in peak height can occur because of TM thinning or ossicular discontinuity. In such cases, the height of the admittance tympanogram will be increased relative to normal, as in type A_D. Conversely, increased stiffness can occur in a variety of disorders from scarring of the tympanic membrane to otosclerosis and can reduce the peak height of the tympanogram, as shown in type A_S.

Table 9.1 provides categories for interpreting tympanometry results with respect to potential pathology. Tympanometry is widely variable in both normal and abnormal ears, thus caution should be used in inferring the pathology from tympanometry alone. Tympanometry should be combined with an otolaryngology examination and history to maximize its use as a diagnostically useful tool.

HISTORY OF TYMPANOMETRY

Tympanometry was invented by Terkildsen and Scott-Nielson in the form of an electroacoustic device that measured the admittance of the human ear across a range of sound pressures introduced into the sealed outer ear canal by a pressure pump. Terkildsen and Thomsen (1959) published the first tympanogram using the first commercially available aural acoustic immittance instrument, the Madsen ZO61. The ZO61 used a very low-frequency input puretone probe of 220 Hz with admittance measured as a function of ear canal air pressure. Amplitude and phase required to counterbalance the input sound were read on a voltmeter. The choice of a low-frequency probe tone (220 Hz) was partly at random, but also chosen to avoid high-frequency microphone artifacts and lower frequency electrical line

TABLE 9.1

Effects of Middle-Ear Pathologies

	Y	TW	Volume	Mass	Springiness	Resonant Frequency
Middle-ear effusion	Low	Wide	Normal	↑	↓	Low
Monomer or ossicular discontinuity	High	Narrow	Normal	↑	↑	Low
Perforation	Flat or variable	–	High	–	–	–
Tympanosclerosis	Normal to low	Normal	Normal	↑	↓	Low
Cholesteatoma	Low	Wide	Normal		↓	Low
Lateral ossicular fixation	Low	Wide	Normal	–		High
Medial ossicular fixation (otosclerosis)	Normal	Normal/narrow	Normal		↓	Normal to high

Reprinted with permission from Hunter and Shahnaz. (2014) Table 4-1, pg. 45.

noise; it was also chosen for the sake of calibration ease. Terkildsen and Thomsen noted that for the relatively small size of the human ear canal, a low-frequency probe tone results in smaller calibration errors than higher frequency probe sources. Indeed, this problem of artifacts at higher probe tones has limited the frequency range to 1,000 Hz or below for single frequency tympanometry. Terkildsen and Thomsen published tympanograms obtained in normal ears and in ears with TM atrophy and otitis media.

In 1963, the more widely used Madsen ZO70 model appeared. The ZO70 model did not employ a phase adjustment and provided magnitude information in "compliance units" that were not calibrated in measurement units. The choice of the term "compliance" reflected the primary contribution of springiness, or compliance, in normal adult ears at 220 Hz. The use of "compliance units" became standard in clinical practice and most published literature. Because the uncalibrated compliance units could not be averaged or subjected to statistical comparison, the first classification schemes published were based on qualitative tympanometry shapes (A, B, and C shapes).

In 1973, an innovation in tympanometry appeared with the Grason Stadler Model 1720 otoadmittance meter, the first multicomponent and multifrequency tympanometer. This system separately measured subcomponents that contribute to total admittance. Two meters indicated susceptance and conductance, abbreviated as *B* and *G,* respectively. Susceptance includes both mass and spring components of admittance, whereas conductance includes just the friction component. This instrument provided 220-Hz as well as 678- and 1,000-Hz probe tones, which made it the first commercial multifrequency tympanometer. Importantly, this two-component admittance measuring technique allowed ear canal contributions to the measured response to be easily subtracted from the total admittance. Also, the GSI 1720 measured calibrated units, called millimhos (mmho), rather than arbitrary "compliance units." These systems were used in many multicomponent and multifrequency tympanometry studies, as will be described later in this chapter.

Until 1987, immittance instruments, definitions, and terms were variable and there was no accepted standard. The first ANSI standard was published in 1987 with the goal of standardizing instrumentation, terminology, and recording practices. This allowed reporting of results across clinics so that data could be easily interpreted. The terms used in this chapter conform to the current ANSI standard for immittance instruments (S3.39, revised 2012).

More recently, innovations in tympanometry have taken the form of PC-based handheld clinical admittance systems that measure low-frequency, single-component tympanometry, as well as higher frequency (660, 800, and 1,000 Hz) and multicomponent (susceptance and conductance) tympanometry, as will be discussed in later sections.

Clinical research using tympanometry has been strongly influenced by the original choice of the 220- or 226-Hz probe tone as the standard. As a result, most clinical studies have focused on the low-frequency puretone probe. However, beginning with the early studies of tympanometry, there was recognition that higher frequencies produced different tympanometry patterns. In fact, the original publication by Lidén et al. (1970) provided results for both 220 and 800 Hz. At 220 Hz, three primary shapes were observed (A, B, C). At 800 Hz, two additional shapes were seen in pathologic ears—a broad double-peaked shape for ears with ossicular discontinuity and post-stapedectomy and a sharply notched pattern for eardrum atrophy or scarring. These same ears showed single peaks at 220 Hz. The reason for the difference was later determined to be due to stiffness effects at low frequencies and mass effects at high frequencies through modeling work by Vanhuyse et al. (1975).

Newer multifrequency and wideband (WB) immittance systems are now commercially available, and more clinical data using these instruments have recently been published that provide clinicians with more normative references, compared to results in various ear pathologies. WB immittance will be described later in this chapter along with normative ranges compared to results from ears with middle-ear disorders.

After the publication of the ANSI (1987) standard, manufacturers began to conform to the recommendation that immittance instruments provide calibrated physical units of measurement rather than arbitrary compliance units. Virtually all immittance instruments produced since then have been calibrated admittance meters. Quantitative analysis of tympanograms is preferable, especially when assessing infants and children, for whom different age-based normative values are needed. Judging tympanometric shape provides an overall impression, but to distinguish normal from abnormal tympanometry, four basic tympanometric measurements are required. These measurements are (1) equivalent ear canal volume (V_{ea}); (2) static-compensated acoustic admittance (Y_{tm}); (3) tympanometric peak pressure (TPP); and (4) tympanometric width (TW) or gradient.

Tympanometric Shape

Qualitative and quantitative approaches have been used in the interpretation of 226-Hz tympanograms. Since early instruments were uncalibrated and presented tympanometric results as arbitrary compliance units, qualitative measurements were necessary to describe tympanogram shapes. The most popular of these was the classification scheme originally described by Lidén (1969) and Jerger (1970). As shown in Figure 9.2, tympanograms using the Lidén–Jerger classification scheme are typed according to the height and pressure range of the tympanogram peak. Type A tympanograms have normal admittance and tympanometric peak pressure. Type B tympanograms have abnormally low admittance with no discernible peak. Type C tympanograms have normal admittance, with a peak occurring at negative middle-ear pressure. Lidén also described a type D tympanogram characterized by a double peak. Later, Feldman (1976) described subtypes A_D and A_S indicating abnormally high admittance and low admittance respectively. Although the qualitative classification approach is useful for identifying abnormal tympanometric features, and simplifies interpretation, its lack of precision can lead to diagnostic errors and misinterpretations. Without objective criteria for classification, there can be substantial clinical variability in distinguishing among types A, A_D, and A_S. Even distinguishing between types B and A is problematic when small or broad peaks occur, or shifts in the positive compared to the negative tails occur.

The following sections describe specific measures, which provide quantitative analysis of tympanometry. Use of these measures is recommended to clearly identify normal versus pathologic cases.

Equivalent Ear Canal Volume (V_{ea} or V_{ec})

Before performing tympanometry, the audiologist should examine the ear canal with otoscopy to identify cerumen blockages, foreign bodies, drainage, TM perforation, or a collapsed canal. Any of these conditions can affect estimates of ear canal volume and other tympanometry measurements and thus should be documented. In the case of active drainage, it is best to defer tympanometry and refer the patient for medical assessment by a physician since the drainage can affect measurements, could transfer bacteria to the opposite ear or to other. Generally, if an open path to the TM can be visualized, cerumen blockages of less than 50% do not affect tympanometry measurements, although the volume will be less than for a clear ear canal.

The purpose of tympanometry is to accurately estimate the middle-ear admittance under varying ear canal air pressure. Because the probe tip of the admittance measurement system is remote from the surface of the TM, admittance measured at the probe tip reflects the combined admittance of the external auditory canal and the admittance of the middle ear. Accuracy of the middle-ear admittance estimate relies on obtaining an accurate estimate of the "equivalent" ear canal admittance (volume). Because the admittance of the volume of air in the ear canal contributes to the total middle-ear admittance measurement it must be subtracted out to determine the admittance because of the middle ear alone. This process is called tympanometric "compensation" and is used to the determine admittance of the middle ear at the plane of the TM (Y_{tm}) as described in the following section. Figure 9.3 illustrates an admittance tympanogram and how the ear canal volume is compensated using a baseline method by subtracting out the admittance at either the positive or the negative "tail" of the tympanogram. The ear canal volume, referred to as V_{ea}, is affected by numerous factors such as the depth of insertion of the probe tip, the dimensions of the ear canal, and the amount of volume occupied by cerumen. The equivalent volume has also been referred to as V_{ec} or V_{eq}.

Most clinical immittance units provide a *baseline correction* feature when measuring tympanometry. Baseline correction subtracts the equivalent ear canal volume so that the tympanogram is plotted with admittance starting at 0 mmho at the positive or negative tail, depending on the instrument setting. The tympanogram shown in Figure 9.3 is plotted without baseline compensation. Baseline compensation may be done at either an extreme positive value, usually +200 daPa, or an extreme negative value, such as −400 daPa. At these extreme pressure values, it is assumed that the middle ear is sufficiently stiff to cause a decrease of the admittance of the middle ear close to zero. Therefore, assuming that the ear canal walls are rigid, the admittance measured at the probe tip could be attributed only to the air trapped in the ear canal itself. This measure is called "equivalent ear canal volume" because under standard reference conditions using a probe tone of 226 Hz, the volume of trapped air in a hard-walled cavity is equal to the acoustic volume of that same cavity. In other words, 1 cubic

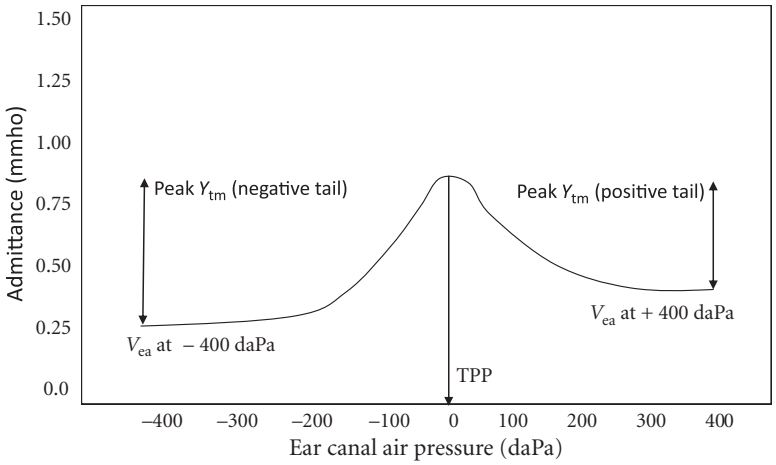


FIGURE 9.3 A normal 226-Hz admittance tympanogram. TPP, tympanometric peak pressure; Y_{tm}, peak-compensated static admittance; either positive or negative tail pressure values are used to compensate for ear canal volume [V_{ea}].

centimeter (cc) or milliliter (mL) of trapped air equals the acoustic admittance of 1 mmho in a hard-walled cavity. This equivalency is the reason that some tympanometers measure admittance in cc or mL. For the sake of clarity, volume units should be used to when report V_{ea} (i.e., cc or mL) and acoustic admittance units (mmho) to report static admittance (Y).

Tympanograms are normally asymmetric, so that the negative tail value falls slightly below the positive tail, thus the volume estimate at extreme negative pressures is typically lower than the volume estimate at extreme positive pressures (Margolis and Shanks, 1985). This asymmetry is due to lower conductance at extreme negative pressures than at extreme positive pressures (Margolis and Smith, 1977). Physical volume differences also occur when positive air pressure pushes inward on the TM and as negative pressure pulls outward on the TM. If the negative tympanogram tail sharply descends below the positive tail, approaching 0 cc at the negative tail value, this usually indicates an ear canal collapse. Ear canal collapse occurs most frequently in newborns and infants due to highly compliant ear canals.

Actual physical volume of adult ear canals was measured by Shanks and Lilly (1981) by filling the ear canal with alcohol and then comparing the measured volumes to tympanometric estimates. The volume of the trapped air is more accurately estimated from the negative tail than from the positive tail, and at a probe frequency of 660 Hz rather than at 226 Hz. The V_{ea} estimated from either the positive or the negative tail value is always greater than the actual ear canal volume. Despite these known differences, clinical measurements of V_{ea} are most commonly taken from the admittance positive tail with a 226-Hz probe tone, which overestimates ear canal volume by about 40% (Shanks and Lilly, 1981). The normal range for V_{ea} is positively related to age because of increases in ear canal volume. A study by Margolis and Heller (1987) reported an average increase in ear canal volume from 0.63 in children to 1.46 in adults. Equivalent ear canal volume is larger in males than females (Roup et al., 1998; Shahnaz and Davies, 2006) because males generally have a larger overall body size compared to females, and body size is correlated with ear canal size (Shahnaz and Davies, 2006). Table 9.2 provides normative values for V_{ea} for adults according to gender for V_{ea} measurement.

To interpret the V_{ea} measurement, the clinician must ensure that the probe tip is not blocked and perform otoscopy to determine if the TM is intact. In cases of blocked probes, cerumen impaction, or OME, tympanograms are generally flat or rounded. Tympanograms in ears with TM perforations are not usually flat, but may have irregular curves because of the geometry of the middle-ear space and effects of air pressure on ET opening. To interpret tympanograms when flattened or irregular shapes are obtained, it is necessary to compare V_{ea} to age-appropriate normative values. Equivalent volumes that are smaller than the lowest expected value for age may indicate a blockage of the probe tip or the ear canal. Blockages most commonly occur because of cerumen impaction or a probe tip angled against the ear canal wall. Figure 9.4 illustrates tympanograms that could occur in the same ear because of different conditions, and thus, result in different V_{ea} measurements.

Peak-Compensated Static Acoustic Admittance (Y_{tm})

Static admittance is the most often measured feature of the 226-Hz tympanogram and is commonly referred to as "compliance." This older term is inaccurate, since admittance tympanometry includes not only compliance, but also mass and resistance. Although it is true that in normal adult ears at 226 Hz, compliance is the most dominant component, this is not the case for infants or in pathologic conditions. Static admittance is lower in middle-ear conditions that increase stiffness of the middle ear, including OME, cholesteatoma and ossicular adhesions, and space occupying lesions of the middle ear that contact the TM or ossicular chain. Conversely, in conditions that decrease stiffness, such as TM atrophy, ossicular disarticulation, or post-stapedectomy, static

TABLE 9.2

Normative Values for Tympanometry Measurements at 226 Hz for Adults

Study	Gender (N)		Y_{tm} (SA) + mmho	TW + daPa	TPP daPa	V_{ea} + (cm^3)
Roup et al. (1998)	M	Mean	0.87	59.8	–26.18	1.40
20–30 yr	*N* = 51	SD	0.46	17.3	31.66	0.32
		90% Range	0.30–1.80	35.0–87.0	–110.00 to 9.0	1.00–2.10
	F	Mean	0.58	73.9	–27.75	1.18
	N = 51	SD	0.27	17.2	23.50	0.22
		90% Range	0.30–1.12	45.0–107.0	–80.0 to –3.0	0.80–1.60
	Overall	Mean	0.72	66.9	–29.96	1.29
	N = 102	SD	0.40	18.6	27.76	0.29
		90% Range	0.30–1.19	32.8–95.0	–103.50 to 4.2	0.90–1.80
Wiley et al. (1996)	M	Mean	0.72	73		1.49
48–90 yr	*N* = 825	SD				
		90% Range	0.2–1.60	35–125		1.0–2.20
	F	Mean	0.62	76		1.28
	N = 1,322	SD				
		90% Range	0.2–1.40	40–120		0.9–1.90
	Overall	Mean	0.66	75		1.36
	N = 2,147	SD				
		90% Range	0.2–1.50	35–125		0.9–2.0

Peak-compensated static admittance, Y_{tm}; tympanometric width, TW; tympanometric peak pressure, TPP; equivalent ear canal volume, V_{ea}.

admittance is higher. Normative values and cutoff criteria for adults are provided in Table 9.2.

As discussed in the previous section, compensation at the extreme positive or negative pressure baseline is used to derive the V_{ea}. The peak of the tympanogram after subtraction of V_{ea} is called the "peak-compensated static acoustic admittance" or, more commonly, static admittance (Y_{tm}). Y_{tm} is derived through the formula $Y_{peak} - Y_{+400}$ for the positive tail method or $Y_{peak} - Y_{-400}$ for the negative tail method, as shown in Figure 9.3.

Measurement of static admittance can be affected by different procedural variables. One variable which has a very clear effect is the choice of pressure value for compensation of ear canal volume. The compensated static admittance is typically higher when extreme negative (rather than extreme positive) pressure is used to estimate ear canal volume because of tympanometric asymmetry (Margolis and Smith, 1977; Shanks and Lilly, 1981). Other procedural variables that affect static admittance are pump speed, direction of pressure change, and repeated tests in close succession. Faster pump speeds produce higher static admittance, whereas decreasing the pressure from positive to negative produces lower static admittance than recording in the opposite direction.

Another procedural variable that can affect static admittance is whether the measurement is taken directly

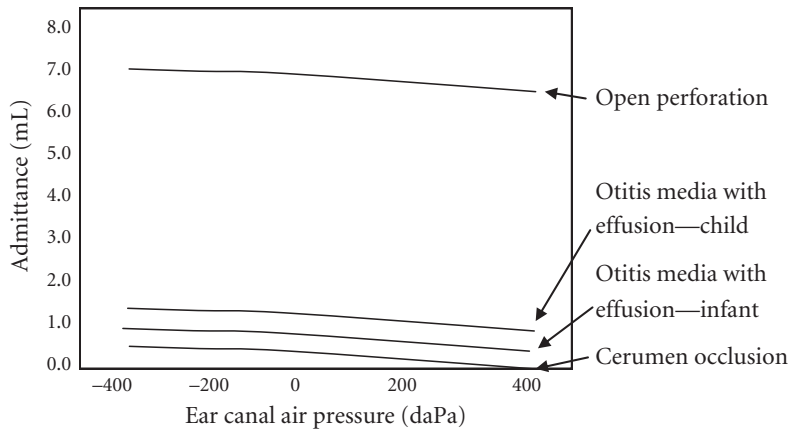


FIGURE 9.4 Uncompensated tympanograms in various conditions, illustrating the effect of pathology on ear canal volume measurements.

from the admittance tympanogram or from the rectangular subcomponents (susceptance and conductance). Admittance is a vector quantity with both magnitude and phase, so it cannot be added or subtracted unless the phase angle of the two admittance values is similar. In adults at low frequencies, this assumption generally holds, but at higher frequencies and in young infants, this assumption is not valid. Therefore, at higher probe tone frequencies, it is necessary to convert admittance vector data to rectangular form, compensate for the effect of ear canal from admittance rectangular components (susceptance and conductance), and then convert the data back to admittance (see Hunter and Shahnaz, 2014 for further details and formulas for this calculation).

Tympanometric Gradient and Width (TW)

Sharpness of the tympanometric peak is associated with middle-ear pathology and is more sensitive to middle-ear effusion (MEE) than static admittance (Nozza et al., 1992, 1994). Two closely related measures of the sharpness of the tympanogram are the TW and gradient. Both measures provide an index of the shape of the tympanogram in the vicinity of the peak and quantify the relative sharpness (steepness) or roundness of the peak. The presence of MEE decreases the gradient and increases the width of the tympanogram.

The preferred and simpler measure is TW, which is measured by drawing a horizontal line halfway between the peak admittance and the baseline. The intersection of this line with either side of the tympanogram is the width, measured in daPa (de Jonge, 1986; Koebsell and Margolis, 1986). Calculation of TW is illustrated in Figure 9.5. A large TW is measured when the tympanogram is rounded and a small TW results when the tympanogram has a sharp peak. Normative values and cutoff criteria for adults are provided in Table 9.2.

An alternative measure of sharpness, tympanometric gradient, is a ratio measure of the steepness of the slopes on either side of the tympanometric peak. A method for measuring gradient was first proposed by Brooks (1968). There are several methods for calculating gradient, but the most common is to calculate the difference in acoustic admittance at the peak and the average of the acoustic admittance at +50 and −50 daPa relative to the acoustic admittance at peak pressure. A line is drawn across the tympanogram at this average admittance (A), and then A is divided by the peak height of either the positive or negative tail. This method is shown in Figure 9.6. The gradient is an index that ranges from 1.0 (flat tympanogram) to very high values depending on the value at TPP. The higher the gradient, the sharper and more narrow the tympanogram.

Two studies have compared gradient measures obtained with the various techniques in normal children and adults (de Jonge, 1986; Koebsell and Margolis, 1986). These studies concluded that the preferred method is TW rather than gradient, as the latter is highly correlated with static admittance and therefore redundant with static admittance. TW is also more straightforward to calculate, making it easy to determine even if the instrument does not provide automatic calculation.

Tympanometric Peak Pressure

The ET serves the important function of regulating pressure within the middle ear and thus protecting the eardrum, ossicles, and cochlea from extreme changes in pressure that could cause tissue damage. When the ET is not functioning normally, negative or positive pressure may develop within the middle ear. This condition is called ET dysfunction and will have the effect of stiffening the ossicular chain and the eardrum. Thus, the most effective operating point of the middle ear will not be at atmospheric pressure, but rather near the pressure contained within the middle ear. In cases of extreme negative pressure or a middle ear filled with

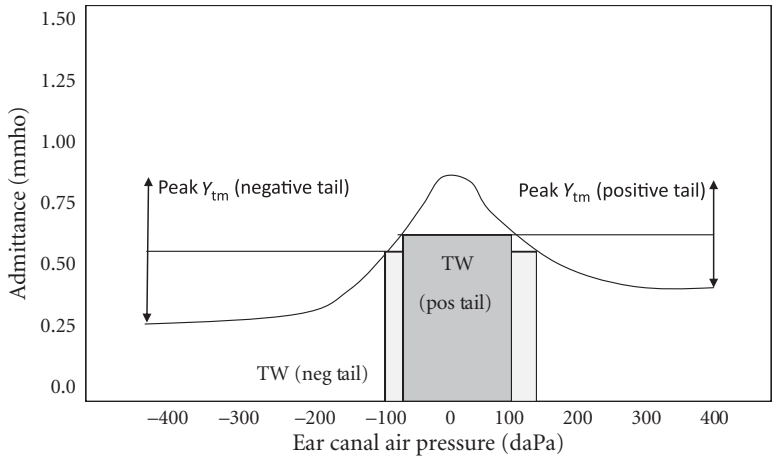


FIGURE 9.5 Calculation of tympanometric width (TW) in daPa from peak-compensated admittance (Y_{tm}). TPP, tympanometric peak pressure; Y_{tm}, peak-compensated static admittance. Refer to text for measurement details.

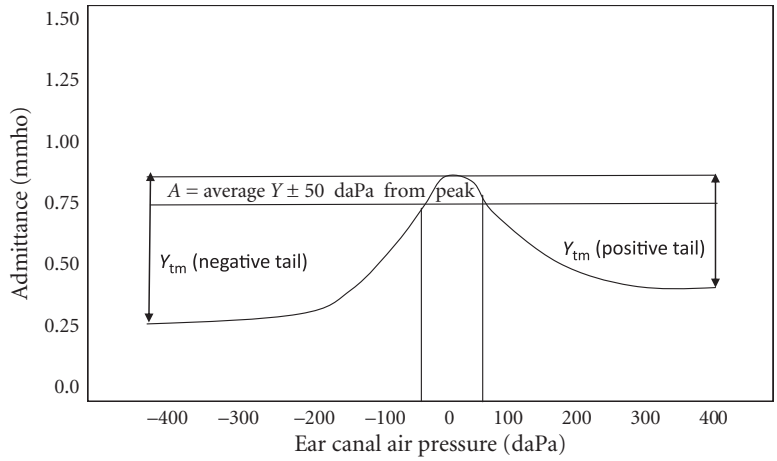


FIGURE 9.6 Gradient measurement of the tympanogram. The distance from the peak to the average at ±50 daPa from the peak on either side is denoted as *A*. The height (*A*) is then divided by the compensated peak static admittance. This measurement results in a ratio from 0 to 1.0. Further measurement details are provided in the text.

fluid, the tympanogram will not show an effective operating point or peak in immittance.

Related to ET function, the ear canal air pressure at which the peak of the tympanogram occurs is the TPP, as was shown in Figure 9.3. Because Y_{tm} reaches its highest value when the pressures on both sides of the TM are equal, TPP is an indicator, but not a direct measure, of the pressure in the middle-ear space. In fact, TPP overestimates the actual middle-ear pressure by as much as 100%. A TPP of −300 daPa, for example, could occur with actual middle-ear pressure of only −150 daPa. The value of measuring TPP is that it can detect the presence of negative or positive middle-ear pressure because of ET dysfunction. Positive pressure may occur in the early stages of acute otitis media (AOM) because of the production of gases secondary to inflammation of the mucosal lining of the middle ear. As the infection progresses, the inflammation results in swelling of the mucosa and production of mucous, known as OME. Because of the swelling, the ET is blocked and the fluid cannot naturally drain out of the ear into the throat; thus, a negative TPP develops. As the MEE increases and thickens, the tympanogram flattens and widens into a type B pattern. The majority of AOM cases spontaneously resolve within days to weeks. If they do not, they become chronic OME and are less likely to resolve if persisting longer than 2 to 3 months. As the MEE resolves, the flat tympanogram progresses back to negative pressure, finally returning to normal atmospheric pressure when ET function has returned to normal and once the fluid has dried or drained from the middle ear.

As discussed earlier, TPP measurement is imprecisely relative to actual middle-ear pressure, thus negative TPP does not provide reliable diagnostic specificity or sensitivity to otitis media in children (Nozza et al., 1994; Paradise et al., 1976), and thus is not currently recommended as a reason to refer children for treatment. In the absence of significant hearing loss, symptoms, or otoscopic abnormality, negative TPP probably does not indicate a significant middle-ear problem and by itself should not constitute a referral for medical assessment or intervention. Positive middle-ear pressure can occur in patients with AOM.

TPP is useful for equilibrating ear canal air pressure to improve acoustic reflex thresholds and otoacoustic emission (OAE) responses (Trine et al., 1993). This is because better sound transmission occurs at TPP. Prieve et al. (2008) measured TEOAE and noise levels in 18 children under two conditions: On a day when the tympanogram TPP was normal and on a day when the tympanogram TPP was negative. They reported that TEOAE level decreased by about 4 dB from 1,000 to 4,000 Hz when TPP was negative, although negative TPP affected the overall pass rate in only 5% to 6% of cases.

Developmental and Aging Effects

Development and aging each affect tympanometry measures and thus need to be considered in normative criteria. Static admittance increases, ear canal volume increases, and TW decreases from infancy up to age 6 years (Roush et al., 1995). These changes are because of the increase in ear canal and middle-ear space, which make the middle-ear system more compliant with increased age. These changes continue into adulthood, especially for ear canal volume. Young adults aged 20 to 30 years have larger ear canal volume and narrower TW relative to children (Roup et al., 1998). Older adults (48 to 90 years) have lower static admittance, higher ear canal volume, and lower TW than younger adults (Wiley et al., 1996). Gender also affects immittance audiometry results. Several tympanometry studies have demonstrated a gender effect, with males having a higher static admittance and ear canal volume and narrower TW than females (Roup et al., 1998; Shahnaz and Davies, 2006; Wiley et al., 1996). Normative studies for adults are shown in Table 9.2.

Eustachian Tube Function Tests

The ET serves two main functions in the middle ear: pressure equalization (PE) and mucus drainage. Normally, the

ET is closed to protect the middle ear, but it opens during actions such as chewing, swallowing, and yawning. When the ET opens via active muscular contraction, a small amount of air is allowed into the middle ear, which serves to equalize pressure between the middle ear and ambient air. Pressure differences cause temporary low-frequency conductive hearing loss (CHL) because of stiffening of the TM and ossicular chain. Upper respiratory infections or allergies can cause the ET to become inflamed and swollen, trapping bacteria and causing ear infections. In children, the ET is shorter and straighter, as well as having poorer muscular control, which to otitis media. If the ET is blocked, it is unable to open to equalize pressure and negative pressure can develop. During activities that cause extreme pressure changes, such as flying or diving, ET malfunction can result in barotrauma (injury because of barometric pressure alterations). Barotrauma can cause TM perforation, CHL, and, in rare cases, a fistula of the oval window.

Measurement of TPP is an indirect measure of ET function, since significant negative or positive TPP indicates that the ET is not functioning normally to equalize middle-ear pressure. ET function tests are designed to actively test the function of the ET. ET function tests can be performed whether the TM is intact or not and are variants of tympanometry combined with active maneuvers to open the ET. A tympanogram is recorded before and after the maneuver, and shifts in TPP are observed. In an intact TM, shifts in TPP indicate ET functioning. In a perforated TM, the manometer of the immittance system can be observed for middle-ear pressure changes. Three main tests of ET function were described by Bluestone (1975) as discussed below.

Valsalva Test

The Valsalva test (Bluestone, 1975) introduces positive pressure into the middle ear via the ET using the classic Valsalva maneuver. A pretest tympanogram is recorded, the patient is instructed to perform the Valsalva maneuver by holding the nose and gently blowing air into the posterior nasopharynx. Then, a posttest tympanogram is recorded. Tubal opening is indicated by a positive shift in TPP.

Toynbee Test

The Toynbee test (Bluestone, 1975) uses the classic Toynbee maneuver and is considered more reliable than the Valsalva test. The patient is instructed to hold the nose and swallow, which introduces negative pressure into the middle ear.

Inflation–Deflation Procedure

The inflation–deflation test (Bluestone, 1975) uses high positive pressure (inflation) or negative pressure (deflation) introduced into the ear canal using the tympanometer (±400 daPa) while the patient is asked to swallow several times (Bluestone, 1975). Pre- and posttest tympanograms are recorded. Tubal opening is indicated by a shift in the pressure peak in the opposite direction of applied pressure.

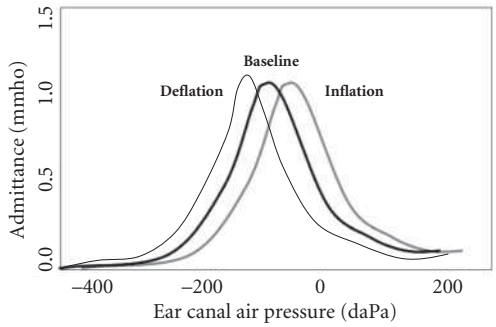


FIGURE 9.7 Baseline and posttest Eustachian tube function tympanograms. A positive shift is expected in the Valsalva and inflation tests. The Toynbee and deflation tests normally show a shift in the negative direction.

ET tests are simple to perform, are fast, and have face validity for the patient complaining of problems suggestive of ET dysfunction. Although easy to perform and seemingly useful, tests of ET function have become less popular over time because of a lack of evidence that they are predictive of pathologic problems. If TPP is normal and there are no clinical symptoms or signs of middle-ear problems, then there is no reason to suspect ET dysfunction and further ET function tests are not indicated. Based on complaints, history, or presence of negative pressure, ET dysfunction may be suspected and further tests may be useful. If ET tests are successful at demonstrating tubal opening, as shown in Figure 9.7, one can conclude that ET can open with active pressure, but do not tell us how the ET will function during various activities or conditions. If ET function tests do not demonstrate opening, this is a risk factor for recurrent otitis media and barotrauma under conditions such as flying and diving.

Patulous Eustachian Tube and Assessment

A patulous ET is abnormally open at rest, which can cause discomfort because of autophony (hearing one's own voice and breathing) that coincides with breathing. When patients present with complaints about discomfort because of hearing their own voice, breathing, or chewing, a patulous ET should be suspected. Patulous ET can be evaluated using immittance tests performed during breathing tasks. McGrath and Michaelides (2011) compared results of endoscopy and physician examination for 25 patients (8 to 82 years) referred for suspected patulous ET to 10 control patients. Admittance fluctuations greater than 0.07 mL during breathing tasks were found in 75% of ears with patulous ET whereas none of the control ears exhibited fluctuations during breathing. Thus, this study demonstrated a clear difference in ears with and without patulous ET on immittance testing.

Sensitivity and Specificity of Tympanometry

In order to truly understand the clinical usefulness of any diagnostic test, studies are needed of the test's performance in various populations at high and low risk for the disease of interest. Depending on the protocol, tympanometry has variable sensitivity to OME in children. It is important to note that the below studies examining test performance of pneumatic otoscopy have used experienced otoscopists who have received specific training and have been validated or compared to other expert otoscopists. Most OM is diagnosed by primary care physicians. In many cases, clinicians who use otoscopy to diagnose OME do not use pneumatic otoscopy and have not been validated against experienced otoscopists. For otoscopy to reach high levels of sensitivity and specificity, pneumatic otoscopy must be performed after ear canal cleaning by a highly experienced clinician. Because of these practical considerations, tympanometry is very useful as an alternative, especially when pneumatic otoscopy is not done by an experienced clinician. Performance of two tympanometry screening measures was assessed by Roush et al. (1992) in 374 ears of 3- to 4-year-old children in a preschool program against the gold standard of pneumatic otoscopy by an experienced, validated otoscopist. A procedure based on TPP less than −200 daPa or absent acoustic reflexes was compared with a proposed protocol, later published (ASHA, 1997). The procedure using TPP showed high sensitivity (95%), but low specificity (65%). The ASHA procedure had high sensitivity (84%) and specificity (95%), with a positive predictive value of 69% and a negative predictive value of 98%.

Nozza et al. (1992, 1994) studied six different combinations of static admittance, gradient, and acoustic reflexes in two related studies. In the first study (1992), two groups of children were evaluated. One group (n = 61, aged 1 to 8 years) received tympanostomy tubes and thus was at high risk for OME. The second group of children (n = 77, aged 3 to 16 years) attended an allergy clinic and was unselected with regard to otitis media history, thus was low risk. For the high-risk group, sensitivity (90%) and specificity (86%) were highest for gradient combined with acoustic reflexes. Gradient combined with static admittance also produced relatively high sensitivity (83%) and specificity (87%). In the low-risk group, sensitivity was 78% for all protocols except ipsilateral acoustic reflex alone (sensitivity = 88%) and gradient or static admittance <0.1 mmho (sensitivity = 67%). Gradient + ipsilateral reflex and gradient + static admittance performed equally well for specificity (99%). Positive predictive value was higher for gradient + static admittance (88%) than it was for gradient + ipsilateral reflex (78%). In a second study (Nozza et al., 1994), a group of children with recurrent or chronic OME (n = 171, aged 1 to 12 years), who were scheduled for myringotomy and tubes, received otoscopy by a validated otoscopist and tympanometry by a certified audiologist. The prevalence of OME in this group was 55%. Eleven criteria were evaluated for sensitivity and specificity. Best overall performance was found for TW or Y_{tm} combined with pneumatic otoscopy (sensitivity and specificity = 80%) or for TW alone greater than 275 daPa (sensitivity = 78%, specificity = 82%). The ASHA (1997) protocol showed high sensitivity (95%), but poor specificity (24%). These various studies demonstrate that the population and choice of cutoff criteria affect test performance greatly. Combinations of criteria, such as static admittance and TW or gradient and static admittance, perform better than single criterion. Static admittance (Y) alone has poor sensitivity but good specificity, depending on the cutoff criteria selected. Ipsilateral reflex or TW combined with Y provides good overall test performance, as does otoscopy combined with TW. Static admittance needs to be combined with pneumatic otoscopy, width, gradient, or ipsilateral acoustic reflexes to improve sensitivity.

Shahnaz and Polka (1997) compared standard and multifrequency tympanometry to diagnose otosclerosis. They provided guidelines and normative data for interpreting tympanometric data obtained using the Virtual 310 multifrequency tympanometry system. Tympanometric measures were compared in 68 normal ears and 14 subjects with surgically confirmed otosclerosis. Two traditional measures, Y and TW, were derived from a standard single-component 226-Hz tympanogram. Seven additional measures were obtained from multifrequency tympanometry, including resonant frequency (RF) and frequency corresponding to an admittance phase angle of 45° (F45°). Although Y tended to be lower and TW tended to be narrower in otosclerotic ears, there were no statistically significant differences between normal and otosclerotic ears. Test performance analysis showed that Y and TW were performing at chance levels and did not distinguish normal from otosclerotic ears. Shahnaz et al. (2009) also tested 62 normal-hearing adults and 28 patients diagnosed with otosclerosis. They reported that no measure obtained using standard low-frequency tympanometry was able to distinguish otosclerotic ears from normal ears, consistent with previous studies.

Calibration of Acoustic Immittance Systems

ANSI/ASA standard S3.39 (1987, R2012) specifies standards for acoustic immittance instruments. Calibration cavities should be provided with the immittance system with volumes of 0.5, 2.0, and 5.0 cm^3. The calibration cavities should be hard walled, cylindrical, and acoustically nonporous and provide a hermetic seal with supplied probe tips. For ipsilateral acoustic activators, the acoustic output should be measured in a standard HA-1 (2-cm^2) coupler connected to a sound level meter, frequency counter, and harmonic distortion analyzer.

Tympanogram Standard Format

The ANSI standard specifies many variables that are important for consistent tympanometry results. Various measurement units (mmho, kohm, mm mercury) have been used, so the ANSI S3.39 standard specifies uniform methods to label and scale the *x* and *y* axis of the tympanogram to allow for uniformity and easier correspondence between different instruments and clinics. The standard 226-Hz probe tone is referenced to, an acoustic admittance value of 10^{-8} m^3/Pa s (1 acoustic mmho), which corresponds to an acoustic admittance value of 10^8 Pa s/m^3 (1 acoustic kohm). An acoustic kohm equals the acoustic immittance of a 1-cm^3 volume of air at sea level, when barometric pressure equals 101 Pa (760 mm Hg) and temperature equals 20°C. These various units (mmho, kohm, mm Hg) have been used, so the ANSI S3.39 standard specifies uniform methods to label and scale the *x*- and *y*-axes of the tympanogram to allow for uniformity and easier correspondence between different instruments and clinics.

The tympanogram should be plotted with the probe ear indicated. The tympanogram may display compensated or baseline middle ear admittance relative to specified pressure (compensated or baseline) or it may display total peak admittance, including the ear canal admittance (compensation or baseline off). In either case, the compensation method and pressure used for baseline (e.g., +200, −400 daPa, or ambient) should be noted. The vertical or *y*-axis label should read: Acoustic admittance 10^{-8} m^3/Pa s (1 acoustic mmho) or acoustic admittance of an equivalent volume of air (cm^3) or both. The label on the horizontal or *x*-axis should read: Air pressure (daPa) [1 daPa − 1.02 mm H_2O]. Both the *x*- and *y*-axes should be linear. Negative air pressure values (below ambient) are scaled to the left of 0 daPa and positive air pressure values (above ambient) are plotted to the right.

For a probe frequency of 226 Hz, the scale aspect ratio should be 300 daPa equal to 1 acoustic mmho (1 cm^3). The scale aspect should remain the same if the total quantity in acoustic admittance is increased or decreased relative to the height of the measured tympanogram or if the recorded pressure range is changed.

SCREENING TYMPANOMETRY

Screening is designed to identify individuals who are at high risk for a given disorder or condition from among the general population with no apparent symptoms. The cost versus benefit of any screening program must be justified, ensuring that benefits outweigh cost. The ASHA (1997) Guidelines for Audiologic Screening endorses the identification of school children at risk for hearing impairment that may adversely affect education, health, development, or communication as an expected outcome for hearing-screening programs. Coordinated hearing and middle-ear screening managed by school systems can play an important role in the identification of children with middle-ear malfunction that provides the medical community with important information on the presence of chronic OME so that appropriate management options can be determined. Because the largest number of children will be identified through mass screening in preschool and elementary school hearing-screening programs, it is likely that the initial suspicion of chronic OME that significantly affects hearing will come from the school hearing screening rather than from the medical home. For this reason a coordinated effort among school screening programs and the medical community will result in the optimum management for students with OME (American Academy of Audiology [AAA], 2011; ASHA, 2004).

Most programs combine audiometric screening with tympanometric screening to detect both hearing loss and middle-ear disorders (AAA, 2011; ASHA, 2004). High-risk populations should be screened because of the higher risk of OME and hearing loss and the greater potential for negative impact on development and medical status. High-risk groups include children in Head Start preschools and Early Childhood Special Education (such programs are common in the United States), children with autism, developmental delay, speech–language delay, craniofacial anomalies, or syndromes associated with CHL, Native American or Canadian ethnicity, and children in day care settings. Children with family histories of chronic OME are also at high risk.

Tympanometry screening can be done by healthcare providers such as nurses, physicians, physician assistants, and speech–language pathologists. Support personnel trained and supervised by an audiologist can be used to carry out large-scale screening programs. Applicable state licensure laws and institutional policy should be consulted to determine credentialing requirements for support personnel. Tympanometry screening can be completed in any clean, well-lit space of sufficient size for a chair, the tympanometer, child and examiner, and parent, if applicable. Access to a sink or other hand hygiene and power supply is needed. Handheld screening instruments can run on rechargeable batteries, so testing at bedside in hospitals and clinics is also possible. A sound-treated booth is not necessary because the probe stimuli are presented at levels above ambient noise with insert or supra-aural earphones.

The AAA Childhood Hearing Screening Guidelines (2011) recommend the following procedures for tympanometry screening:

1. Calibrate tympanometry equipment daily.
2. Tympanometry should be used as a second-stage screening method following failure of puretone or OAE screening.
3. Use defined tympanometry screening and referral criteria: A ≥250-daPa TW is the recommended criterion. If it is not possible to use TW, then <0.2-mmho static admittance can be used as the criterion. A final choice for failure criterion is a negative pressure of >200 to 400 daPa; however, it is not appropriate to use this criterion alone to elicit a referral.

4. Young child populations should be targeted for tympanometry screening.
5. Use results of puretone or OAE and tympanometry rescreening to inform next steps.
6. Rescreen with tympanometry after a defined period, after failing the immediate puretone rescreening, and in 8 to 10 weeks for children failing puretone or OAE screening and tympanometry.

Because of the transient nature of otitis media and the need to minimize overreferrals, screening protocols for middle-ear disorders have recommended to rescreen for abnormal tympanometry results before a medical referral (AAA, 2011). The rationale for the length of the period between initial mass hearing screening and rescreening is based on information known about spontaneous resolution of transient MEE. Although MEE in children is prevalent, especially for the preschool population, it often resolves spontaneously without treatment. Based on an evidence-based review, AAA (2011) guidelines recommend that if both puretone and tympanometry screening is failed on the day of screening, children should be rescreened. The rescreening period should occur at a minimum of 8 weeks after the initial screening date and no later than 10 weeks after failing hearing screening to allow temporary middle-ear conditions to resolve.

Tympanometry in Newborns and Infants

Tympanograms recorded from newborn infants are often very different from those obtained from older infants, children, and adults mainly because of ear canal flaccidity in newborns. In neonate ears with confirmed middle-ear disease, 226-Hz tympanograms may not provide accurate diagnostic information. In addition, the variability of 226-Hz tympanometry in young infants because of the presence of M-shaped or notched patterns casts doubt on the clinical utility of these measures for newborns (Hunter and Margolis, 1992; Paradise et al., 1976; Sprague et al., 1985). For these reasons, 226-Hz tympanometry is not an effective test for middle-ear measurement in newborns.

The earliest tympanometric recordings from neonate ears were made with single-component instruments that used a 220-Hz probe tone and "arbitrary compliance units" (Bennett, 1975; Keith, 1973). These studies reported a frequent occurrence of double-peaked tympanograms. Later studies recorded resistance and reactance tympanograms at two probe frequencies, 220 and 660 Hz (Himelfarb et al., 1979; Sprague et al., 1985). Overall, these studies have shown that the newborn ear is highly resistive and has low negative reactance, suggesting a significant mass effect that offsets the stiffness of the middle-ear system. These effects are probably related to developmental differences between infant ear canals and middle ears relative to those of older children and adults. Anatomical and physical differences in the infant ear, ear canal wall flaccidity (Holte et al., 1991), smaller ear canal and middle-ear space, TM thickening, presence of middle-ear fluid and mesenchyme in some ears, and a more horizontal orientation of the TM with respect to the axis of the ear canal, are the most likely contributors (Eavey, 1993; Ruah et al., 1991).

Evidence has accumulated that tympanometry using a higher probe tone frequency (e.g., 1,000 Hz) is more sensitive to middle-ear status, compared with 226-Hz tympanometry, in infants less than 4 to 6 months old. Some studies have reported normative data for a variety of young ages, and some have investigated test performance of specific 1,000-Hz admittance criteria in predicting OAE screening results. Table 9.3 provides tympanometry normative data for infants and toddlers for 226 and 1,000-Hz probe tones.

Using a shape classification technique, Baldwin (2006) compared admittance tympanometry results at 226, 678, and 1,000 Hz between young infants (mean = 10 weeks) classified

TABLE 9.3

Normative Data for Tympanometry (226- and 1,000-Hz Probe Tones) in Infants and Children

Study	Age	Probe Frequency (Hz)	Y_{tm} 5–95 Percentiles (mmho)	Tympanic Width (daPa)
Margolis et al. (2003)	Birth to 4 wks	1,000	0.60–4.3 (–400 tail)	NA
Shahnaz et al. (2008)	32 wks gestational age	1,000	0.10–1.50 (+250 tail) 0.53–2.31 (–400 tail)	NA
Kei et al. (2003)	1–6 days	1,000	Right ears 0.39–2.28 (+200 tail)	Right ears 56.6–154
	1–6 days	1,000	Left ears 0.39–1.95 (+200 tail)	Left ears 46.1–144.2
Roush et al. (1995)	6–12 mos 12–18 mos 18–24 mos	226 226 226	0.20–0.50 (+200 tail) 0.20–0.60 (+200 tail) 0.30–0.70 (+200 tail)	102–234 102–204 102–204

Reprinted with permission from Hunter and Shahnaz. (2014) Table 8-1, pg. 117.

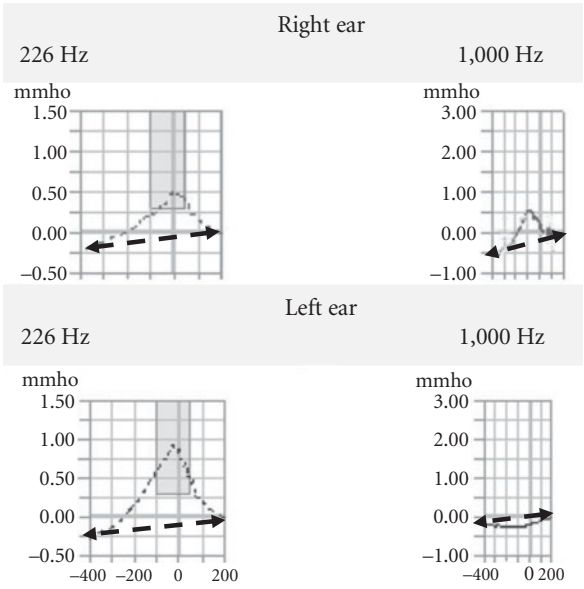


FIGURE 9.8 Tympanograms measured at 226 and 1,000 Hz in a newborn infant with a normal and disordered middle ear for the right and left ears, respectively. [Modified from Zhiqi L, Ku Y, Zhiwu H. (2010). Tympanometry in infants with middle ear effusion having been identified using spiral computerized tomography. *Am J Otolaryngol.* 31, 96–103.]

as having either normal or disordered middle-ear function, based on a combination of air- and bone-conduction auditory brainstem response (ABR) results and behavioral assessments. The tympanograms were organized using the traditional visual classification scheme discussed earlier (Jerger, 1970; Lidén, 1969) and an alternative method proposed by Marchant et al. (1986). The alternative method for classifying 1,000-Hz tympanograms provided the best results, with sensitivity of 0.99 and specificity of 0.89. The alternative method simply requires identification of the positive and negative *Y* tympanogram "tails" and drawing (visually or in reality) a line between the two points. Tympanograms measured at 226 and 1,000 Hz in a newborn infant with a normal middle ear as determined by CT scan in the right ear are shown in the left and right panels of Figure 9.8, respectively (Zhiqi et al., 2010). A tympanogram tracing falling above the dashed line connecting the tympanogram tails is suggestive of a normal middle ear, whereas a tracing falling below the line would be suggestive of middle-ear dysfunction. These plots show that 1,000 Hz is diagnostic in the left ear, whereas 226 Hz is not.

MULTIFREQUENCY, MULTICOMPONENT TYMPANOMETRY

Multifrequency, multicomponent tympanometry (MFT) expands on conventional 226-Hz tympanometry through the use of more than one probe tone frequency, typically ranging from 226 to 2,000 Hz, and measurement of more than one acoustic immittance component (e.g., admittance (Y_a), conductance (G_a), susceptance (B_a), and phase angle ϕ_a). A strength of MFT is the ability to identify both quantitative and qualitative changes in immittance components across frequency to obtain a more detailed view of the admittance characteristics of the middle ear than is possible with only a single-frequency or single-immittance component.

The qualitative hallmarks of MF tympanograms for adults with normal middle-ear function are the systematic and predictable tympanometric patterns that result with changes in frequency. Early work by Colletti (1975) demonstrated a systematic change in tympanometric shape from the well-known single-peaked tympanogram, to a slight notched peak shape, followed by a tympanogram with a deeper notched peak, eventually progressing to a "M"-shaped tympanogram. Examples of systematic changes in tympanogram morphology are shown in Figure 9.9. Each panel in Figure 9.9 represents uncompensated tympanograms for different immittance components and demonstrates the relationships between changes in frequency and tympanogram morphology; tympanogram shape becomes more varied and complex as frequency increases. Note, that whereas the tympanograms for each immittance component follow a similar sequence of morphologic change, the frequencies

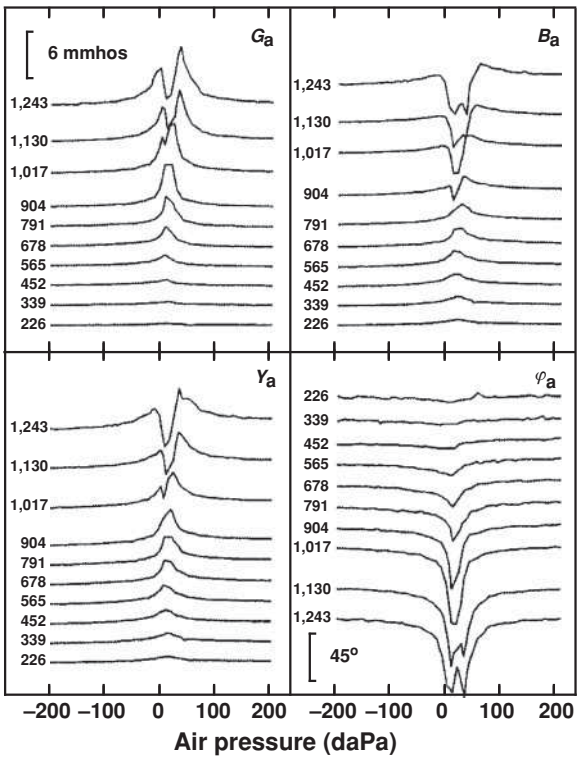


FIGURE 9.9 Uncompensated multiple frequency conductance [G_a], susceptance [B_a], admittance [Y_a], and phase angle [ϕ_a] tympanograms recorded from a 40-year-old man with normal middle ear function.

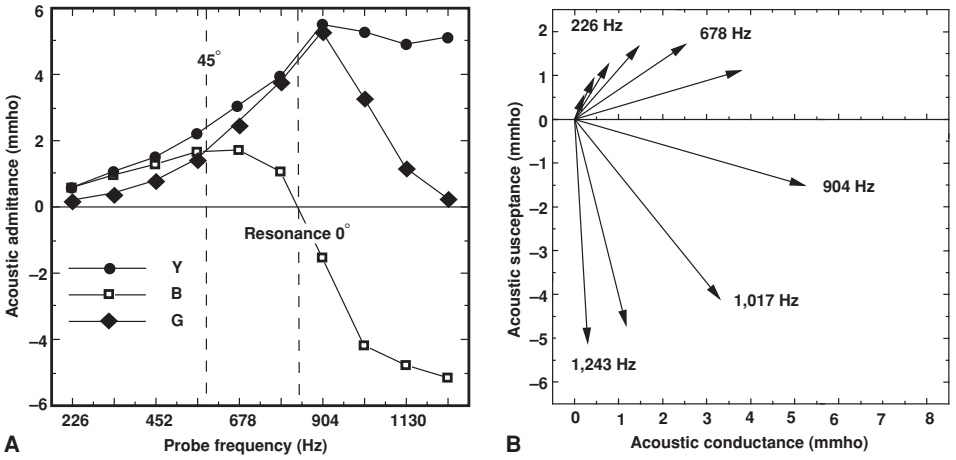


FIGURE 9.10 Peak-compensated static acoustic admittance calculated from the normal multiple frequency tympanograms in Figure 9.9. Peak $200B_{tm}$ (*open squares*), $200G_{tm}$ (*filled diamonds*), and $200Y_{tm}$ (*closed circles*) are plotted in rectangular format as a function of probe frequency in (**A**); the rectangular admittance values corresponding to phase angles of 45° and 0° are indicated by *dashed lines.* Rotation of the admittance vector as a function of probe frequency is plotted in polar format in (**B**).

at which notching begins and the extent to which changes in tympanogram morphology occur vary by immittance component. Identification of frequencies at which some of the characteristic changes in tympanogram morphology occur can provide useful diagnostic information.

The tympanograms shown in Figure 9.9 present a much more complicated picture than is portrayed by single-frequency, single-component tympanograms. Fortunately, there are several ways to analyze and interpret the information presented in Figure 9.9. Recall the discussion earlier in the chapter regarding the calculation of peak-compensated static acoustic admittance (Y_{tm}) with 226-Hz tympanometry; Y_{tm} is derived by simply finding the peak to tail (+200 daPa) amplitude of the admittance tympanogram. This same procedure can be applied to the group of tympanograms in Figure 9.9, with the note that when a notch or trough is present, the trough to tail amplitude is measured. The results are shown in Figure 9.10A where Y_{tm} for three tympanometric components (Y_a, B_a, and G_a) is plotted as a function of frequency (Hz). Plots for each component present with a similar monotonically rising magnitude (mmho) from 226 through approximately 550 Hz. The dashed line at approximately 550 Hz represents the frequency at which the amplitudes of B_a and G_a are equal or when the admittance phase angle (ϕ_a) is at 45°. As frequency increases, the magnitudes of Y_a and G_a continue to increase until both begin to decrease at approximately 900 Hz, with G_a decreasing more rapidly, but neither falling below 0 mmho. Alternatively, B_a follows a significantly different path above 550 Hz and crosses 0 mmho at approximately 850 Hz, then continues to −5 mmho at 1,243 Hz. The point at which B_a crosses 0 mmho is denoted by a dashed line at approximately 850 Hz and represents middle-ear resonance, or the point at which mass and stiffness susceptance are equal. Identifying the extent to which mass and stiffness influence the acoustic response properties of the middle ear can provide important clues regarding the state of the middle ear. For example, positive B_a would indicate that an ear is in a stiffness-dominated state, whereas negative B_a would indicate that a middle ear is in a mass-dominated state.

Figure 9.10B shows an alternative plotting scheme for data from Figure 9.10A. In this case, the vectors (arrowed lines of varying length) represent the admittance magnitude (algebraic sum of B_a and G_a) by frequency, with an orientation or angle determined by ϕ_a. Similar to the plot in Figure 9.10A, estimation of RF and contributions of mass and stiffness can be determined from Figure 9.10B. For vectors above zero, phase angle is positive and would therefore be considered stiffness controlled; below zero, phase angle is negative and would therefore be considered mass controlled. The plot in Figure 9.10B highlights the importance of knowing the ϕ_a; without phase angle, it is not possible to determine the "direction" of the vector, or therefore, the frequency at which resonance occurs or if a middle ear is mass or stiffness dominated. The information presented in Figure 9.10B suggests that middle-ear resonance occurs at a frequency between 791 and 904 Hz. An alternative to calculating and/or plotting data obtained from MFT is to rely on the information that is obtained through the morphologic changes in the tympanograms themselves.

The Vanhuyse Model

During the same period of time when Colletti's (1975) early work was published, a group of nuclear physicists, at an otolaryngologist's request, were trying to explain the underlying cause of a series of complex tympanometric shapes obtained from a patient. A physicist in the group suspected

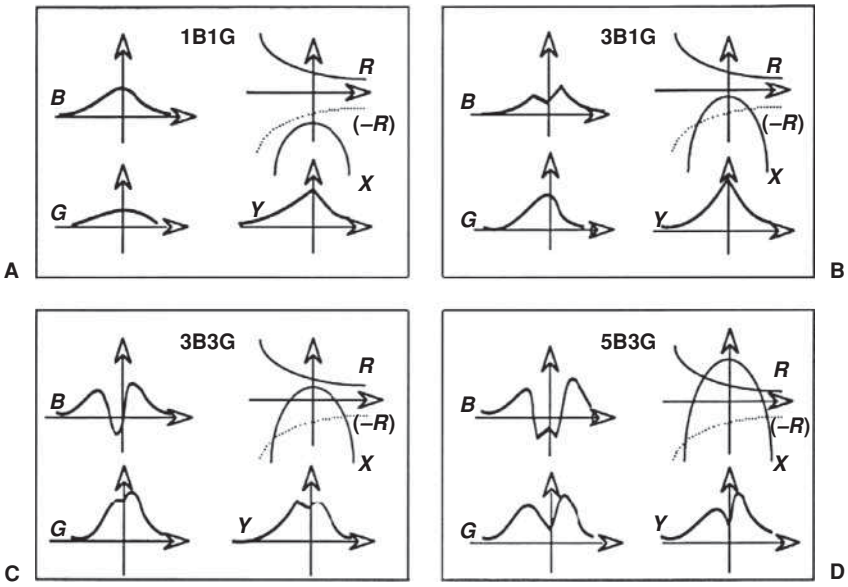


FIGURE 9.11 A model proposed by Vanhuyse et al. (1975) shows patterns of susceptance (*B*), conductance (*G*), resistance (*R*), reactance (*X*), and admittance (*Y*) tympanograms. (**A–D**) depict the interaction of resistance and reactance and the effects on *B, G,* and *Y* tympanograms in a healthy adult ear, beginning with what would be expected for low-frequency probe tone in (**A**) to progressively higher frequencies in (**B–D**). (Adapted from Hunter LL, Shahnaz N. (2014) *Acoustic Immittance Measures: Basic and Advanced Practice.* San Diego, CA: Plural Publishing, reprinted with permission.)

a simple explanation based on the interaction of the impedance components of resistance and reactance and quickly presented a model that accounted for the different tympanometric patterns. This model, first presented in publication by Vanhuyse et al. (1975), has become one of the most important contributions to the understanding of multifrequency tympanometry. The Vanhuyse model, as it has come to be known (Figure 9.11, Hunter and Shahnaz, 2014), is based on the assumption of the changes in the shapes of resistance and reactance as a function of ear canal pressure. The model in Figures 9.11A, 9.11B, 9.11C, and 9.11D depicts the interaction of resistance and reactance in a healthy adult ear, beginning with what would be expected for low-frequency probe tone in Figure 9.11A to progressively higher frequencies in Figures 9.11B, 9.11C, and 9.11D. The influence of the interaction of resistance and reactance on susceptance (B_a), conductance (G_a), and admittance (Y_a) is also shown in each subfigure. The model is divided into categories based on the number of positive and negative peaks, or extrema, in the susceptance and conductance tympanograms. For example, in Figure 9.11A, the "BG" tympanograms, as they have come to be known, each have one peak or extrema and would be classified as 1B1G tympanograms. The complexity of the BG tympanogram morphology increases as resistance (R) and reactance (X) interact, changing from a 1B1G pattern, to a 3B1G pattern in Figure 9.11B, and so forth. Resistance (R) is assumed to decrease monotonically as a function of negative to positive change in air pressure, as shown in the upper right hand corner of Figures 9.11A to 9.11D. On the other hand, reactance (X) is a single-peaked function, symmetric around ambient ear canal pressure, also shown in the upper right hand corner of panels A through D. Although single-frequency, single-component tympanograms can be interpreted using the Vanhuyse model, employing this model for interpreting tympanograms at multiple frequencies and with multiple components is more useful since more complexity in tympanometric shape is typically seen at higher probe frequencies and with individual admittance components. For example, note that the admittance tympanograms (Y_a), located in the bottom right hand corner of panels A to D, present with essentially only two patterns (a 3Y pattern) as resistance and reactance interact. The BG tympanograms on the other hand are more sensitive to the interactions of resistance and reactance, which is reflected in the more complex tympanometric patterns as frequency increases (e.g., a 5B3G pattern in Panel D). The Vanhuyse model's depiction of the effects of the interaction of resistance and reactance on susceptance (B_a), conductance (G_a), and admittance (Y_a) led to the use of this model as the dominant strategy for interpreting MFT data.

Resonant Frequency

The primary diagnostic utility of MFT is found in the ability to determine whether the middle ear is characterized by an RF that is typical or higher than normal, such as with otosclerosis, or lower than normal, such as with ossicular discontinuity. Examination of both quantitative and qualitative changes in tympanogram morphology can provide clues regarding the RF of the middle ear. Although different qualitative methods of estimating RF have been suggested, a simple and accurate method involves examining the point at which the notch in the susceptance tympanogram approximates the plane of the positive tail of the tympanogram (Hunter and Margolis, 1992). Figure 9.12A shows an example of the notch in a susceptance tympanogram equal to the magnitude of the plane of the positive tail (illustrated with the horizontal line); this pattern is indicative of middle-ear resonance. Figures 9.12B and 9.12C represent the admittance characteristics of the same

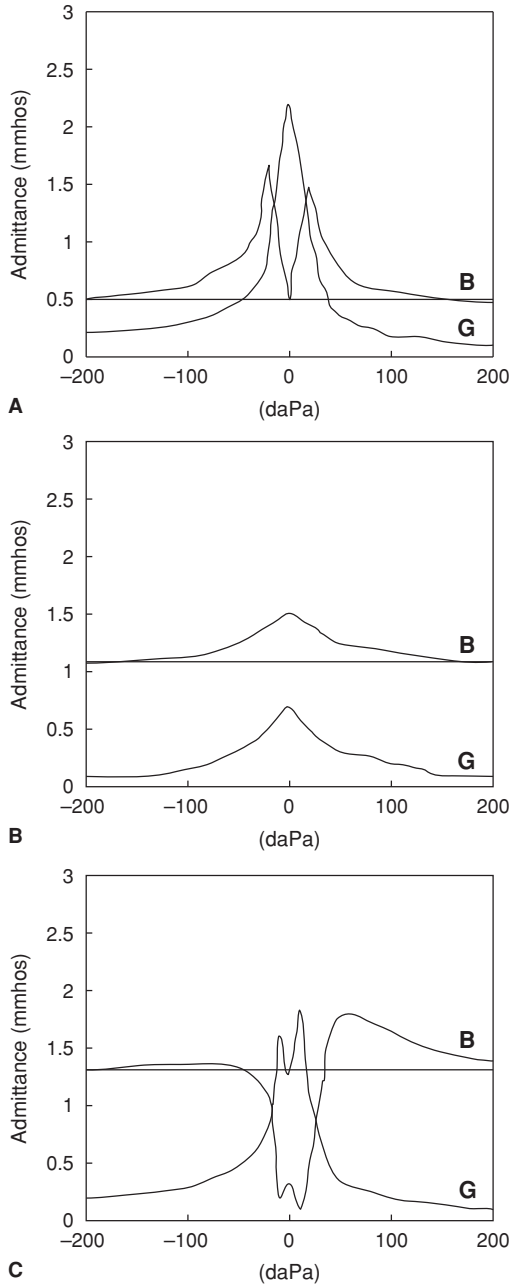


FIGURE 9.12 Susceptance (*B*) and conductance (*G*) tympanograms representative of a middle ear at resonance (**A**), a middle ear that is stiffness dominated (**B**), and a middle ear that is mass dominated (**C**). (Wiley TL, Fowler CG. (1997) *Acoustic Immittance Measures in Clinical Audiology. A Primer.* San Diego, CA: Singular Publishing.

middle ear measured at a frequency below RF and a frequency above RF, respectively. The tympanograms represented in Figure 9.12 were obtained in the same manner in which conventional single-frequency tympanograms are obtained by holding frequency constant and sweeping pressure in a positive to negative direction (e.g., a sweep-pressure procedure).

To truly make the information in Figure 9.12 useful it is necessary to know the frequency at which the tympanograms were obtained. The important clinical question would be: Is the frequency at which the susceptance notch crosses the plane of the positive tail above or below the range in which a normal RF is expected? Mean middle-ear RF for adult ears ranges from approximately 1,100 to as high as 1,800 Hz. If the BG tympanogram presented in Figure 9.12A was observed at a probe frequency within the normal range for middle-ear RF, this result would suggest normal middle-ear function. If middle-ear resonance was identified at a much lower frequency, outside the normal range such as 500 Hz, this would be consistent with a mass-dominated pathology; a resonance identified at a much higher frequency such as beyond 1,800 Hz may suggest the possibility of a stiffness-dominated pathology. A quantitative way of estimating RF includes a sweep-frequency approach where the difference between *B* at a tympanogram tail (either positive or negative) and *B* at peak pressure (e.g., ΔB) is plotted as a function of frequency; the frequency at which ΔB approaches 0 (0 mmhos) is considered the middle-ear RF (see Hunter and Shahnaz, 2014). It is important to note that the range of RF observed in ears with normal middle-ear function is quite large and data from some studies show a range that is skewed toward the upper frequencies; some studies report an upper limit of normal at or close to 2,000 Hz. The large range of normal for RF and the potential "ceiling" effect observed in some normative data lessen the clinical utility of MFT in detecting pathologies that produce stiffening effects (Shanks et al., 1993). Although the frequency resolution over which MFT data can be obtained varies by equipment manufacturer, it seems that increasingly options for MFT tests are limited to three frequencies (e.g., 226, 678, and 1,000 Hz).

Some equipment allows for a quantitative approach for estimating RF that does not require the clinician to make a judgment based on visual classification of tympanogram morphology. For example, some Grason Stadler systems (GSI-33 and Tympstar) with MFT capabilities can obtain a sweep-frequency MFT recording that estimates the B_a tympanogram peak (or notch) to tail difference (e.g., ΔB) across a range of frequencies; for sweep-frequency recordings, ear canal pressure is held constant and frequency is changed. The point at which a ΔB value of 0 is obtained (e.g., 0 mmhos, or the point where mass and stiffness are equal and phase angle is 0) is identified as middle-ear RF.

Low Resonant Frequency

Pathologies that typically result in abnormally low RF and abnormal tympanogram morphology include ossicular discontinuity and external otitis. For example, Figure 9.13 represents the effects of ossicular discontinuity on BG tympanograms. First, it is obvious that the tympanogram morphology is significantly different than what

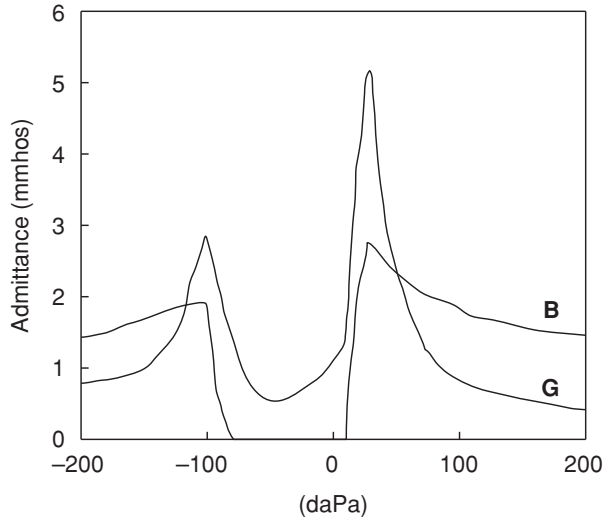


FIGURE 9.13 A susceptance (*B*) and conductance (*G*) tympanogram representative of a middle ear with ossicular discontinuity. (Wiley TL, Fowler CG. (1997) *Acoustic Immittance Measures in Clinical Audiology. A Primer.* San Diego, CA: Singular Publishing.

is expected from a normal middle ear (re: BG tympanograms in Figure 9.12). In addition, because the frequency at which these BG tympanograms were obtained (678 Hz) is below the normal limits for RF, and the B_a notch extends well below the plane of the positive tail, it can be concluded that this middle ear is mass dominated and is operating at a low RF; RF is potentially far below 678 Hz, since the notch extends significantly below the plane of the positive tail. Also, notice the wide notching for both B_a and G_a tympanograms (peak-to-peak difference on the *x*-axis of approximately 150 daPa). This wide-notch configuration is another tympanogram quality consistent with ossicular discontinuity. Figure 9.14 shows MFT for a 9-year-old boy with recurrent OME and a thickened, retracted right eardrum. The left ear had a normal appearance, with 226-Hz tympanograms normal in both ears (see inset). In this case, the BG tympanograms at 500 Hz shows notching in the right ear for *B* (susceptance) and appears normal for the left ear. Examination of the changes in BG tympanograms as frequency increases suggests that RF is about 500 Hz in the right and just above 710 Hz in the left. At 500 Hz in the right ear, a 3B3G pattern is apparent, showing mass domination. Thus, the multifrequency tympanograms clearly show abnormal patterns that are not found in the 226-Hz admittance tympanograms.

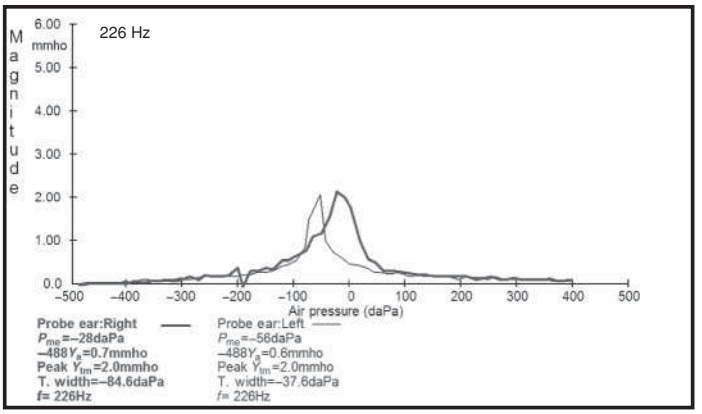
High Resonant Frequency

Pathologies that create a middle-ear system dominated by stiffness can result in abnormal tympanogram morphology and RF that exceeds the normative range. Because stiffening pathologies have the effect of reducing the mobility of the TM and low-frequency stimuli are less affected by increased stiffness, BG tympanograms from stiffness-dominated ears are generally more flat, with fewer peaks. For stiffness-dominated ears, as frequency increases, changes in tympanogram morphology may occur and may progress to a point indicative of resonance; however, RF may be beyond the upper limits of the immittance device, so RF may not be identified. Figure 9.15A illustrates the stiffening effects of partial OME on BG tympanograms. Note that although the tympanograms in Figure 9.15A exhibit peaks that are more widely spaced than normal, similar to the BG tympanograms in Figure 9.13, their amplitudes are greatly reduced. Figure 9.15B demonstrates a BG tympanogram pattern for an ear completely filled with effusion; in this case the middle ear is dominated by extreme stiffness and no tympanometric movement is detected.

Several studies have investigated the potential utility of MFT to detect otosclerosis. Ogut et al. (2008) obtained conventional and MFT data from 28 ears with surgically confirmed otosclerosis and 100 ears with normal middle-ear function. Using a static acoustic admittance criterion of ≤0.3 mmhos (for 226-Hz tympanometry) to classify an ear as disordered, sensitivity and specificity values of 40% and 98% were obtained. For RF with a criterion of ≥1,025 Hz, sensitivity and specificity values of 80% and 82%, respectively, were reported; a sweep-frequency method to identify ΔB was used to estimate middle-ear resonance. Other studies have reported improved test performance for detecting otosclerosis when using the frequency most closely approximating a 45° admittance phase angle (Shahnaz and Polka, 1997); phase angle of 45° is defined as the point at which the admittance contributions of B_a and G_a are equal (see Figure 9.10A).

Although some results from the studies mentioned previously suggest that MFT is more sensitive to otosclerosis than single-frequency tympanometry, a systematic review of the literature examining the diagnostic accuracy of MFT in identifying the presence or absence of otosclerosis suggested that criterion references of RF or phase angle of 45° (F45°) are not strong predictors of otosclerosis (Sanford et al., 2012). Specifically, Sanford et al. (2012) identified three studies that met strict inclusion criteria and had suitable data for the systematic review. Based on sensitivity and specificity data from each study, likelihood ratios (both positive and negative ratios) and 95% confidence intervals were computed. Whereas the high proportion of positive likelihood ratios in the 95% confidence interval range suggested that RF might be potentially useful in ruling out otosclerosis, the low proportion of negative likelihood ratios suggested that RF and a phase angle of 45° are weak predictors of otosclerosis.

The primary clinical assets of MFT are the ability to assess the relative contributions of mass and stiffness of the middle ear and to help identify middle-ear RF. However,



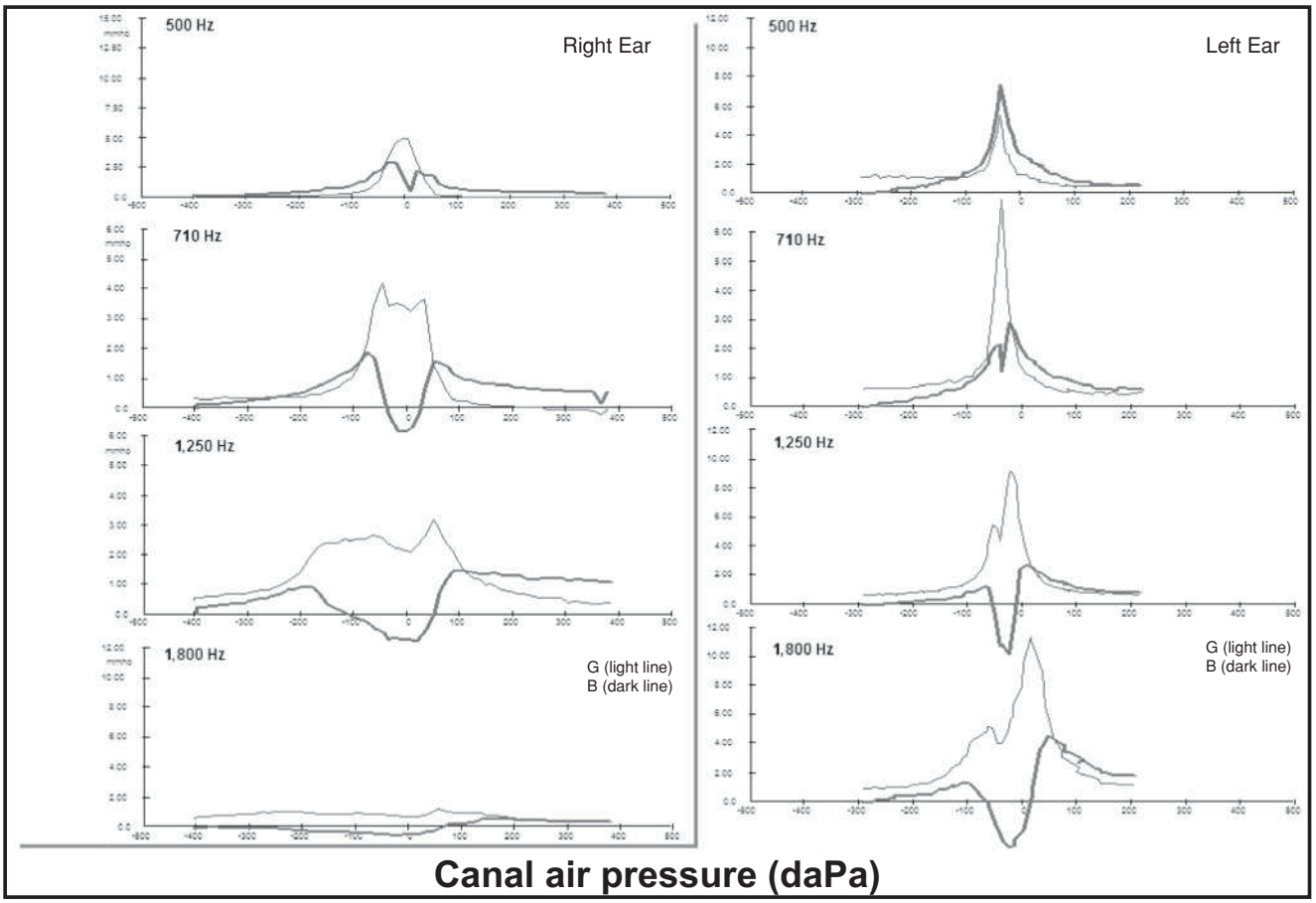


FIGURE 9.14 The 226-Hz admittance tympanograms and multifrequency tympanograms for a 9-year-old boy with recurrent otitis media with effusion and a thickened, retracted right eardrum.

one limitation associated with MFT tests is the restricted range of probe frequencies (<2,000 Hz), which could limit identification of RF in some cases (Shanks et al., 1993). In addition, the more complicated multifrequency tympanometric response patterns are often difficult for clinicians to interpret, and the normative range of RF is fairly wide (Margolis and Goycoolea, 1993). However, MFT test results, in conjunction with other audiometric test findings, can be used to provide more information regarding middle-ear function than would be available from a single audiometric test interpreted in isolation.

WIDEBAND ACOUSTIC IMMITTANCE

Imagine interpreting audiometric test findings from a patient using only the information from the audiogram at 250 Hz. With this limited amount of information it would

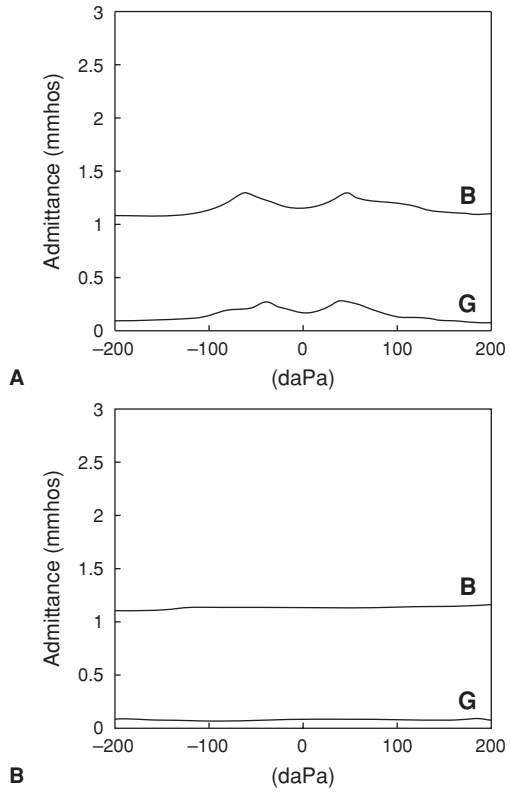


FIGURE 9.15 Susceptance (*B*) and conductance (*G*) tympanograms representative of a middle ear with partial otitis media with effusion (**A**) and an ear completely filled with effusion (**B**); in the latter case, no tympanometric movement is detected. (Wiley TL, Fowler CG. (1997) *Acoustic Immittance Measures in Clinical Audiology. A Primer.* San Diego, CA: Singular Publishing.

be difficult to comprehensively characterize the patient's auditory sensitivity. However, for decades, audiologists have relied on tests that use single or a limited range of frequencies to describe middle-ear function. Although conventional single- and multifrequency tympanometry have been and continue to be useful tests of middle-ear status in adults and children presenting with a variety of middle-ear disorders, a broadband measure of middle-ear function provides a more comprehensive view of the middle ear's acoustic response properties over a wide range of frequencies. Wideband acoustic immittance (WAI) tests provide this broadband view of middle-ear function and are showing promise as powerful tools for evaluating middle-ear status (Feeney and Keefe, 2012).

PRINCIPLES AND CALIBRATION OF WAI

WAI responses have several desirable qualities. Most importantly, WAI tests utilize stimuli (clicks or simultaneously presented puretones) with broad frequency spectra (usually 250 to 8,000 Hz) to assess middle-ear status, whereas for conventional tympanometry, generally only a single puretone is used. The capability to assess a broad frequency range allows for improved understanding of how the middle ear is functioning across the range of frequencies important for human hearing. Second, unlike admittance tympanometry, WAI measurement results are relatively independent of the location of measurement in the ear canal; this allows for a direct measure of middle-ear function without as much concern for ear canal effects.

WAI measurement theory takes advantage of the fact that when sound is presented to the external ear canal some of the sound is absorbed by the middle ear and transferred into the inner ear, whereas some of the sound is reflected back along the ear canal. The application of a rigorous calibration technique facilitates reliable measurement of the absorbed (or reflected) sound.

The most common calibration routine reported in the literature involves the calculation of the Thevenin source impedance and source pressure associated with the transducers within the WAI probe (Keefe et al., 1992; Liu et al., 2008; Voss and Allen, 1994). This process typically involves making acoustic pressure measurements within a set of at least two rigid cylindrical cavities that are similar in diameter to the ear; different sets of cavities, which approximate the average ear canal diameter for adults and infants, are used. Once the Thevenin parameters of the WAI probe assembly are known, the same probe and stimulus used in calibration can be applied to an unknown system (e.g., the human ear canal) and pressure measurements can be made. The pressure measurements made in the ear canal are then compared to the characteristic impedance of the ear canal using standard transformations to derive the pressure reflectance coefficient, *R,* which is then squared to derive the energy/power reflectance, $\Re$ (Keefe et al., 1992). Absorbance is calculated as $1 - \Re$ as a function of frequency and represents the proportion of sound power absorbed by the middle ear. Use of this calibration routine and data transformation overcomes the problems introduced with standing waves for frequencies above 2,000 Hz, as encountered with conventional impedance measurements (Stinson et al., 1982). Therefore, if negligible sound is absorbed by the ear canal, which is generally assumed in adult ears, WAI measured at the plane of the probe tip is essentially the same as if the measurement was taken next to the TM.

WIDEBAND ACOUSTIC IMMITTANCE MEASUREMENTS

The umbrella term, WAI, covers a variety of acoustic measurement types, such as energy or power reflectance, absorbance, conductance, and admittance. The variety of measurement types derived from WAI data provide alternative perspectives on how the transfer of acoustic information

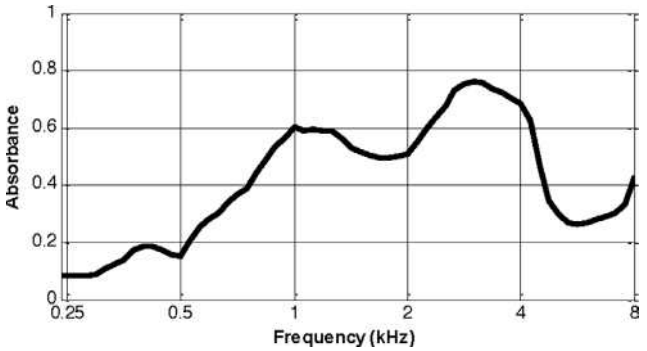


FIGURE 9.16 Wideband absorbance data, plotted as a function of frequency (kHz), from an adult with normal middle-ear function.

is handled by the middle ear. Whereas the term wideband reflectance has been used regularly to describe this emerging measurement tool, in this chapter, we will focus primarily on the quantity of absorbance and use the term WAI when referring, generally, to this family of WB ear canal–based measurements (Feeney et al., 2013). Whereas absorbance is simply 1 minus power reflectance, absorbance is better suited for some WAI analysis techniques (Liu et al., 2008) and the peak-shaped absorbance function bears resemblance to conventional tympanometry morphology. Currently, two audiologic device manufacturers provide commercially available systems capable of making WAI measurements (Mimosa Acoustics, Champaign, IL and Interacoustics, Assens, Denmark).

Unlike tympanometry measurements, WAI data can be obtained at ambient ear canal pressure. An ambient WAI test, which takes just 1 to 2 seconds to complete, provides a broad spectral view of middle-ear function. As evidenced in Figure 9.16, WB absorbance changes as a function of frequency and ranges from 1, meaning much of the acoustic power is absorbed, to 0, meaning little of the acoustic power is absorbed. Higher amounts of absorbance occur in the mid-frequency range (750 to 4,000 Hz) with relatively less absorbance in both the low and high frequencies. A growing body of work has described WAI for infants, children, and adults with normal middle-ear function (Hunter et al., 2013; Kei et al., 2013; Shahnaz et al., 2013) and with middle-ear disorders (Nakajima et al., 2013; Prieve et al., 2013).

Wideband Tympanometry

Just as the addition of ear canal pressure sweeps increased the usefulness of early work with admittance measurements in adults and children, it was hypothesized that WAI measurements would reveal more developmental effects and be more diagnostically useful if they were obtained in the presence of ear canal pressure changes (Keefe and Simmons, 2003; Margolis et al., 1999; Piskorski et al., 1999; Sanford et al., 2009). WAI data obtained in the presence of ear canal pressure sweeps (e.g., WB tympanometry) are presented in Figure 9.17; the WB tympanogram provides a multidimensional representation of middle-ear function with absorbance plotted as a joint function of frequency and pressure. Because the pressure sweep used in WB tympanometry is similar to that used in conventional tympanometry, it is possible to extract both WB and traditional single-frequency tympanometry measurements in a single measurement of approximately 7 seconds. Currently, the Titan system (Interacoustics, Assens, Denmark) is the only FDA-approved device capable of measuring both ambient and tympanometric WAI.

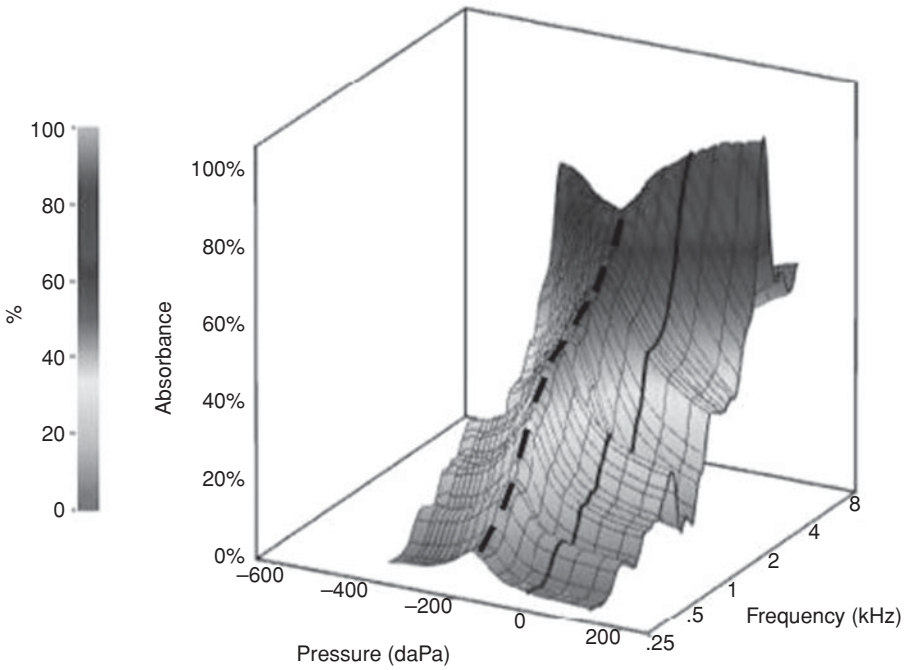


FIGURE 9.17 Wideband tympanogram data (absorbance), plotted as a function of frequency (kHz) and pressure (daPa), from an adult with negative tympanometric peak pressure, but otherwise normal middle-ear function. Tympanometric peak pressure is located at approximately –100 daPa (denoted with the *dashed line*); the *solid black line* denotes absorbance at 0 daPa.

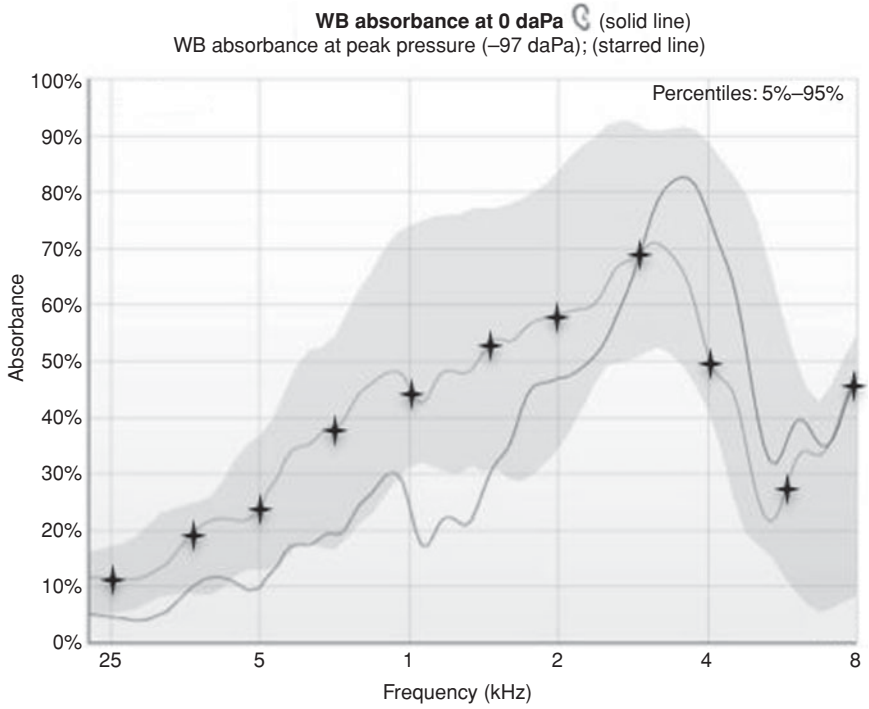


FIGURE 9.18 Wideband absorbance data, plotted as a function of frequency (kHz), from an adult with negative tympanometric peak pressure, but otherwise normal middle-ear function; these data were extracted from the wideband tympanogram presented in Figure 9.17. Absorbance at 0 daPa is denoted by the *solid line* and absorbance at tympanometric peak pressure is denoted by the *starred line.*

The WB tympanogram in Figure 9.17 is from an adult with mild, negative TPP, as evidenced by the peak of the response located at approximately −100 daPa (dashed line). The solid black line denotes absorbance at 0 daPa and represents how the middle ear is operating at atmospheric pressure. The ability to assess the middle ear at TPP, relative to ambient conditions, may provide useful diagnostic information, especially in situations where excess middle-ear pressure, often the more lateral pathology, may be obscuring the presence of another middle-ear disorder (Margolis et al., 1999). Figure 9.18 shows WB absorbance data (extracted from the WB tympanogram in Figure 9.17), plotted at ambient pressure (solid line) and at TPP (starred line). Note that absorbance at ambient pressure in the low frequencies is decreased (relative to absorbance at TPP) because of negative middle-ear pressure. Because absorbance at TPP represents an estimate at which the middle ear is most efficient at transferring acoustic power, evaluation of absorbance data at TPP may be of interest.

Effects of Ear Disorders on Wideband Acoustic Immittance

A number of studies have reported changes in WAI responses in the presence of middle-ear dysfunction including otitis media with fluid (Beers et al., 2010; Ellison et al., 2012; Feeney et al., 2003; Piskorski et al., 1999), otosclerosis (Nakajima et al., 2012; Shahnaz et al., 2009; Voss et al., 2012), excess middle-ear pressure (Beers et al., 2010), perforation of the TM (Feeney et al., 2003; Nakajima et al., 2012; Voss et al., 2012), and ossicular discontinuity (Feeney et al., 2003; Voss et al., 2012).

Excess middle-ear pressure results in increased stiffness of the TM and a systematic decrease in absorbance with increasing negative TPP across the majority of measured frequencies (Beers et al., 2010). Figure 9.19 shows the effects of increased middle-ear stiffness from fluid behind the TM on absorbance (data replotted from Feeney et al., 2003). Specifically, small increases in absorbance occur across low to mid-frequencies, with sharp peaks occurring between 4,000 and 6,000 Hz. A completely different pattern is observed for the ears with TM perforations (Figure 9.19, see right panel), with absorbance as high as 1 in the low frequencies and nonmonotonic patterns above 1,000 Hz. Voss et al. (2012) demonstrated the effect of TM perforation size on WAI for cadaveric ears, showing that smaller perforations resulted in the largest effects; Voss et al. (2012) suggested that resonance effects created by hole in the TM may be the dominant factor responsible for these effects.

Ellison et al. (2012) assessed the accuracy of WAI in predicting MEE in a group of 44 children (median age = 1.3 years) with surgically confirmed OME; an age-matched group of 44 children (median age = 1.2 years) with normal pneumatic otoscopic findings and no history of middle-ear surgery or ear disease was used as the control group. Ellison et al. (2012) found that absorbance was reduced in ears with MEE compared to ears from the control group. In addition, whereas WAI measurement types (absorbance and admittance magnitude) were the best univariate predictors of MEE, a predictor combining absorbance, admittance magnitude, and phase was the most accurate overall. Results from this study suggest that absorbance is sensitive to MEE,

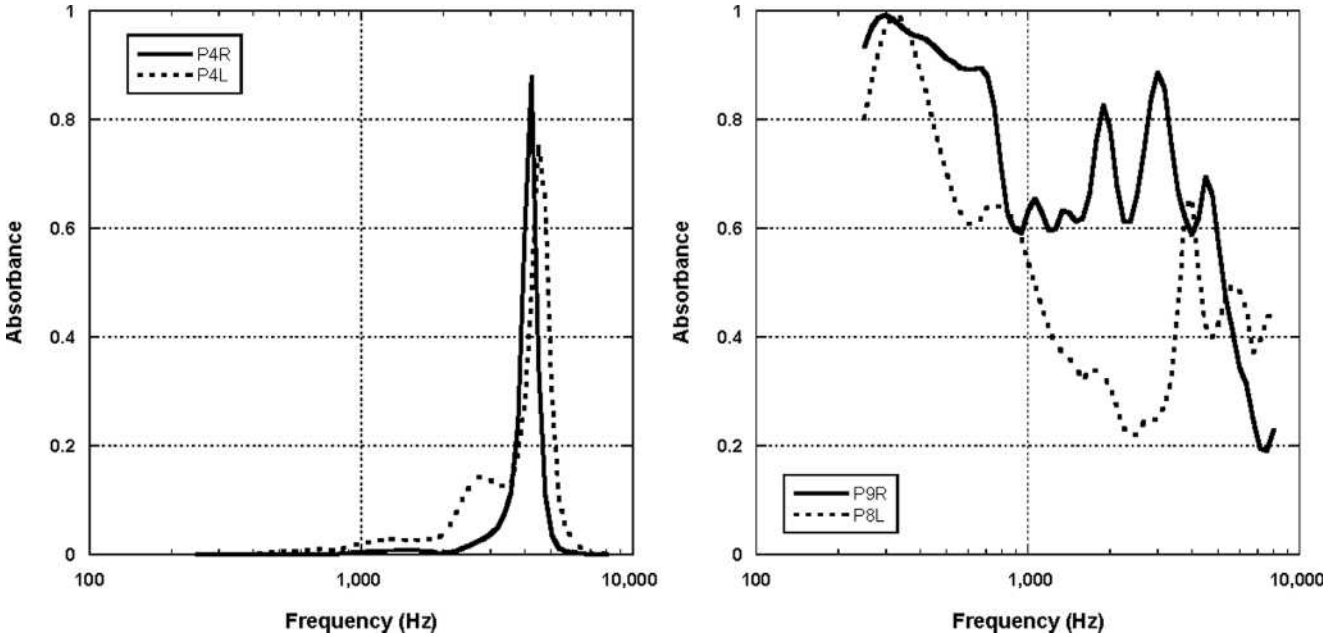


FIGURE 9.19 Wideband absorbance data, plotted as a function of frequency (Hz), from an adult with otitis media with effusion in both ears (**left panel**) and from two adults with tympanic membrane perforations (**right panel**). Replotted from Feeney MP, Grant IL, Marryott LP (2003).

and WAI measurements are accurate predictions of MEE in young children.

Sanford and Brockett (in press) obtained WAI data from 20 ears with suspected OME (sOME), 19 ears with PE tubes, and 15 ears with negative TPP (ranging from −115 to −275 daPa); mean WAI data are presented in Figure 9.20. Ears with sOME presented with reduced absorbance across a majority of frequencies (250 to 8,000 Hz) with more significant reduction in absorbance and narrowing of the absorbance peak from 1,000 to 5,000 Hz. The presence of

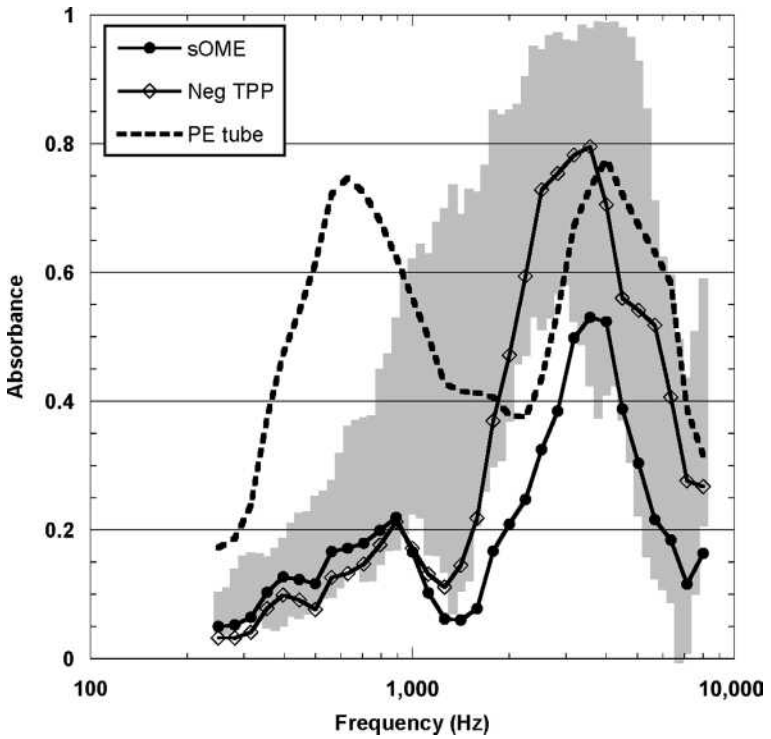


FIGURE 9.20 Wideband absorbance data, plotted as a function of frequency (Hz), from children with suspected otitis media with effusion (sOME), negative tympanometric peak pressure (TPP), and pressure equalization tubes (PE tube). The shaded region represents the 10th to 90th percentiles for absorbance for 0.5-7 year-old children with normal middle-ear function (N = 59 ears; mean age of 1.8 yrs., unpublished data from Boys Town National Research Hospital). Recreated figure using data from Sanford CA, Brockett JE. (in press) article is still In Press Characteristics of wideband acoustic immittance in ears with middle ear dysfunction. *J Am Acad Audiol.*

PE tubes creates an additional absorbance peak in the low frequencies, perhaps because of the resonance effect of the PE tube. Changes in WAI for ears with negative TPP are less significant than those with OME and PE tubes; for ears with negative TPP, the general trend is for decreased absorbance below 2,000 Hz, with a mixture of decreased and increased absorbance above 2,000 Hz.

Work by Shahnaz et al. (2009) demonstrated statistically significant differences in power reflectance below 1,000 Hz for individuals with surgically confirmed otosclerosis compared with a group of individuals with normal middle-ear function. However, the authors noted that the range of variability for power reflectance for otosclerotic ears overlapped considerably with the power reflectance in the normal middle-ear group; this overlapping variability would make it difficult to detect the presence of otosclerosis on an individual case-by-case basis.

Nakajima et al. (2012) presented interesting WAI findings from individuals with superior semicircular canal dehiscence (SCD). Although the site of lesion for SCD is not in the middle ear, the "third window" created by the dehiscence in the semicircular canal allows energy to leave the inner ear via the dehiscence, which can result in a decreased impedance at the oval window. For six ears with SCD, Nakajima et al. (2012) noted a consistent notching pattern in the power reflectance close to 1,000 Hz. These results suggest that WAI may be a useful tool to help diagnose SCD.

Predicting Conductive Hearing Loss

Whereas increased TW and decreased Y_{tm} are sometimes associated with CHL, conventional tympanometric characteristics are not accurate predictors of CHL. Results from a number of studies suggest that WAI tests may be accurate predictors of CHL (Keefe et al., 2012; Keefe and Simmons, 2003; Piskorski et al., 1999; Prieve et al., 2013). Building on work by Piskorski et al. (1999) and Keefe and Simmons (2003), Keefe et al. (2012) tested the hypothesis that WAI accurately predicts CHL in young children suspected of having OME. The reference standard for identification of CHL was air–bone gaps (ABGs) at octave frequencies from 250 to 4,000 Hz, based on behaviorally measured audiometric thresholds. Absorbance and conventional 226-Hz tympanometric measurements were obtained from 25 children (36 ears aged 3.5 to 8.2 years) with CHL and 23 children (44 ears aged 2.6 to 8.2 years) with normal hearing. For WAI measurements, a likelihood ratio was calculated (using the average mean and standard deviation of WAI responses across frequency, weighted more heavily where differences in WAI between the OME and control groups were greater) to predict hearing status (e.g., CHL or normal). The area under the receiver operating characteristic curve (AUC) was calculated using criterion ABGs of ≥20, 25, and 30 dB. WAI and conventional tympanometric predictors were evaluated for individual octave frequencies and for a range of frequencies (250 to 4,000 Hz) at which a CHL was present. Results showed WB absorbance as the best overall predictor of CHL with AUC values ≥0.97. These results support the hypothesis that WAI tests are accurate predictors of CHL in children, offering improved test performance for predicting CHL relative to conventional tympanometric measurements.

Effects of Maturation and Aging

Understanding maturational and aging-related changes in the middle ear and their effects on transfer of sound power through the middle ear is important for our understanding of developmental processes in the auditory system and for the development of clinical norms for hearing and middle-ear assessment. These changes may also impact the interpretation of ABR tests and to a greater extent OAE measurements, which depend on both forward and reverse transfer of sound power through the middle ear.

Several studies have examined WAI responses in the newborn period (Aithal et al., 2013; Hunter et al., 2010; Sanford et al., 2009) with aims of examining developmental effects on WAI or comparing WAI measurements from ears that either passed or were referred on newborn hearing tests, such as OAEs. WAI data from these studies of infants in the newborn period are in general agreement and the overall shape and magnitude of the WAI data are similar. However, studies involving infants ranging in age from just a few days and into childhood have revealed significant age-related differences in WAI (Keefe et al., 1993; Werner et al., 2010) and results suggest that dramatic changes in WAI occur throughout infancy. Figure 9.21, containing replotted data from Keefe et al. (1993), shows the most dramatic changes in absorbance occurring during the first year of life; however, differences in absorbance persist beyond 12 months of age. Sanford et al. (2009) and Hunter et al. (2010) reported test performance results for WAI and 1,000-Hz tympanometry in terms of ability to predict newborn hearing-screening outcomes based on DPOAE screening outcomes. For large numbers of ears, both studies showed that WAI had high sensitivity and specificity for predicting DPOAE screening outcomes and outperformed 1,000-Hz tympanometry.

Relative to WAI data from newborns, a limited number of studies have evaluated WAI data from toddlers, young children, and adolescents and results are inconsistent with respect to identification of significant age-related differences in WAI (Beers et al., 2010; Hunter et al., 2008). Beers et al. (2010) compared WAI data from children ranging in age from 5 to 7 years to WAI data from adult subjects ranging in age from 22 to 32 years and found a significant difference in WAI for frequencies ranging from 310 to 1,250 Hz. However, Hunter et al. (2008) examined WAI data from children aged 6 months to 4 years and reported a lack of significant differences across age, other than for high frequencies. Other studies have reported WAI data for children with normal middle-ear function. Ellison et al. (2012) reported

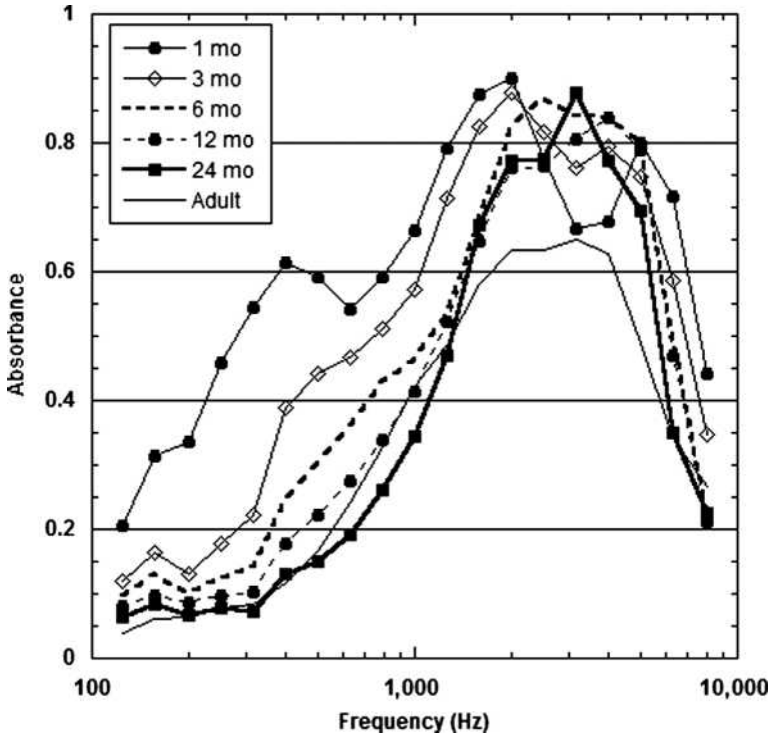


FIGURE 9.21 Wideband absorbance data, plotted as a function of frequency (Hz), from infants (1, 3, 6, 12, and 24 months) and adults. (Replotted data from Keefe DH, Bulen JC, Arehart KH, Burns EM. (1993) Ear-canal impedance and reflection coefficient in human infants and adults. *J Acoust Soc Am.* 94, 2617–2638 data.)

WAI data for 44 children with a median age of 1.2 years and Keefe et al. (2012) reported WAI data for 26 children with a mean age of 5.5 years. A comparison of the overall shape and magnitude of the WAI data from both studies reveals similar results for the 1- and 5-year-old children; however, WAI data from both groups are somewhat different compared to adult WAI data. Although reasons for these differences are not clear, differences in equipment and calibration methods may be contributing factors. Although the specific maturational influences on WAI data in young children have not been pinpointed, postnatal increases in middle-ear cavity volume, which continues throughout childhood, may be an influence (Anson and Donaldson, 1981). Furthermore, Eby and Nadol (1986) reported that mastoid bone dimensions increase in growth spurts, the first occurring between birth and approximately 7 years of age and the second occurring between ages 11 and 15 years.

It is believed that the TM and middle ear undergo anatomical and physiological changes with advancing age, which cause an increase in middle-ear stiffness (Ruah et al., 1991). Feeney and Sanford (2004) examined 226-Hz tympanometry and WAI in a group of 40 young adults (18 to 28 years) and a group of 30 older adults (60 to 85 years). Whereas the 226-Hz admittance tympanometry data from the two groups were not different, there were significant age effects observed for the absorbance data. Specifically, the older group exhibited a comparative increase in absorbance for frequencies ranging from 800 to 2,000 Hz and a decrease at approximately 4,000 Hz. These results suggest a decrease in middle-ear stiffness as a function of age; these findings are contrary to what would be expected based on the anatomical studies cited above.

Conclusions

An important part of translating WAI techniques to clinically useful tests is to identify alternative ways of analyzing the large amount of data obtained with WAI measurements. Whereas a *qualitative,* pattern recognition approach may be informative in individual cases, *quantitative* analysis techniques will be important for more accurate interpretation of WAI measurements. Although strategies to simplify large, multivariate data sets to univariate predictors have shown promising results (Hunter et al., 2010; Keefe et al., 2012; Sanford et al., 2009; and others), additional approaches, with goals of making data analysis and interpretation relatively straightforward, should improve the clinical utility of WAI tests. In addition, investigations should seek to identify key WAI characteristics for both normal and disordered ears in an effort to develop middle-ear tests with high sensitivity and specificity. Work is also needed to construct normative databases for a variety of age groups since age-related differences in ambient and tympanometric WAI have been reported. With new WAI technology and equipment options becoming available, the development of clinically friendly features will be an important factor for advancements in research and clinical utility of WAI measurements.

FOOD FOR THOUGHT

In this chapter, we have covered principles that govern middle-ear function and how it can be measured clinically. If one could apply a known force directly to different parts of the ear such as the TM, the ossicles, or the oval window,

it would be possible to determine precisely how effectively each of these parts of the ear is able to respond to the applied force. This would theoretically provide exact measures of the impedance properties of each part of the ear. However, it must be appreciated that none of these anatomical components operate in isolation to transmit sound energy. The outer ear and middle ear represent a functionally connected mechanical system, and so changes in impedance for one part affect the function of the entire system. Using acoustic immittance measurements, we are able to indirectly infer the function of the entire middle-ear system by applying a known force to the input of the system, at the plane of the TM, and then measuring how that force is altered. If we measure this input sound compared to the resultant sound as a function of frequency, we can gain an appreciation for how the middle ear reacts in a dynamic way across the speech frequency range. These measures can then be compared to other functional measures, such as audiometry and OAEs, to determine possible pathology and functional impact.

Single-frequency tympanometry using a single low-frequency probe tone and measuring admittance with qualitative type interpretation (A, B, and C types) has been the standard practice in audiometry for over 50 years. This simplified procedure has persisted for so long because it is straightforward, quick, and able to detect OME with reasonable accuracy. Gaining additional diagnostic sensitivity and specificity through more sophisticated stimuli and measurement may be desirable.

The primary clinical assets of MFT are the ability to assess the relative contributions of mass and stiffness of the middle ear and to help identify middle-ear RF. Some of the challenges associated with MFT tests include equipment constraints, which limit the upper frequency range to 2,000 Hz, which could limit identification of RF in some individuals (Shanks et al., 1993). In addition, the more complicated multifrequency tympanometric response patterns are often difficult for clinicians to interpret and the frequency range of what is currently considered normal is fairly wide (Margolis and Goycoolea, 1993). However, MFT test results, in conjunction with other audiometric test findings, can be used to provide more information regarding middle-ear function than would be available from either test interpreted in isolation.

Results from studies using commercial WAI systems are demonstrating greater diagnostic accuracy relative to traditional middle-ear measurement techniques; however, more studies of diagnostic accuracy are needed. It is our hope that future generations of audiologists will be able to make use of these advanced techniques and contribute to the literature regarding their clinical utility. Here are some thoughts we should be aware of and try to answer:

1. Discuss how single-frequency tympanometry differs from multi-frequency and wideband tympanometry, including any evidence that sensitivity is improved with the addition of multiple frequencies or broad-band stimuli.
2. Describe how tympanometry changes with development from newborn to childhood age ranges, and how this affects sensitivity of tympanometry to middle ear effusion.
3. How does increased stiffness affect tympanometry shape and resonant frequency, and what conditions are primarily due to increased stiffness?

REFERENCES

Aithal S, Kei J, Driscoll C, Khan A. (2013) Normative wideband reflectance measures in healthy neonates. *Int J Pediatr Otorhinolaryngol.* 77, 29–35.

American Academy of Audiology. (2011) Childhood hearing screening guidelines. Available online at: http://www.audiology.org/resources/documentlibrary/pages/pediatricdiagnostics.aspx (accessed January 6, 2014).

American Speech-Language-Hearing Association. (1997) Guidelines for screening infants and children for outer and middle ear disorders, birth through 18 years. In: *Guidelines for Audiologic Screening.* Rockville, MD: Author; pp 15–22.

American Speech-Language-Hearing Association. (2004) Guidelines for the audiologic assessment of children from birth to 5 years of age. Available online at: www.asha.org/policy.

ANSI/ASA S3.39. (1987) *Specifications for Instruments to Measure Aural Acoustic Impedance and Admittance (Aural Acoustic Immittance)* (R2012). New York: American National Standards Institute.

Anson BJ, Donaldson JA. (1981) *Surgical Anatomy of the Temporal Bone and Ear.* Philadelphia, PA: Saunders.

Baldwin M. (2006) Choice of probe tone and classification of trace patterns in tympanometry undertaken in early infancy. *Int J Audiol.* 45, 417–427.

Beers AN, Shahnaz N, Westerberg BD, Kozak FK. (2010) Wideband reflectance in normal Caucasian and Chinese school-aged children and in children with otitis media with effusion. *Ear Hear.* 31, 221–233.

Bennett M. (1975) Acoustic impedance bridge measurements with the neonate. *Br J Audiol.* 9, 117–124.

Bluestone CD. (1975) Assessment of Eustachian tube function. In: Jerger J, ed. *Handbook of Clinical Impedance Audiometry.* Dobbs Ferry, NY: American Electromedics; pp 127–148.

Brooks DN. (1968) An objective method of determining fluid in the middle ear. *Int Audiol.* 7, 280–286.

Colletti V. (1975) Methodologic observations on tympanometry with regard to the probe tone frequency. *Acta Otolaryngol.* 80, 54–60.

Eavey RD. (1993) Abnormalities of the neonatal ear: otoscopic observations, histologic observations, and a model for contamination of the middle ear by cellular contents of amniotic fluid. *Laryngoscope.* 103 (suppl 58), 1–31.

Eby TL, Nadol JB. (1986) Postnatal growth of the human temporal bone. *Ann Otol Rhinol Laryngol.* 95, 356–364.

Ellison JC, Keefe DH, Fitzpatrick DF, Gorga MP, Cohn ES, Sanford CA. (2012) Wideband acoustic transfer functions predict middle-ear effusion. *Laryngoscope.* 22, 887–894.

Feeney MP, Grant IL, Marryott LP. (2003) Wideband energy reflectance measurements in adults with middle-ear disorders. *J Speech Lang Hear Res.* 46:901–911.

Feeney MP, Hunter LL, Kei J, Lilly DJ, Margolis RH, Nakajima HH, et al. (2013) Consensus statement: Eriksholm workshop on wideband absorbance measures of the middle ear. *Ear Hear.* 34 (suppl 1), 78S–79S.

Feeney MP, Keefe DH. (2012) Physiological mechanisms assessed by aural acoustic transfer functions. In: Tremblay J, Burkard R, eds. *Translational Perspectives in Auditory Neuroscience: Hearing Across the Life Span-Assessment and Disorders.* San Diego, CA: Plural Publishing; pp 85–122.

Feeney MP, Sanford CA. (2004) Age effects in the human middle ear: wideband acoustical measures. *J Acoust Soc Am.* 116, 3546–3558.

Feldman AS. (1976) Tympanometry – procedures, interpretations and variables. In: Feldman AS, Wilber LA, eds. *Acoustic Impedance and Admittance – The Measurement of Middle Ear Function.* Baltimore, MD: Williams and Wilkins; pp 103–155.

Himelfarb MZ, Popelka GR, Shanon E. (1979) Tympanometry in normal neonates. *J Speech Lang Hear Res.* 22, 179–191.

Holte L, Margolis RH, Cavanaugh R. (1991) Developmental changes in multifrequency tympanograms. *Audiology.* 30, 1–24.

Hunter LL, Feeney MP, Lapsley Miller JA, Jeng PS, Bohning S. (2010) Wideband reflectance in newborns: normative regions and relationship to hearing-screening results. *Ear Hear.* 31, 599–610.

Hunter LL, Margolis RH. (1992) Multifrequency tympanometry: current clinical application. *Am J Audiol.* 1, 33–43.

Hunter LL, Prieve BA, Kei J, Sanford CA. (2013) Pediatric applications of wideband acoustic immittance measures. *Ear Hear.* 34 (suppl 1), 36S–42S.

Hunter LL, Shahnaz N. (2014) *Acoustic Immittance Measures: Basic and Advanced Practice.* San Diego, CA: Plural Publishing.

Hunter LL, Tubaugh L, Jackson A, Propes S. (2008) Wideband middle ear power measurement in infants and children. *J Am Acad Audiol.* 19, 309–324.

Jerger J. (1970) Clinical experience with impedance audiometry. *Arch Otolaryngol.* 92, 311–324.

Keefe DH, Bulen JC, Arehart KH, Burns EM. (1993) Ear-canal impedance and reflection coefficient in human infants and adults. *J Acoust Soc Am.* 94, 2617–2638.

Keefe DH, Ling R, Bulen JC. (1992) Method to measure acoustic impedance and reflection coefficient. *J Acoust Soc Am.* 91, 470–485.

Keefe DH, Sanford CA, Ellison JC, Fitzpatrick DF, Gorga MP. (2012) Wideband aural acoustic absorbance predicts conductive hearing loss in children. *Int J Audiol.* 51, 880–891.

Keefe DH, Simmons JL. (2003) Energy transmittance predicts conductive hearing loss in older children and adults. *J Acoust Soc Am.* 114, 3217–3238.

Kei J, Allison-Levick J, Dockray J, Harrys R, Kirkegard C, et al. (2003). High-frequency (1000 Hz) tympanometry in normal neonates. *Journal of the American Academy of Audiology*. 14, 20–28.

Kei J, Sanford CA, Prieve BA, Hunter LL. (2013) Wideband acoustic immittance measures: developmental characteristics (0–12 months). *Ear Hear.* 34, 17–26.

Keith RW. (1973) Impedance audiometry with neonates. *Arch Otolaryngol.* 97, 465–476.

Koebsell KA, Margolis RH. (1986) Tympanometric gradient measured from normal preschool children. *Audiology.* 25, 149–157.

Lidén G. (1969) The scope and application of current audiometric tests. *J Laryngol Otol.* 83, 507–520.

Lidén G, Peterson JL, Björkman G. (1970) Tympanometry. *Arch Otolaryngol.* 92, 248–257.

Liu Y, Sanford CA, Ellison JC, Fitzpatrick DF, Gorga MP, Keefe DH. (2008) Wideband absorbance tympanometry using pressure sweeps: system development and results on adults with normal hearing. *J Acoust Soc Am.* 124, 3708–3719.

Marchant CD, McMillan PM, Shurin PA, Johnson CE, Turczyk VA, Feinstein JC, et al. (1986) Objective diagnosis of otitis media in early infancy by tympanometry and ipsilateral acoustic reflex thresholds. *J Pediatr.* 109, 590–595.

Margolis RH, Bass-Ringdahl S, Hanks WD, Holte L and Zapala DA. (2003). Tympanometry in newborn infants: 1 kHz norms. *Journal of the American Academy of Audiology*. 14, 383–392.

Margolis RH, Goycoolea HG. (1993) Multifrequency tympanometry in normal adults. *Ear Hear.* 14, 408–413.

Margolis RH, Heller JW. (1987) Screening tympanometry: criteria for medical referral. *Audiology.* 26, 197–208.

Margolis RH, Saly GL, Keefe DH. (1999) Wideband reflectance tympanometry in normal adults. *J Acoust Soc Am.* 106, 265–280.

Margolis RH, Shanks JE. (1985) Tympanometry. In: Katz J, ed. *Handbook of Clinical Audiology.* 3rd ed. Baltimore, MD: Williams & Wilkins; pp 438–475.

Margolis RH, Smith P. (1977) Tympanometric asymmetry. *J Speech Hear Res.* 20, 437–446.

McGrath AP, Michaelides EM. (2011) Use of middle ear immittance testing in the evaluation of patulous Eustachian tube. *J Am Acad Audiol.* 22, 201–207.

Nakajima HH, Pisano D, Roosli C, Hamade MA, Merchant GR, Mohfoud L, et al. (2012) Comparison of ear-canal reflectance and umbo velocity in patients with conductive hearing loss: a preliminary study. *Ear Hear.* 33, 35–43.

Nakajima HH, Rosowski JJ, Shahnaz N, Voss SE. (2013) Assessment of ear disorders using power reflectance. *Ear Hear.* 34 (suppl 1), 48S–53S.

Nozza RJ, Bluestone CD, Kardatzke D, Bachman R. (1992) Towards the validation of aural acoustic immittance measures for diagnosis of middle ear effusion in children. *Ear Hear.* 13, 442–453.

Nozza RJ, Bluestone CD, Kardatzke D, Bachman R. (1994) Identification of middle ear effusion by aural acoustic admittance and otoscopy. *Ear Hear.* 15, 310–323.

Ogut F, Serbetcioglu B, Kirazli T, Kirkim G, Gode S. (2008) Results of multiple-frequency tympanometry measures in normal and otosclerotic middle ears. *Int J Audiol.* 47, 615–620.

Paradise JL, Smith CG, Bluestone CD. (1976) Tympanometric detection of middle ear effusion in infants and young children. *Pediatrics.* 58, 198–210.

Piskorski P, Keefe DH, Simmons JL, Gorga MP. (1999) Prediction of conductive hearing loss based on acoustic ear-canal response using a multivariate clinical decision theory. *J Acoust Soc Am.* 105, 1749–1764.

Prieve BA, Calandruccio L, Fitzgerald T, Mazevski A, Georgantas LM. (2008) Changes in transient-evoked otoacoustic emission levels with negative tympanometric peak pressure in infants and toddlers. *Ear Hear.* 29, 533–542.

Prieve BA, Feeney MP, Stenfelt S, Shahnaz N. (2013) Prediction of conductive hearing loss using wideband acoustic immittance. *Ear Hear.* 34 (suppl 1), 54S–59S.

R de Jonge. (1986) Normal tympanometric gradient: a comparison of three methods. *Audiology.* 25, 299–308.

Roup CM, Wiley TL, Safady SH, Stoppenbach DT. (1998) Tympanometric screening norms for adults. *Am J Audiol.* 7, 55–60.

Roush J, Bryant K, Mundy M, Zeisel S. & Roberts J. (1995) Developmental changes in instatic admittance and tympanometric width in infants and toddlers. *Journal of American Academy of Audiology.* 6, 334–338.

Roush J, Drake A, Sexton JE. (1992) Identification of middle ear dysfunction in young children: a comparison of tympanometric screening procedures. *Ear Hear.* 13, 63–69.

Ruah CB, Schachern PA, Zelterman D, Paparella MM, Yoon TH. (1991) Age related morphologic changes in the human tympanic membrane. *Arch Otolaryngol Head Neck Surg.* 117, 627–634.

Sanford CA, Brockett JE. (in press) Characteristics of wideband acoustic immittance in ears with middle ear dysfunction. *J Am Acad Audiol.*

Sanford C, Keefe DH, Liu YW, Fitzpatrick D, McCreery RW, Lewis DE, et al. (2009) Sound-conduction effects on distortion-product otoacoustic emission screening outcomes in newborn infants: test performance of wideband acoustic transfer functions and 1-kHz tympanometry. *Ear Hear.* 30, 635–652.

Sanford CA, Schooling T, Frymark T. (2012) Determining the presence or absence of middle-ear disorders: an evidence-based systematic review on the diagnostic accuracy of selected assessment instruments. *Am J Audiol.* 21, 251–268.

Shahnaz N. (2008) Wideband reflectacne in neonatal intensive care unit. *Journal of the American Academy of audiology*. 19 (5), 419–429.

Shahnaz N, Bork K, Polka L, Longridge N, Bell D, Westerberg BD. (2009) Energy reflectance and tympanometry in normal and otosclerotic ears. *Ear Hear.* 30, 219–233.

Shahnaz N, Davies D. (2006) Standard and multifrequency tympanometric norms for Caucasian and Chinese young adults. *Ear Hear.* 27 (1), 75–90.

Shahnaz N, Feeney MP, Schairer KS. (2013) Wideband acoustic immittance normative data: ethnicity, gender, aging, and instrumentation. *Ear Hear.* 34 (suppl 1), 27S–35S.

Shahnaz N, Miranda T, Polka L. (2008) Multifrequency tympanometry in neonatal intensive care unit and well babies. *J Am Acad Audiol.* 19, 392–418.

Shahnaz N, Polka L. (1997) Standard and multifrequency tympanometry in normal and otosclerotic ears. *Ear Hear.* 18, 326–341.

Shanks JE, Lilly DJ. (1981) An evaluation of tympanometric estimates of ear canal volume. *J Speech Hear Res.* 24, 557–566.

Shanks JE, Wilson RH, Cambron NK. (1993) Multiple frequency tympanometry: effects of ear canal volume compensation on static acoustic admittance and estimates of middle ear resonance. *J Speech Hear Res.* 36, 178–185.

Sprague BH, Wiley TL, Goldstein R. (1985) Tympanometric and acoustic-reflex studies in neonates. *J Speech Hear Res.* 28, 265–272.

Stinson MR, Shaw EA, Lawton BW. (1982) Estimation of acoustical energy reflectance at the eardrum from measurements of pressure distribution in the human ear canal. *J Acoust Soc Am.* 72, 766–773.

Terkildsen K, Thomsen KA. (1959) The influence of pressure variations on the impedance of the human ear drum. *J Laryngol Otol.* 73, 409–418.

Trine MB, Hirsch JE, Margolis RH. (1993) The effect of middle ear pressure on transient evoked otoacoustic emissions. *Ear Hear.* 14, 401–407.

Vanhuyse VJ, Creten WL, Van Camp KJ. (1975) On the W-notching of tympanograms. *Scand Audiol.* 4, 45–50.

Voss SE, Allen JB. (1994) Measurement of acoustic impedance and reflectance in the human ear canal. *J Acoust Soc Am.* 95, 372–384.

Voss SE, Merchant GR, Horton NJ. (2012) Effects of middle-ear disorders on power reflectance measured in cadaveric ear canals. *Ear Hear.* 33, 195–208.

Werner LA, Levi EC, Keefe DH. (2010) Ear-canal wideband acoustic transfer functions of adults and two- to nine-month-old infants. *Ear Hear.* 31, 587–598.

Wiley TL, Cruickshanks KJ, Nondahl DM, Tweed TS, Klein R, Klein BEK. (1996) Tympanometric measures in older adults. *J Am Acad Audiol.* 7, 260–268.

Wiley TL, Fowler CG. (1997) *Acoustic Immittance Measures in Clinical Audiology. A Primer.* San Diego, CA: Singular Publishing.

Zhiqi L, Ku Y, Zhiwu H. (2010) Tympanometry in infants with middle ear effusion having been identified using spiral computerized tomography. *Am J Otolaryngol.* 31, 96–103.

CHAPTER 10

Acoustic Stapedius Reflex Measurements

M. Patrick Feeney and Kim S. Schairer

INTRODUCTION

Metz (1952) measured middle ear impedance and found that it increased when a loud sound was presented to the opposite ear. He hypothesized that the increased impedance was because of middle ear muscle reflexes (MEMR) which stiffened the middle ear system. Metz reported patterns of reflexes in individuals with normal hearing, cochlear hearing loss, and hearing loss due to otosclerosis and vestibular schwannoma. He reported that MEMR thresholds were similar between ears with normal hearing and ears with mild to moderate hearing loss and loudness recruitment (presumably a cochlear site of lesion, e.g., **Ménière** disease). In contrast, no recruitment or reflex was observed in ears with acoustic neuromas (retrocochlear site of lesion) or otosclerosis (middle ear site of lesion). Since Metz's initial observations, MEMR tests have become a standard component of the clinical test battery (1) to cross-check behavioral results, and (2) in conjunction with other test results, to differentiate among middle ear, cochlear, retrocochlear and even non-auditory sites of lesion (e.g., superior semicircular canal dehiscence).

Anatomy and Acoustic Stapedius Reflex Pathways

The two middle ear muscles are the tensor tympani and the stapedius. The body of the tensor tympani muscle is located in a canal above the Eustachian tube in the anterior-medial wall of the middle ear. The tendon stretches from the body of the tensor tympani muscle to the manubrium of the malleus. When the muscle contracts, the tendon pulls the malleus anteriorly and medially, which stiffens the ossicular chain and tympanic membrane (TM). This muscle is innervated by cranial nerve V which is also called the trigeminal nerve. The tensor tympani may contract in response to tactile stimulation or as a startle response to loud unexpected sounds. However, the tensor tympani does not typically contribute to the MEMR measured clinically in humans.

The stapedius muscle is the smallest skeletal muscle in the human body, and it is the main contributor to the MEMR measured clinically in humans. Therefore, in this chapter, the response will be called the acoustic stapedius reflex (ASR). The body of the stapedius muscle is located in the pyramidal eminence, or small bony protrusion, on the posterior wall of the middle ear. The tendon stretches anteriorly from the body of the stapedius muscle to the posterior surface of the neck of the stapes. The stapedius muscle is innervated by the motor branch of cranial nerve VII which is also called the facial nerve. When an intense sound is presented to the ear, the stapedius muscle contracts and pulls the head of the stapes posteriorly toward the muscle body, which causes an increase in stiffness of the ossicular chain and the TM. The stiffening causes a decrease in admittance of sound into the middle ear that can be measured by a probe in the ear canal, and this is the basis of clinical ASR measurements. The stapedius reflex also can be activated by vocalizations, chewing, yawning, and tactile stimulation.

The ASR is a bilateral response which means that when an activating stimulus is presented to one ear the stapedius muscle contracts in both ears. Therefore, the ASR may be measured in the same ear in which the activating stimulus is presented (ipsilateral conditions) or in the opposite ear (contralateral conditions). There are four overlapping pathways of the reflex arc, including two ipsilateral and two contralateral pathways (Borg, 1973; Lyons, 1978). One ipsilateral pathway is represented by the black arrows in Figure 10.1 and includes the ipsilateral ear canal, middle ear, cochlea, VIIIth nerve, ventral cochlear nucleus (VCN), medial superior olivary complex (MSO), motor nucleus of the facial nerve (MN VII), VIIth nerve, and stapedius muscle. The second ipsilateral pathway includes a connection between the VCN and the ipsilateral MN VII. One contralateral pathway represented by gray arrows in Figure 10.1 includes the middle ear, cochlea, VIIIth nerve, VCN, and MSO on the side of the activating stimulus, and the MN VII, VIIth nerve, and stapedius muscle on the contralateral side. The second contralateral pathway includes a connection between the ipsilateral VCN and the contralateral MSO which connects with the MN VII.

As the intensity of the eliciting stimulus increases, the amplitude of the ASR response increases, and an example of this response growth measured with a clinical immittance system is shown in Figure 10.2. The latency of the acoustic reflex varies by activator (the stimulus used to induce the ASR), but in general it is around 100 ms which reflects the travel time

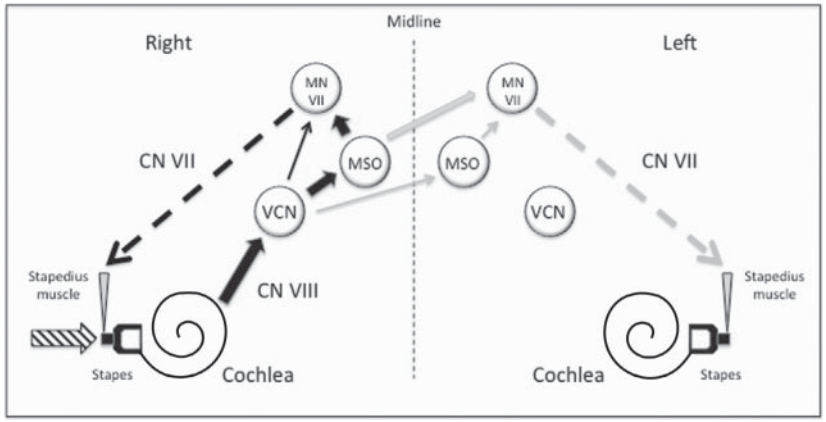


FIGURE 10.1 Right ipsilateral and right contralateral acoustic stapedius reflex pathways are shown as if facing the patient. The activator stimulus (*striped arrow*) is presented to the right ear canal. The pathway is through the right middle ear and cochlea to the VIIIth cranial nerve (*CN VIII*). From that point, the right ipsilateral reflex arc (*black arrows*) consists of the ventral cochlear nucleus (*VCN*), medial superior olivary complex (*MSO*), and motor nucleus of the VIIth cranial nerve (*MN VII*), which results in efferent stimulation (*dashed black arrow*) of the stapedius muscle via the VIIth cranial nerve (*CN VII*). A second ipsilateral path goes directly from the VCN to the MN VII. The right contralateral reflex arc (*gray arrows*) is simultaneously activated from the ipsilateral VCN to the ipsilateral MSO, to the contralateral MN VII, which results in efferent stimulation (*dashed gray arrow*) of the contralateral stapedius muscle via the CN VII. A second path goes from the ipsilateral VCN to the contralateral MSO, and the remaining pathway.

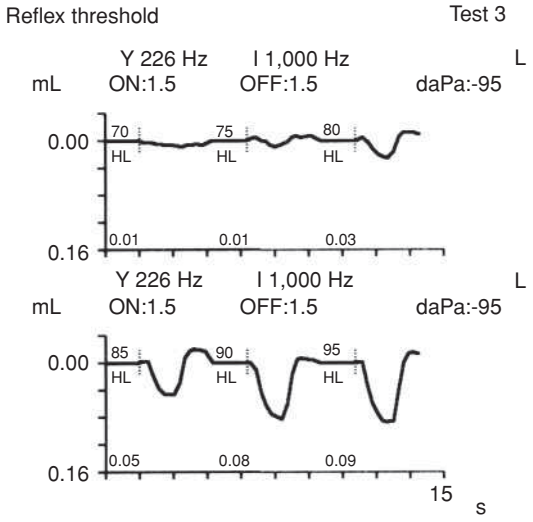


FIGURE 10.2 Acoustic stapedius reflex (ASR) response growth in an adult human ear with normal middle ear function and hearing in the left ipsilateral condition elicited by a 1,000-Hz activator (226-Hz probe tone) at levels of 70 to 95 dB HL. Response amplitude is plotted in mmho as a function of time in seconds, with three activator levels on the top row and three activator levels on the bottom row. The numbers at the bottom of each plot ranging from 0.01 to 0.09 are the amplitudes. ASR is not elicited by 70- or 75-dB HL activators. However, from 80 to 95 dB HL the ASR is present and the amplitude increases as the activator level increases.

of the signal from the cochlea through the pathway to the stapedius muscle, and when the response is being clinically measured, the latency includes the inherent delay of the measurement system (e.g., Qui and Stucker, 1998). ASR increased latencies have been reported in ears with retrocochlear disorders such as vestibular schwannomas (e.g., Clemis and Samo, 1980) and it has been suggested that latency may be useful in separating cochlear from retrocochlear sites of lesion. However, ASR latency is not routinely measured clinically; rather amplitude is usually measured to establish threshold.

Theories of Functional Significance

There are two main theories about the functional significance of the ASR. First, it was thought that the ASR reduces the amount of sound pressure that reaches the cochlea, and therefore it has a protective effect from high-level sounds (e.g., Brask, 1979). For example, temporary threshold shifts (TTS) in hearing were observed after intense stimulation on the affected side of listeners with Bell palsy during an episode of facial paralysis, but no TTS (or less TTS) was observed on the unaffected side or on the affected side after recovery from an episode (Brask, 1979). The main problems with this theory are that the ASR (1) is not fast enough to protect the cochlea from high-level transient sounds and (2) the ASR can fatigue and thresholds may increase in the presence of high-level, long-duration sounds (e.g., Gerhardt and Hepler, 1983). In contrast, Borg et al. (1982) reported ASR-mediated resistance from fatigue in the presence of long-duration industrial noise. Therefore, the protective effect (or lack thereof) is not entirely clear and, if present, may

depend on the frequency response, duration, and level of the sound to which listeners are exposed.

The second theory is that the ASR provides humans with an advantage for understanding speech in noise because lower frequencies are attenuated relative to higher frequencies when the stapedius muscle contracts. For example, Aiken et al. (2013) reported that listeners with stapedius tendons that were transected during stapedotomy had poorer speech discrimination scores in moderate levels of noise in comparison to listeners with intact ASR, but that this benefit did not persist for high levels of noise. It is not clear whether the effect provides protection from upward spread of masking (i.e., lower frequencies masking higher frequencies) at high speech levels in quiet. Some authors have reported no "rollover," or worsening in speech discrimination scores with increases in stimulus level, in individuals with Bell palsy (e.g., Phillips et al., 2002), whereas other authors have reported significant rollover (e.g., Wormald et al., 1995). For a more detailed discussion of the theories of functional significance, see Borg et al. (1984).

Relevance to Clinical Practice

ASR threshold and decay tests can be included in a test battery, along with tympanometry, otoacoustic emissions (OAEs), and behavioral puretone and speech tests, to differentiate among middle ear, cochlear, and retrocochlear sites of lesion. ASR threshold also can be used as a cross-check with the behavioral audiogram to increase confidence in the diagnosis of hearing loss in young children with whom behavioral results may be questionable, and in older children and adults who may present with false or exaggerated hearing loss. Finally, the ASR thresholds can be used in cochlear implant assessments to verify function of the device and to set minimum or maximum stimulation levels in young children who cannot provide reliable threshold and loudness information. ASR thresholds are typically obtained with multiple puretone activators and with ipsilateral and contralateral stimulation. The pattern of thresholds across conditions is examined and compared with puretone behavioral thresholds and speech recognition scores. The procedure for obtaining ASR thresholds is described in the next section, and patterns of ASR thresholds that can be associated with a variety of underlying pathologies are described in the section on disorders.

MEASUREMENT OF ASR THRESHOLD

Terminology

Two stimuli are currently used for clinical measurement of the ASR. The *probe* is a puretone that is presented continuously to the ear in which the response is to be measured. In early clinical equipment and studies the probe tone was 220 Hz. In current equipment, the standard probe tone frequency for children approximately 6 months of age through adulthood is 226 Hz presented at 85 dB SPL (70 dB HL) (ANSI S3.39). A low-frequency probe tone is advantageous due to reduced interaction between the probe and the activator (see below) in ipsilateral test conditions in which both the probe and activator are presented in the same ear. The 220- or 226-Hz probe tones are well separated from higher-frequency activators. Despite this benefit of lower frequency probe tones, higher probe tone frequencies are required to increase the probability of observing an ASR in newborns and infants (see the Gender and Age Effects section).

ANSI (2012) refers to the high-level acoustic stimulus that elicits the ASR as simply "stimulus." However, "stimulus" can be considered a generic term (e.g., the probe is a stimulus), therefore, to be clear in this chapter the term *activator* will be used to refer to the stimulus used to induce the ASR. Activators that are used in clinical measurements include 500-, 1,000-, 2,000-, and 4,000-Hz puretones and broadband noise (BBN) of 1 to 2 seconds in duration. Although 4,000-Hz activators are often available on test equipment, this activator has relatively less diagnostic value because it is often elevated or absent in ears with normal hearing (Gelfand, 1984). Thresholds are lower for BBN than for puretone activators (e.g., see Wiley et al., 1987 normative data in Table 10.1). Activator levels are typically available in 5-dB steps and should be limited in level, particularly for ASR decay tests, due to potential for temporary or permanent threshold shift (see the ASR Decay section).

During an ASR test, the *admittance* (Ya) of the 226-Hz probe tone is continuously monitored. Admittance is the ease with which acoustic energy is admitted into the middle ear as estimated at the lateral plane of the TM. When the activator is presented, the stapedius muscle contracts, which stiffens the ossicular chain and TM, which creates a reduction in admittance of the probe tone at the TM. Therefore, the ASR is measured as a decrease in admittance of the probe tone at the TM when the activator is presented.

TABLE 10.1

Contralateral and Ipsilateral ASR Thresholds in dB HL for Puretone and BBN Activators in Young Adults with Normal Hearing (Wiley et al., 1987)

	Contralateral		Ipsilateral	
	Mean	SD	Mean	SD
500 Hz	84.6	6.3	79.9	5.0
1,000 Hz	85.9	5.2	82.0	5.2
2,000 Hz	84.4	5.7	86.2	5.9
4,000 Hz	89.8	8.9	87.5	3.5
BBN	66.3	8.8	64.6	6.9

SD, standard deviation.

Although current clinical measurements of the ASR are plotted as decreases in admittance when the activator is presented, early measurements were often plotted as increases in impedance.

Instrumentation

ASR measurements should be completed using equipment that is calibrated to the ANSI S3.39 (2012) Specifications for Instruments to Measure Aural Acoustic Impedance and Admittance (Aural Acoustic Immittance). The equipment is the same as used for tympanometry and consists of a physical probe assembly (not to be confused with the probe tone stimulus) that is coupled to the ear with a small ear tip to create a hermetic seal. The instrument should have a manometer to monitor pressurization of the ear canal with an air pump, two speakers (one for the probe and one for the activator) and a microphone to measure the sound pressure level of the probe tone stimulus in the ear canal. An example probe assembly for an ipsilateral measurement is shown in Figure 10.3 (from Feeney and Sanford, 2008). Diagnostic equipment will have an additional cable and transducer through which an activator can be presented to the contralateral ear. The data should be plotted as admittance in mmho on the *Y*-axis and time in seconds on the *X*-axis. Diagnostic immittance equipment may have multiple activators, probe frequencies, and ipsilateral and contralateral selections. Screening immittance equipment may only have the ability to present an ipsilateral activator at a single frequency and level.

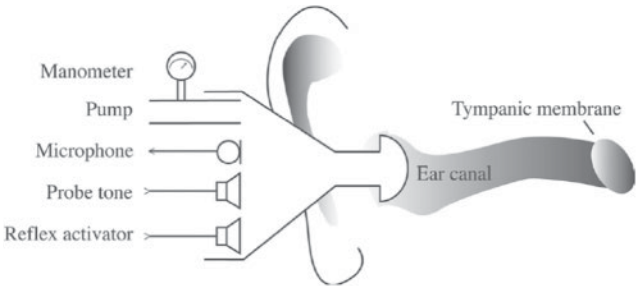


FIGURE 10.3 Components of a tympanometer probe assembly. The pressure pump varies the air pressure in the ear canal as monitored by the manometer. The pump is used to hold pressure in the ear canal at or near tympanometric peak pressure (TPP) during acoustic stapedius reflex (ASR) tests. One receiver is used to present the probe tone that is continuously monitored by the microphone during tympanometry and ASR tests. The second receiver is used to present the reflex activator to the same (ipsilateral) ear as shown in the figure. A cable is required to deliver the activator to the opposite ear for contralateral ASR tests (not shown). (From Feeney MP, Sanford CA. (2008) Middle-ear measurement. In: Madell J, Flexer C, eds. *Pediatric Audiology: Diagnosis, Technology, and Management*. New York: Thieme; pp 115–122.)

Methodology

The ASR is measured in the ear canal as a decrease in admittance, and therefore it should be measured at the pressure at which the admittance is the greatest, or tympanometric peak pressure (TPP). ASR thresholds may be higher if estimated at pressures above or below TPP (e.g., Martin and Coombes, 1974). The ASR is considered to be present if the admittance at TPP decreases by a criterion amount when the activator is presented. The criterion amount will depend on the manufacturer specifications, the normative data used, and the state of the patient. ASR thresholds have been found to be repeatable in adult ears with normal hearing and hearing loss (e.g., Forquer, 1979) and in newborns who passed a hearing screening (Kei, 2012; Mazlan et al., 2009).

The ASR test conditions are described based on the ear to which the activator is presented (ANSI, 2012). The four test conditions (left and right, ipsilateral and contralateral) are described in Table 10.2. In a typical test battery for a new patient, all four conditions are presented and the pattern of reflexes across conditions is examined. Different patterns can be associated with different underlying pathologies (see the section on disorders in this chapter). However, ipsilateral and contralateral conditions have their own relative advantages. Contralateral conditions have the advantages of (1) being sensitive to crossed pathways, and therefore mid-brainstem pathologies; (2) being less susceptible to artifact because the activator and probe stimuli are presented through separate transducers to the two ears; and (3) having more normative data available. Ipsilateral conditions have the advantages of (1) being sensitive to middle ear effects because the probe and activator are presented to the same ear; (2) ease of use in young children and other individuals who are difficult to test because it requires only one ear at a time; and (3) no concern for collapsed ear canals (for systems in which a supra-aural earphone is used to present a contralateral activator).

During manual ASR threshold estimation, the probe tone is presented continuously and the activator is manually presented in 5-dB steps either from higher to lower levels or

TABLE 10.2

Description of Stimulus Presentation Conditions for Ipsilateral and Contralateral ASR Measurements

Condition	Activator Ear	Probe Ear	Ear in Which ASR is Measured
Right contralateral	Right	Left	Left
Left ipsilateral	Left	Left	Left
Left contralateral	Left	Right	Right
Right ipsilateral	Right	Right	Right

from lower to higher levels. In controlled experiments, no significant differences were found between ASR thresholds obtained with ascending and descending approaches (e.g., Wilson, 1979), although lower thresholds can be observed on occasion in practice with a descending approach. The lowest level at which an activator elicits a criterion change in admittance is the ASR threshold for that activator. In a typical threshold search, the criterion change in admittance in the presence of the activator is 0.02 or 0.03 mmho, depending on the selected normative data, and the response should be time-locked to the onset of the stimulus (e.g., not associated with patient movement, swallowing). The response should be repeatable at the level defined as threshold with at least two presentations of the activator. Moreover, a response should be present (with possible growth of the admittance change) for an activator 5 dB above threshold as long as that does not exceed 105 dB HL. As activator levels increase, the amount of change in admittance will increase, although it may seem "upside down" because the measurement is actually a decrease in admittance. An example of ASR response growth to a 1,000-Hz activator is shown in Figure 10.2, with no ASR at 70 or 75 dB HL, and ASR responses of increasing size from 80 to 95 dB HL (0.03 to 0.09 mmho). Saturation of the response is often observed at higher levels in which increased activator levels elicit ASR responses, but the response magnitudes do not change. An example threshold search is shown in Figure 10.4 for an ipsilateral condition with a 1,000-Hz activator. Note that the ASR threshold test was completed at TPP which was -105 daPa. In this example, threshold is 80 dB HL, with two responses of at least 0.02 mmho amplitude at 80 dB HL, no response at 75 dB HL, and growth (i.e., an ASR response of larger magnitude, 0.06 mmho) at 85 dB HL. It should be noted that although an objective criterion is used (0.02 or 0.03 mmho), the determination of whether or not the reflex is a true reflex and not artifact is based on a subjective examination by the tester.

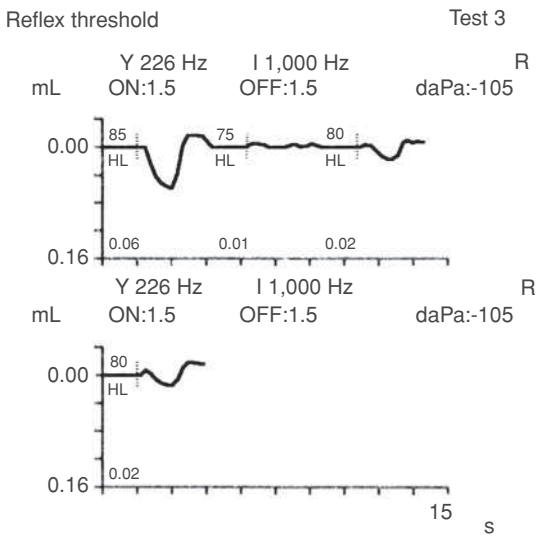


FIGURE 10.4 Acoustic stapedius reflex (ASR) threshold in an adult human ear with normal middle ear function and hearing in the right ipsilateral condition for a 1,000-Hz activator (226-Hz probe tone). Data are represented as in Figure 10.2. The ASR is absent at 75 dB HL, repeatable at 80 dB HL, and grows in amplitude at 85 dB HL. ASR threshold in this example is 80 dB HL.

Automated ASR tests may be available in both screening and diagnostic equipment. In screening equipment, the ASR test may be limited to ipsilateral, single-frequency (usually 1,000 Hz), single-level (90 or 95 dB HL), single presentation tests and the outcome is either pass or fail. Diagnostic equipment may have the same type of screening procedure, and in addition, they may have the ability to store user defined threshold estimation procedures. However, inclusion of an ASR test in hearing screening is not recommended (ASHA, 1990) due to contribution of absent ASRs to high false-positive and medical over-referral rates (e.g., Roush and Tait, 1985). Sells et al. (1997) suggest that the high referral rate found in previous studies may be related to the simultaneous presentation of the activator and probe in the ipsilateral screening mode of the tympanometer. The authors argued that outcomes may be improved by using a system that uses multiplexing (alternating presentation of the probe and activator) and, therefore, ipsilateral ASR would be appropriate for screening procedures.

Normative Data

ASR thresholds should be about 70 to 90 dB above behavioral air conduction thresholds at corresponding activator frequencies in ears with normal hearing (e.g., Metz, 1952). These general guidelines are predicated on the assumption that the ear canal is clear and middle ear function is normal. If the peak static acoustic admittance is very high, for example in an ear with a monomeric TM, the baseline admittance may be unstable. "Monomeric" refers to a spot on a TM that may be thin, or missing one or two of the normal three layers, due to repeated ear infections and/or perforation. This artifact of an unstable baseline may make it difficult if not impossible to observe the small changes in admittance that are associated with ASR activation. DiGiovanni and Ries (2007) found improved detection of the ASR in ears with high peak static acoustic admittance by setting ear canal pressure to –50 daPa relative to TPP.

Normative data for MEMR tests with a 226-Hz probe tone are available for normal hearing young adults in the ipsilateral and contralateral conditions for 500-, 1,000-, 2,000-, and 4,000-Hz puretones and for BBN (Table 10.1, data from Wiley et al., 1987). ASR threshold for a BBN activator is typically lower than for puretone activators, and threshold is often lower in ipsilateral than contralateral conditions. If normative data are used in a clinic, then the same procedures used in the study must be used in the clinic. One way to use the normative data is to consider MEMR thresholds above

the mean plus two standard deviations to be "abnormal." Another approach is to use the 90th percentile of normal data as the cutoff for abnormal ASRs. This issue is revisited in the section on disorders.

Normative data for MEMR thresholds as a function of behavioral puretone hearing thresholds are not available for ipsilateral conditions, but there are data available for contralateral conditions (Gelfand et al., 1990; Silman and Gelfand, 1981). The 10th, 50th, and 90th percentiles for ASR thresholds for 500-, 1,000-, and 2,000-Hz activators from Gelfand et al. (1990) are shown in Figure 10.5. The main trend is that median ASR threshold remains constant as behavioral threshold increases up to thresholds of about 50 dB HL. Above that level, ASR threshold increases as behavioral threshold increases. The 90th percentiles of ASR thresholds from Gelfand et al. (1990) and Silman and Gelfand (1981) are shown in tabular form in Table 10.3. If activator levels are restricted to 105 dB HL to protect hearing, the behavioral threshold above which a "no response" for ASR would be expected may be different for different activator frequencies. For example, in the Silman and Gelfand data, if a limit of 105 dB HL activator level is imposed, the highest associated behavioral threshold for a 500-Hz activator is 65 dB HL, whereas the highest behavioral threshold for a 1,000-Hz activator is 55 dB HL. The cutoffs are also slightly different between the Silman and Gelfand versus Gelfand et al. data.

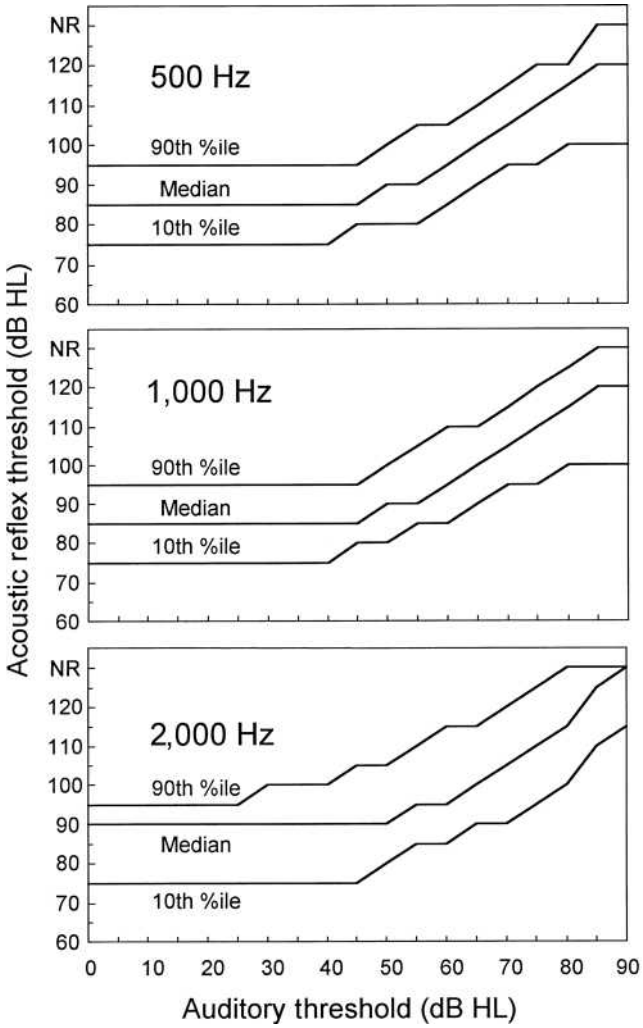


FIGURE 10.5 Acoustic stapedius reflex (ASR) threshold 10th, 50th, and 90th percentiles in dB HL as a function of behavioral threshold in dB HL for activators of 500 Hz (top), 1,000 Hz (middle) and 2,000 Hz (bottom) in adult ears with normal middle ear function. (Gelfand, 2009, pp 203; adapted from Gelfand SA, Schwander T, Silman S. (1990) Acoustic reflex thresholds in normal and cochlear-impaired ears; effects of no-response rates on 90th percentiles in a large sample. *J Speech Hear Disord.* 55, 198–205.)

Clinical Considerations

Gender and Age Effects

There are no significant effects of gender (Gelfand et al., 1990; Osterhammel and Osterhammel, 1979). However, there are two effects of age on ASR thresholds. First, ASR thresholds in adults increase with age for BBN activators beginning around 44 years of age (Silverman et al., 1983), and increase with age above 50 years for puretone activators above 2,000 Hz (Wilson and Margolis, 1999).

Second, higher probe tone frequencies must be used to increase the probability of observing an ASR in newborns and young infants. Weatherby and Bennett (1980) used probe frequencies from 200 to 2,000 Hz and found that probe frequencies from 800 to 1,800 Hz were associated with the lowest contralateral BBN ASR thresholds in newborns. McMillan et al. (1985a) tested newborns with 500-, 1,000-, 2,000-, and 4,000-Hz ipsilateral and contralateral activators, and found that the ASR was observed overall three times more often with the 660-Hz probe tone than with the 220-Hz probe tone. Sprague et al. (1985) reported that the ASR was observed more often with a 660-Hz probe in comparison to a 220-Hz probe in neonates for ipsilateral and contralateral conditions with 1,000-Hz and BBN activators. McMillan et al. (1985b) found similar results in older children 2 weeks to 12 months of age using the same activators and probe tone frequencies, with a higher rate of identification obtained with the 660-Hz probe than the 220-Hz probe. Although the normative database is still developing, contemporary ASR instruments with 1,000-Hz probe tones for infant tympanometry and ASR testing are currently available.

Normative data are available for adults with normal hearing for ipsilateral ASR thresholds (Wiley et al., 1987), and as a function of hearing loss for contralateral activators (Gelfand et al., 1990; Silman and Gelfand, 1981). However, because behavioral thresholds are not available for newborns, normative data are restricted to infants from well-baby nurseries who are assumed to have low risk for hearing

TABLE 10.3

Contralateral ASR Threshold 90th Percentiles for 500-, 1,000-, and 2,000-Hz Activators as a Function of Behavioral Threshold for the Corresponding Puretone Test Frequencies

Behavioral Threshold (dB HL)	ASR Threshold 90th Percentiles (dB HL)[a]					
	Silman and Gelfand (1981)			Gelfand et al. (1990)		
	500 Hz	1,000 Hz	2,000 Hz	500 Hz	1,000 Hz	2,000 Hz
0	95	100	95	95	95	95
5	95	100	95	95	95	95
10	95	100	100	95	95	95
15	95	100	100	95	95	95
20	95	100	100	95	95	95
25	95	100	100	95	95	95
30	100	100	105	95	95	100
35	100	100	105	95	95	100
40	100	105	105	95	95	100
45	100	105	105	95	95	105
50	105	105	110	100	100	105
55	105	105	110	105	105	110
60	105	110	115	105	110	115
65	105	110	115	110	110	115
70	115	115	125	115	115	120
75	115	115	125	120	120	125
80	125	125	125	120	125	>125
85	125	125	125	>125	>125	>125
≥90	125	125	125	>125	>125	>125

[a]Absent reflexes were excluded from the Silman and Gelfand (1981) data, and were included in the Gelfand et al. (1990) data.

loss or infants who have passed a newborn hearing screen. Kankkunen and Liden (1984) used a 660-Hz probe tone and ipsilateral 500-, 1,000-, 2,000-, 4,000-Hz and BBN activators in neonates and in preschool children with normal hearing and sensory/neural hearing loss. In neonates, the mean BBN ASR threshold was 57 dB HL and the mean ASR threshold across tonal activators was 85 dB HL. For separating ears with normal hearing from ears with hearing loss, they reported sensitivity for tonal activators of 58% and specificity of 89% (66% and 86%, respectively, for BBN activators). The authors cautioned that children with hearing loss may be missed when using ASR thresholds to predict hearing loss. Mazlan et al. (2009) reported mean ASR thresholds of 76 and 65 dB HL for ipsilateral activators of 2,000 Hz and BBN, respectively, using a 1,000-Hz probe tone in infants who passed an auditory brainstem response (ABR) and OAE screen. Kei (2012) used a 1,000-Hz probe tone and ipsilateral activators of 500, 2,000, and 4,000 Hz and BBN and reported 95th percentiles of 95, 85, 80, and 75 dB HL, respectively. Both Mazlan et al. and Kei reported that ASR thresholds were reliable across repeated tests.

In summary, the ASR can be elicited in infants with thresholds that are comparable to adults if a higher probe tone frequency is used. By 6 months of age, a 226-Hz probe tone can be used for tympanometry and ASR measurement. For neonates, normative data are available for 660-Hz probe tone and ipsilateral tonal and BBN activators (Kankkunen and Liden, 1984) and 1,000-Hz probe tone with ipsilateral tonal and BBN activators (Kei, 2012; Mazlan et al., 2009). Normative ASR data are not available for infants between the neonatal period to 6 months of age when a 226-Hz probe tone can be used, and normative ASR data are not available for neonates and young children for ASR threshold as a function of hearing loss.

Pharmacological Effects

Elevated ASR thresholds have been reported with alcohol (Borg and Moller, 1967), barbiturates and chlorpromazine, an antipsychotic drug (Simon and Pirsig, 1973). It is not in the audiologist's scope of practice to determine whether pharmacological effects are associated with elevated or absent ASR in a given patient, but it would be helpful to document the patient's current medications for consideration in further testing (e.g., ABR) or in a report to the otolaryngologist if the patient is referred.

ASR DECAY

The ASR decay test can be completed in cases in which retrocochlear involvement is suspected. Indicators of retrocochlear

involvement include asymmetrical puretone behavioral thresholds, asymmetrical word recognition scores or scores that are poorer than expected given puretone thresholds, unilateral tinnitus, report of dizziness, and absent or elevated ASR (in which case the decay test cannot be completed). An abnormal amount of decay, which means that the reflex cannot be sustained, is often associated with retrocochlear site of lesion.

The 226-Hz probe tone is used with a 500- or 1,000-Hz activator for the standard ASR decay test. Higher activator frequencies are not used because decay can be observed even in normal ears. The ipsilateral or contralateral activator is presented for 10 seconds at 10 dB above the ASR threshold for the activator. The activator should elicit a reflex and a decrease in admittance should be observed that is essentially constant for the entire 10 seconds. In ears with VIIIth nerve disorders, "abnormal" or "positive" decay is observed, meaning the amplitude of the reflex decreases by half its initial magnitude in less than 10 seconds (Jerger et al., 1974b). Figure 10.6 shows

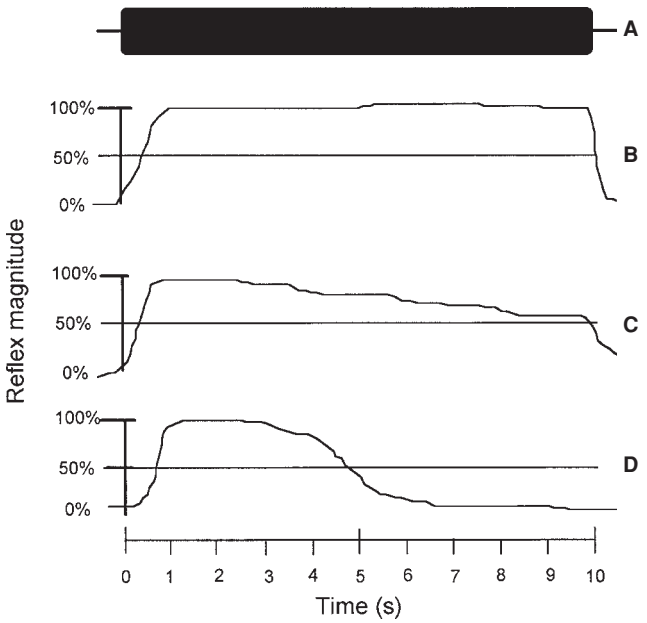


FIGURE 10.6 Examples of normal and abnormal acoustic stapedius reflex (ASR) decay results. Percent of ASR magnitude at stimulus onset is plotted as a function of time in seconds. The stimulus (**A**) is presented for 10 seconds and in a normal response (**B**), the ASR activates and maintains nearly 100% of the initial magnitude for the entire 10 seconds. The magnitude of the response in example (**C**) decreases from the initial magnitude by less than 50% and it is also considered normal. Positive ASR decay, or abnormal ASR decay, is shown in (**D**) in which the response magnitude decreases by more than 50% of the initial magnitude. (From Gelfand SA. (2009) Chapter 10: The acoustic reflex. In: Katz J, Medwetsky L, Burkard R, Hood L, eds. *Handbook of Clinical Audiology*. 6th ed. Philadelphia: Lippincott Williams & Wilkins.)

an example of normal responses (negative decay) in rows b and c, and positive decay of more than 50% in 10 seconds in row d. The reflex decay (RD) is interpreted with reference to the activator ear. If decay is measured in the left contralateral condition, with the activator presented to the left ear and the reflex measured in the probe/right ear, and decay is positive, it means the test is positive for possible retrocochlear pathology in the left ear.

The ABR is used more often today to identify patients with possible retrocochlear involvement. By comparison, the acoustic RD test is more convenient because it can be done with the diagnostic immittance equipment, it is faster, and it requires fewer supplies relative to the ABR which requires another piece of equipment, takes about 30 to 45 minutes, and requires skin preparation pads, electrodes, and electrode wires. However, the RD test has three disadvantages with regard to identification of retrocochlear involvement. First, the test often cannot be completed because ASR is absent or elevated to the point that 10 dB sensation level (SL) re: ASR threshold cannot be presented for the decay test. Second, it has poorer sensitivity/specificity than the ABR for retrocochlear disorders. Finally, temporary and permanent threshold shifts have been reported with high-level activators (Arriaga and Luxford, 1993; Hunter et al., 1999).

USE OF ELECTRICALLY EVOKED ASR IN PATIENTS WITH COCHLEAR IMPLANTS

The ASR can be used in patients with cochlear implants to assess device function and to predict lower or upper stimulus levels of the electrical dynamic range of the electrodes. The stimuli are presented directly to the internal electrodes in the cochlea. The external ear canal, middle ear, and hair cells of the inner ear are bypassed because the VIIIth nerve is directly stimulated. The electrical ASR is recorded using standard clinical immittance equipment and the test can be done intra-operatively during implant surgery or in the audiologist's office during mapping visits. During mapping visits, the ASR can be used to confirm function of the implant in very young children who cannot provide reliable behavioral responses. ASR thresholds have been shown to occur at 70% to 80% of a listener's electrical dynamic range for a given electrode (e.g., Battmer et al., 1990). Levels for each electrode (e.g., "M" levels or most comfortable levels) may be set at some value relative to ASR threshold in children, for example, who can provide limited (or no) information about loudness comfort (e.g., Caner et al., 2007; Hodges et al., 1999), although caution must be taken due to the large subject variability observed within and across studies. ASR may not be recorded in all ears, including ears with ossification of the cochlea related to meningitis (for a review, see Hodges et al., 2003, p. 83). The electrically

evoked ASR is also susceptible to, or may be affected by, middle ear status of the probe ear including otitis media, ventilation tubes, excessive peak static acoustic admittance, and otosclerosis.

ACOUSTIC REFLEXES AND DISORDERS

Acoustic Reflex Thresholds

Various otologic disorders will result in ASR thresholds that are outside the normal range or may be present when expected to be absent. All of these objective outcomes can contribute substantially to the audiologic diagnosis. In this section we will review the expected patterns of ASR threshold responses for a range of pathologies and audiometric patterns.

We will assume that the probe stimulus used to monitor changes in middle ear function induced by the ASR is a 226-Hz tone as stipulated in the ANSI S3.39 standard. For a specific set of measurements, the ear with the 226-Hz probe will be referred to as the probe ear. A basic framework for evaluating ASR threshold responses in the presence of various disorders is to consider the pattern of responses for ipsilateral and contralateral reflex activator stimulation. By convention as described in the ANSI standard, ASR thresholds are named by reporting the ear to which the reflex activator stimulus was presented (right or left) combined with a reference to the probe configuration: Ipsilateral for an activator stimulus presented to the probe ear and contralateral for an activator presented to the opposite ear. For a probe-right condition when a 1,000-Hz tone was presented to the left ear and varied in level to obtain the ASR threshold, we would refer to the reflex threshold as a Left (activator ear) Contralateral (reference to the probe being in the opposite ear) reflex threshold. If we leave the probe in the right ear, but now present the 1,000-Hz activator tone to the right ear, we would call such an ASR threshold a Right Ipsilateral reflex threshold.

The patterns of ipsilateral and contralateral ASR thresholds can be conveniently displayed by using a chart as in Figure 10.7. The chart is organized by Probe ear as if you are facing the patient with the results for the probe in Right ear in the left column and the results for the probe in the Left ear in the right column. The top row of results is for ipsilateral reflex thresholds, thus with the Probe in the Right ear the top left circle indicates results for the R(ight) Ipsilateral reflex threshold. The bottom row is for Contralateral reflex thresholds, so with the probe in the Right ear and the activator in the Left ear we have a L(eft) Contralateral reflex threshold which is indicated by the circle on the bottom left, and so on. The legend at the bottom of the graph shows the symbols for a reflex threshold within normal limits (open circle), elevated (shaded circle) and absent (black circle).

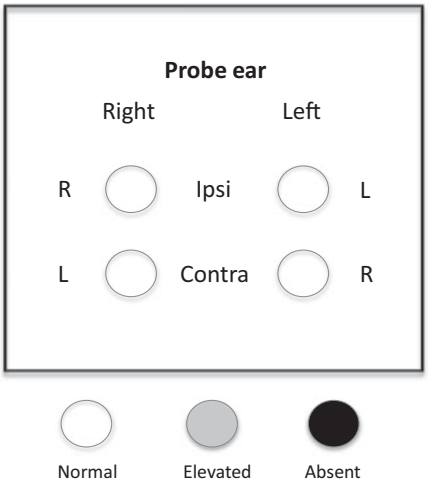


FIGURE 10.7 The basic configuration for reporting ipsilateral and contralateral reflex threshold results for a given activator stimulus. The chart is organized by Probe ear as if you are facing the patient with the results for the probe in Right ear in the left column and the results for the probe in the Left ear in the right column. The *first row* of results is for ipsilateral reflex thresholds, so with the Probe in the Right ear the *top left circle* indicates results for the R(ight) Ipsilateral reflex threshold. The *bottom row* is for Contralateral reflex thresholds, so with the probe in the Right ear and the activator in the Left ear we have a L(eft) Contralateral reflex threshold which is indicated by the *circle* on the *bottom left*, and so on. The legend at the bottom of the graph shows the symbols for a reflex threshold within normal limits (*open circle*), elevated (*shaded circle*) and absent (*black circle*).

Hearing Loss of Cochlear Origin

As discussed previously, based on the normative data in Table 10.3 for listeners with normal hearing, the 90th percentile of ASR thresholds is 95- to 100 dB HL. Sensory/neural hearing loss of cochlear origin results in median ASR thresholds in the normal range (i.e. ≤95 dB HL for 500-, 1,000-, and 2,000-Hz tones) for levels of hearing loss approximately ≤60 dB HL (Gelfand et al., 1990). Although some individuals (10th percentile, Figure 10.5) will have ASR thresholds in the normal range with even a severe hearing loss (≥70 dB HL), the trend is for puretone thresholds above 60 dB HL to result in median reflex thresholds >95 dB HL. The 90th percentile of ASR thresholds increases as a function of cochlear hearing loss from 95 to 105 dB HL for 30 dB HL puretone thresholds to 105 to 115 dB HL for 60 dB HL puretone thresholds (Table 10.3). Note that to prevent high levels of sound exposure many centers will restrict the upper limit of reflex activator stimulus to 105 dB HL, and thus report an absent reflex if the ASR is not observed at 105 dB HL. As can be seen from Table 10.3 many listeners with cochlear hearing loss greater than 60 dB and at the 90th percentile of ASR thresholds will have absent reflex thresholds at 105 dB HL.

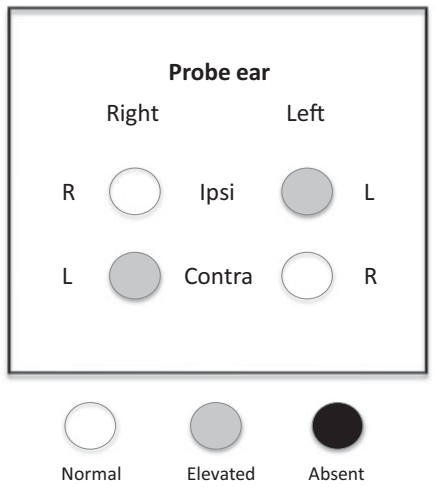
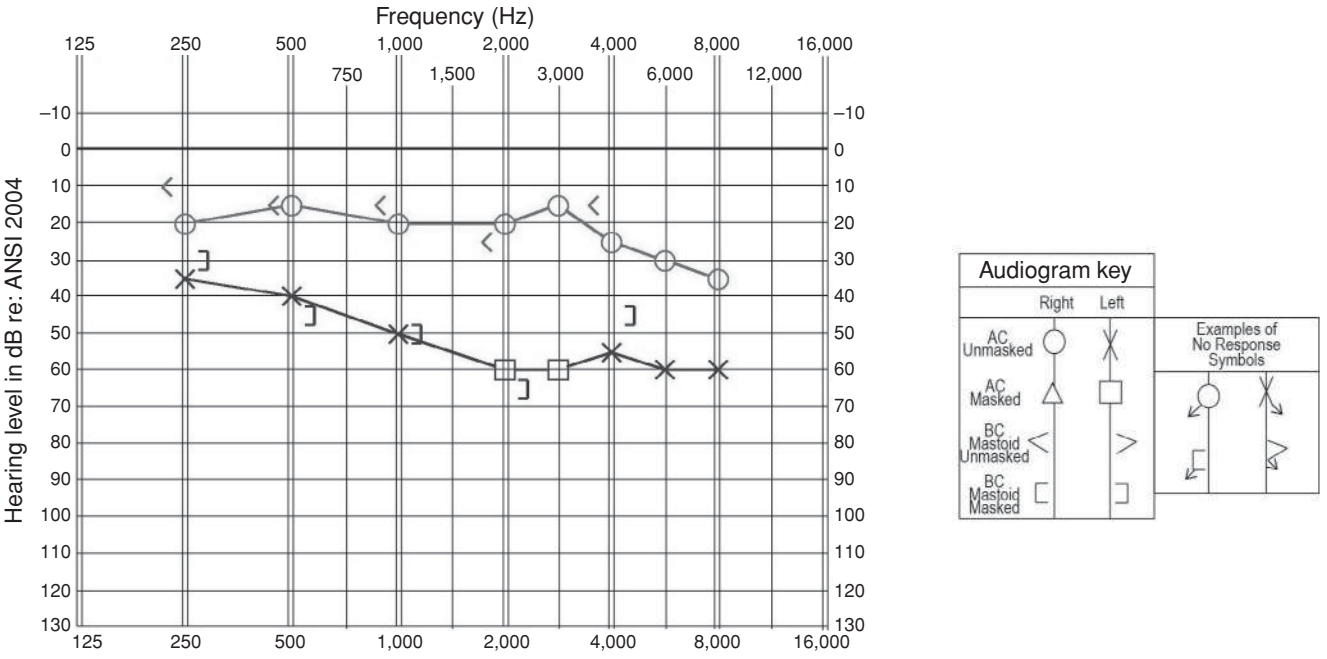


FIGURE 10.8 Shown is a pattern of elevated reflex thresholds with activator in the Left ear (*shaded circles*) due to a sensory/neural hearing loss of 50 dB HL at 1,000 Hz. The reflex thresholds for stimulation of the normal right ear are within normal limits (*open circles*). See audiogram in Figure 10.9.

Figure 10.8 shows the expected ASR threshold results for the case of normal hearing in the right ear and a cochlear hearing loss in the left ear of 50 dB HL at 1,000 Hz (see audiogram in Figure 10.9). The top row shows that the L(eft) Ipsilateral reflex threshold is elevated and the bottom row shows that the L(eft) Contralateral reflex threshold (probe in the right ear and activator in the left ear) is elevated. This diagonal pattern of elevated reflexes was referred to as a "sound effect" by Hannley (1986) because reflexes are abnormal whenever the *sound* (activator) is presented to the affected ear.

ASR thresholds would be expected to be absent at the upper limit of the activator when stimulating an ear with a profound cochlear hearing loss (≥90 dB HL). Figure 10.10 shows the expected ASR threshold results at 1,000 Hz for a right profound sensory/neural hearing loss and a left moderately-severe sensory/neural hearing loss of 65 dB HL (see audiogram in Figure 11.11). Figure 10.10 shows two diagonal patterns of reflex threshold results: elevated thresholds for ipsilateral and contralateral stimulation of the left ear at 1,000 Hz, and absent thresholds for ipsilateral and contralateral stimulation of the right ear. The abnormal pattern



Acoustic reflex thresholds

		Stim in	Meas in	500	1K	2K	4K	Decay 500	Decay 1K
CONTRA	( )	RT	LT	90	90	95			
		LT	RT	100	100	105			
IPSI	( )	RT	RT	85	90	95			
		LT	LT	100	100	105			

Tympanometry

Canal volume (mL)	RT	LT
	1.5	1.6

Static admittance (mL): Rt 0.8 Lt 0.8

Peak pressure (daPa): Rt 10 Lt –10

Type: Rt A Lt A

FIGURE 10.9 Audiometric results for patient whose reflex results at 1,000 Hz are described in Figure 10.8.

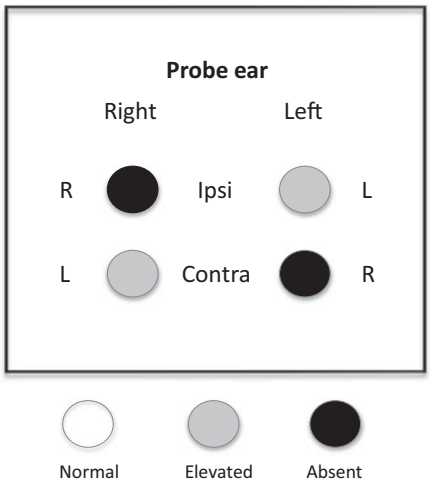


FIGURE 10.10 Results for an asymmetrical hearing loss of cochlear origin. The puretone threshold shows a moderately severe loss of 65 dB HL on the left at 1,000 Hz, and a profound loss of 105 dB HL of cochlear origin on the right. The pattern of elevated reflex thresholds with activator in the Left ear (*shaded circles*) is combined with the pattern of absent reflexes at the upper limits of stimulation due to the profound cochlear hearing loss at 1,000 Hz on the right (*black circles*). See audiogram in Figure 10.11.

of elevated/absent reflexes are related to the ear receiving the activator sound. Thus, this can be thought of as a sound effect for both ears due to the cochlear hearing loss.

Facial Nerve Paralysis

When measuring ASR thresholds, the opposite of a sound effect is a probe effect. This occurs when the ASR is abnormal for an ear whenever the probe is in that ear. This is due to a failure to properly *measure* the acoustic reflex threshold in an ear. The classic case of a probe effect is cranial nerve VII paralysis such as Bell palsy (idiopathic facial nerve palsy). If the site of the inflammation of the VIIth nerve that is causing the paralysis is proximal to (closer to the brain than) the innervation of the stapedius muscle, the muscle will likely not contract due to the paralysis and thus a reflex cannot be *measured,* even though the activator stimulus may be sufficiently intense to activate the reflex arc. If the site of the inflammation of the VIIth nerve is distal to (further from the brain than) the innervation of the stapedius muscle, the stapedius muscle will likely not be affected, even in the presence of facial paralysis (Alford et al., 1973). This ASR result provides diagnostic information about the site of the lesion causing the paralysis. Bell palsy may also result in abnormal reflex adaptation in the affected ear (Silman

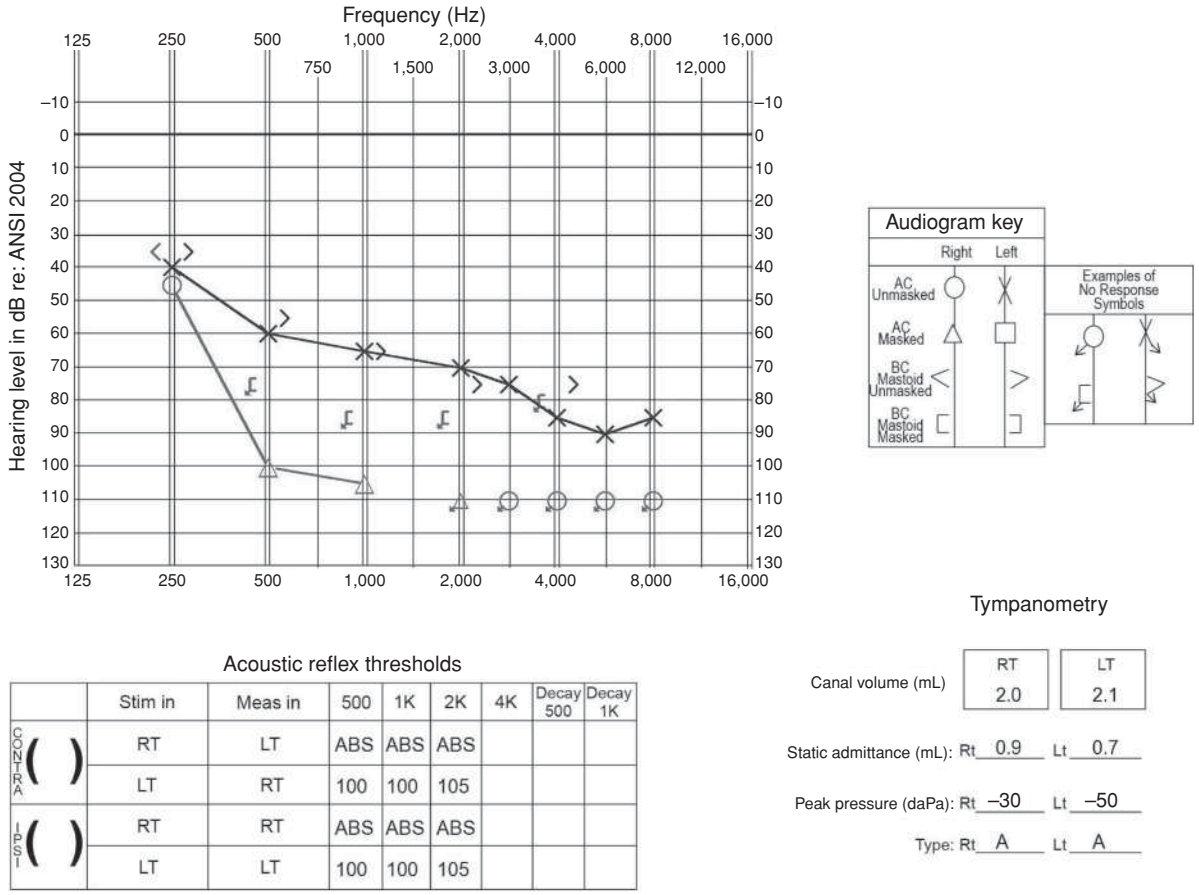


FIGURE 10.11 Audiometric results for patient whose ASR threshold results at 1000 Hz are described in Figure 10.10.

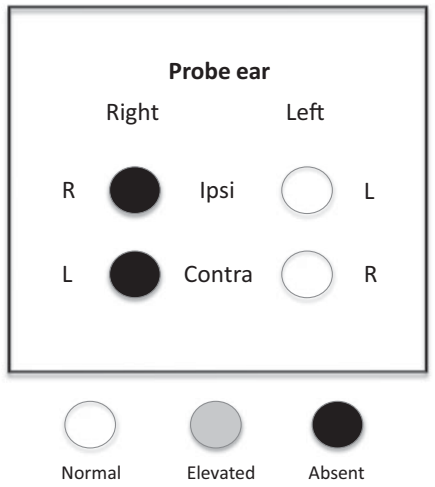


FIGURE 10.12 ASR results are for a patient with normal hearing bilaterally with a right Bell palsy. The ASR threshold results at 1,000 Hz are absent in both conditions where the probe is in the Right ear, and present in both cases where the probe is in the Left ear. The reflex threshold is absent with the probe in the right ear because the reflex cannot be measured due to the dysfunctional stapedius muscle.

et al., 1988). The ASR can be used to monitor the eventual return of function in the case where the site of lesion is proximal to the innervation of the stapedius and the ASR is initially absent. However, assessment for the determination of the need for surgical intervention may require the use of electroneurography and electromyography (Gantz et al., 1999).

Figure 10.12 shows the results of ASR threshold testing at 1,000 Hz for a case of right Bell palsy with an absent reflex with the probe in the right ear, for both the right ipsilateral reflex test and left contralateral reflex test. This patient's audiometric data are shown in Figure 10.13 and are within normal limits except for the reflex pattern. Hannley (1986) referred to this probe-effect pattern as a vertical pattern because the absent reflexes were in one vertical column corresponding to the probe ear with the abnormality.

Conductive Hearing Loss

When a measurement probe is inserted in an ear with a conductive hearing loss (CHL) or mixed hearing loss, even a slight conductive component may result in an absent reflex threshold. Jerger et al. (1974a) reported that with an

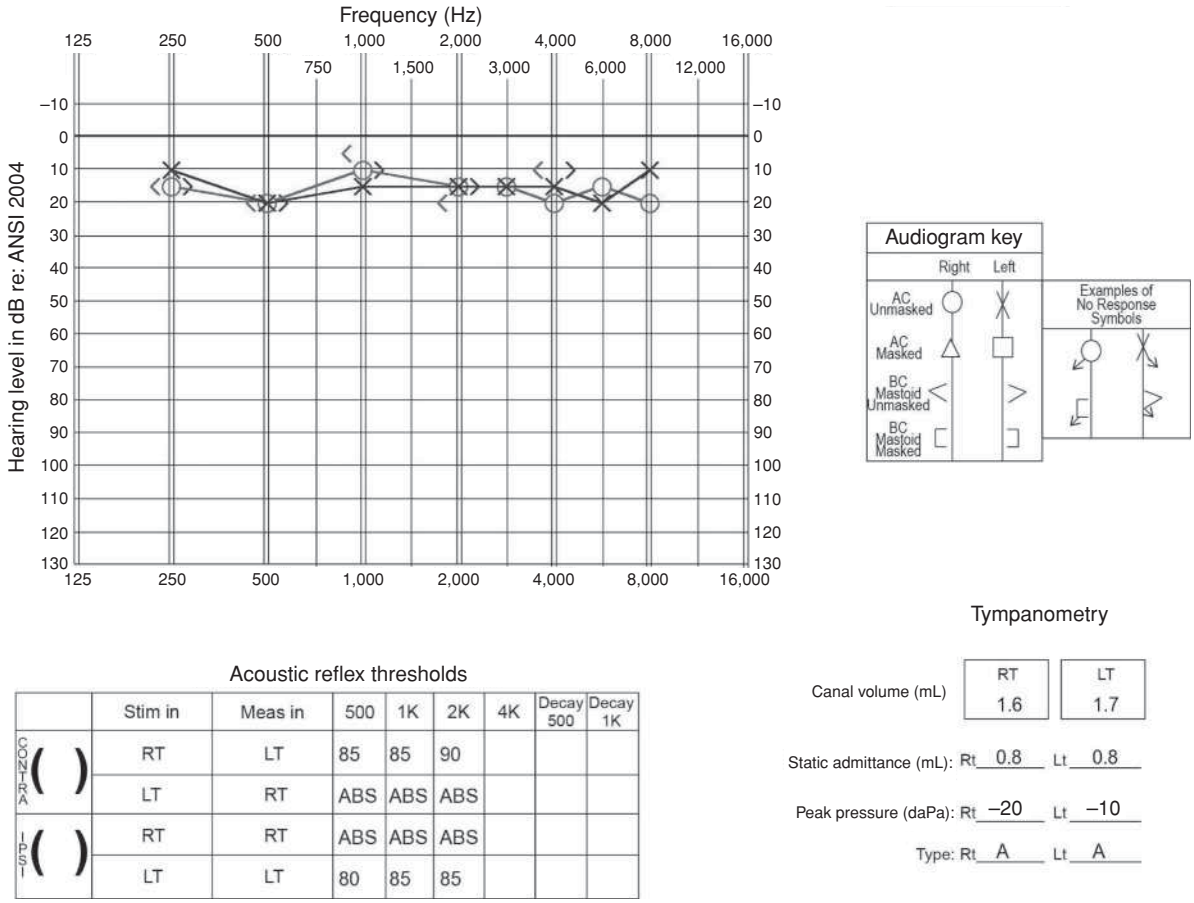


FIGURE 10.13 Audiometric results for the patient whose ASR threshold results at 1,000 Hz are shown in Figure 10.12.

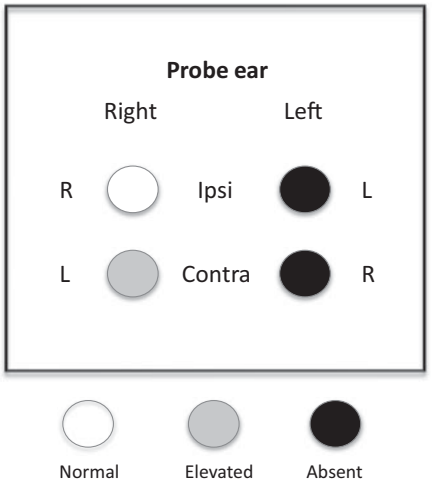


FIGURE 10.14 ASR results for a 500-Hz activator frequency for a patient with normal cochlear function but with a left serous otitis media resulting in a 20-dB air–bone gap at 500 Hz. Reflexes are absent on the left when it functions as a probe ear since the small change in admittance caused by the reflex is masked by the stiffness and mass changes induced by the serous fluid in the middle ear. However, when stimulating the left ear for the contralateral reflex threshold, a reflex is obtained at an elevated level of 105 dB HL. See audiogram in Figure 10-15.

average air–bone gap of only 5 dB in the probe ear, 50% of subjects had an absent reflex threshold. Thus, similar to the case of facial nerve paralysis, a CHL results in a probe effect, an inability to measure a reflex with the probe in an ear with CHL. The small change in admittance induced by the reflex is masked by the conductive component. In addition to the probe effect, a sound effect may exist when trying to stimulate an ear with a conductive component to achieve a contralateral reflex threshold since the CHL attenuates the level of the activator by the degree of CHL. This may result in normal, elevated or absent contralateral reflex threshold depending on the size of the air-bone gap. For example, Jerger et al. (1974a) reported that when an ear with a CHL was the activator ear for the contralateral reflex threshold, the reflex threshold was absent in 50% of cases with average air-bone gaps ≥27 dB HL. Figure 10.14 shows the results of a reflex test at 500 Hz for a patient with left serous otitis media and a 20 dB air–bone gap at that frequency (audiogram in Figure 10.15). The reflex is absent with the probe in the left ear due to the conductive component (probe effect). However, the left contralateral reflex threshold was elevated due to the CHL, but was not eliminated (sound effect). The dB sensation level of the activator was sufficiently high to induce an ASR at a high presentation level. A greater degree of conductive component on the left would have resulted in an absent left contralateral reflex. Bilateral CHL of a significant degree may result in a bilateral probe effect, and thus absent reflexes bilaterally (Figure 10.16, audiogram in Figure 10.17).

A TM perforation or pressure equalization tube in the TM may result in only a slight change in puretone threshold in the low frequencies when measured with standard headphones (but see Voss et al., 2000 for inaccurate results obtained when an insert phone is used in an ear with a perforation). However, this condition when using a 226-Hz probe tone, results in the measurement of a cavity volume including the ear canal and middle ear rather than admittance at the TM. Because the stapedius muscle contraction does not appreciably alter the cavity volume being measured, a probe effect exists for the involved ear. This would result in a reflex pattern like that shown in Figure 10.12 for Bell palsy with absent reflexes in the probe ear. Mixed hearing loss with a significant conductive component results in a probe effect in the involved ear, and may result in an elevated or absent contralateral reflex if that ear is the activator ear. For greater degrees of sensory/neural loss the contralateral reflex threshold for the activator ear would be elevated due to the cochlear component plus the conductive component and would, therefore, likely be absent.

Central Nervous System Disorders

Retrocochlear and Brainstem Disorders

Vestibular Schwannoma

A vestibular schwannoma (acoustic neuroma) is a slow growing benign tumor that most commonly develops in the Schwann cells on the vestibular branch of CN VIII and causes destruction of neural tissue as it grows. The growth pattern of these tumors is not well understood, but the mean growth rate is between 1 and 4 mm per year with up to 75% of these tumors showing no appreciable growth rate, but with some exceptional growth rates over 18 mm per year (Nikolopoulos et al., 2010). Magnetic resonance imaging (MRI) is the definitive diagnostic tool for detection of vestibular schwannomas. These tests are typically ordered based on the presence of an asymmetrical sensory/neural hearing loss. Such MRI scans are typically positive in about 1% to 3% of cases (Newton et al., 2010). ABR testing has been reported to be as effective as MRI for larger tumors (>1 cm) but is not as effective as MRI for smaller tumors (Fortnum et al., 2009).

ASR thresholds in ears with vestibular schwannomas are likely to be elevated or absent. Thus, suspicion for a retrocochlear lesion is raised when the reflex threshold exceeds the normal range for the degree of hearing or is absent. The 90th percentile of ASR thresholds for a given hearing threshold for normal hearing or cochlear hearing loss (Table 10.3) may be used to determine if the reflex threshold is elevated. Even with puretone thresholds in the 0- to 10-dB HL range, as high as 50% of subjects with retrocochlear lesions may have been reported to have absent reflexes (Jerger et al., 1974b); however, these data were likely based on a larger average tumor size than that observed in patients seen currently given the improvements in tumor detection

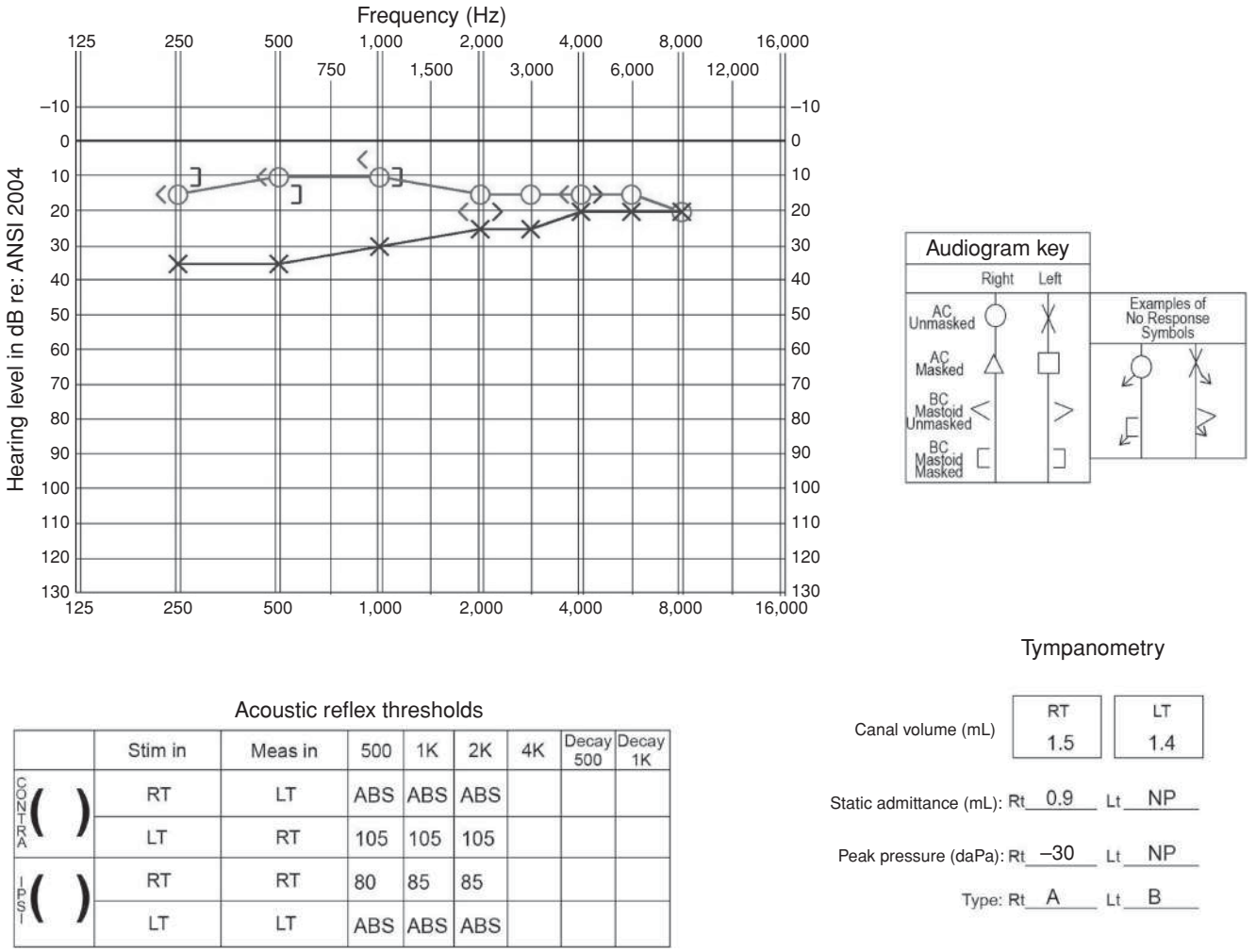


FIGURE 10.15 Audiometric results for the patient with ASR threshold results at 500 Hz are shown in Figure 10.14.

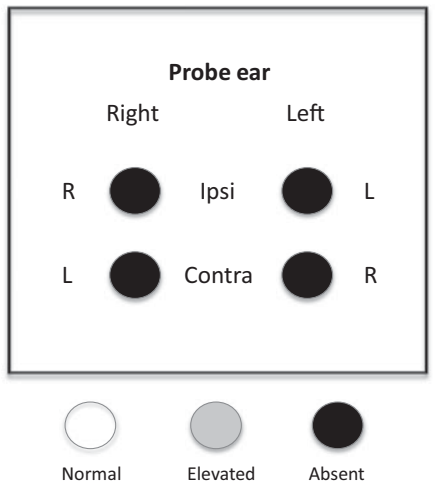


FIGURE 10.16 ASR results for a 500-Hz activator frequency for a patient with bilateral serous otitis media resulting in a bilateral conductive hearing loss at 500 Hz with an air–bone gap of 30 dB. Reflexes are absent in both ears as the result of a bilateral probe effect.

with MRI. Thus, although ASR thresholds may not be a sensitive test for detecting small vestibular schwannomas, positive findings of absent or elevated ASR thresholds, especially in patients with asymmetrical hearing loss, raise the index of suspicion for retrocochlear pathology.

The results for a prototypical retrocochlear finding of ASR thresholds for a case of left vestibular schwannoma are shown in Figure 10.18 for a 1,000-Hz activator tone (audiogram in Figure 10.19). The ASR thresholds are within normal limits when the activator is presented to the right ear for ipsilateral and contralateral stimulation. However, with only a 10 dB increase in threshold at 1,000 Hz on the left compared to the right, the ASR is absent for both left ipsilateral and contralateral stimulation. Hunter et al. (1999) conducted a retrospective analysis of 56 cases of acoustic neuroma compared to 108 adults with hearing loss of cochlear origin. They examined the sensitivity and specificity of the presence or absence of a 1,000-Hz ipsilateral reflex threshold for subjects in these groups whose 1,000-Hz puretone threshold was ≤70 dB HL, ≤50 dB HL or no exclusion for threshold. The

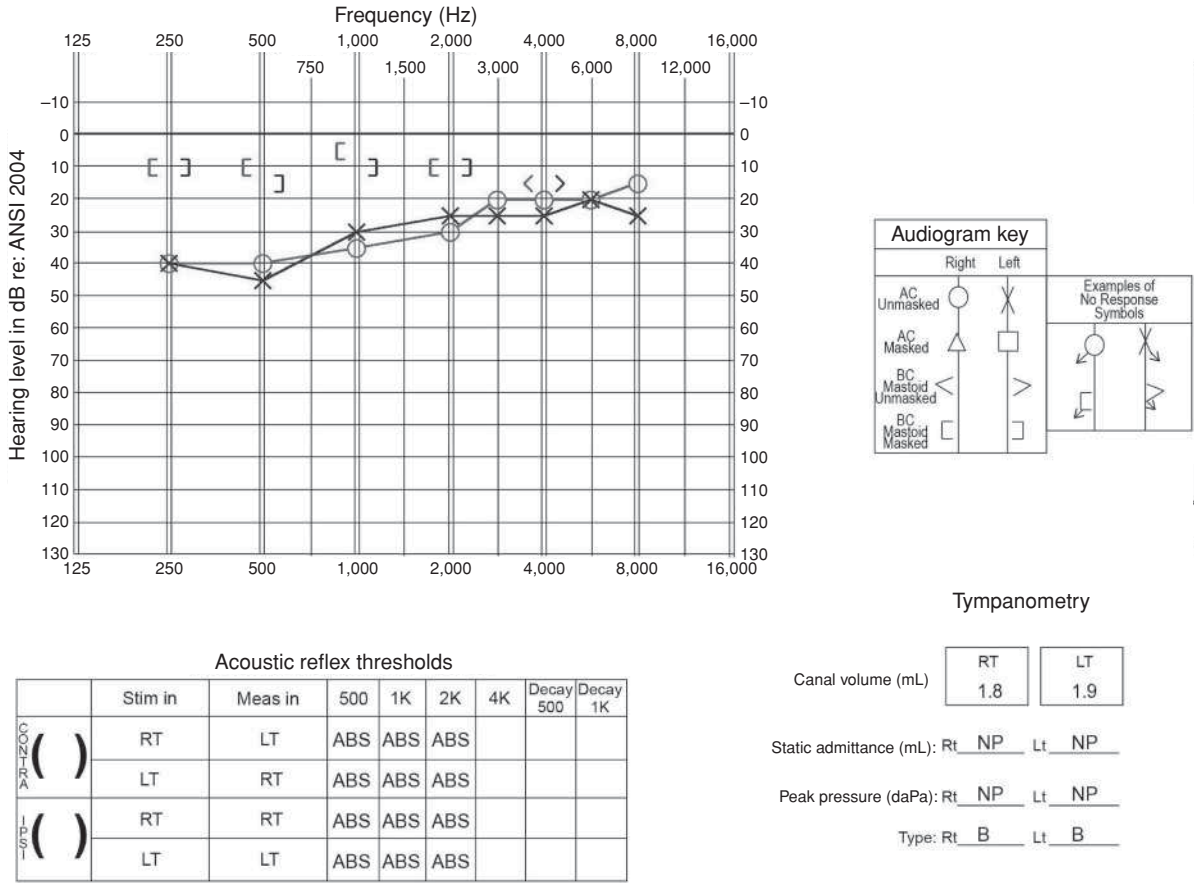


FIGURE 10.17 Audiometric results for the patient with ASR results at 500 Hz in Figure 10.16.

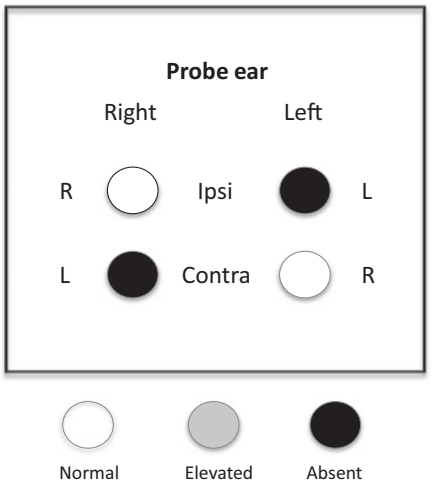
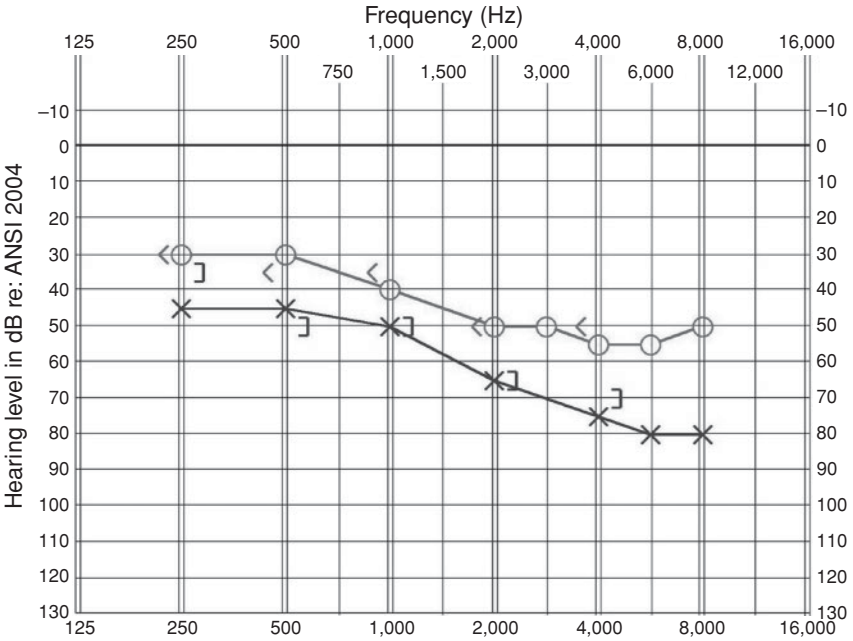


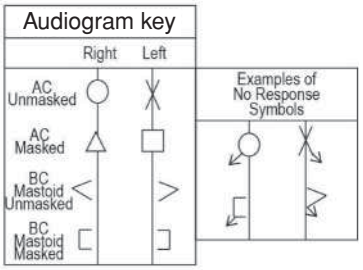
FIGURE 10.18 Results for an asymmetrical sensory/neural hearing loss in a patient with a left vestibular schwannoma. The puretone thresholds show a mild to moderate sensory/neural hearing loss on the right with a threshold of 40 dB HL at 1,000 Hz. There is a moderate to severe sloping sensory/neural hearing loss on the left with a threshold of 50 dB HL at 1,000 Hz. The ASR is absent with stimulation of the left ear at 1,000 Hz for both ipsilateral and contralateral stimulation, which is not expected with a 50-dB HL threshold.

best test performance was found for reflex threshold with a cutoff criterion of >90 dB HL with a true positive rate of 68% and false-positive rate of 46%. Puretone asymmetry at 1,000 Hz provided a better test when considering all hearing losses with >10 dB puretone asymmetry yielding a true positive rate of 93% and a false-positive rate of 32%. This suggests that ASR thresholds alone may not be as useful as puretone threshold asymmetry for suspicion of vestibular schwannoma.

Auditory Neuropathy Spectrum Disorder

Auditory neuropathy/auditory dyssynchrony or auditory neuropathy spectrum disorder (ANSD) is a form of hearing impairment in which neural transmission in the peripheral auditory system is impaired and for which there is often normal cochlear outer hair cell function and present otoacoustic emissions (Starr et al., 2000). Berlin et al. (2005) reviewed the records of 136 patients from a database of 257 subjects with ANSD who had normal otoacoustic emissions and were tested with acoustic reflexes. None of these subjects showed normal reflexes at all frequencies tested and only three subjects had reflexes at 95 dB HL or lower, but not at all frequencies. All of the other subjects had reflex thresholds that were absent or were observed at levels above 100 dB HL. The authors urged that, for perinatal hearing





Acoustic reflex thresholds

		Stim in	Meas in	500	1K	2K	4K	Decay 500	Decay 1K
CONTRA	( )	RT	LT	90	95	100			
		LT	RT	ABS	ABS	ABS			
IPSI	( )	RT	RT	95	95	100			
		LT	LT	ABS	ABS	ABS			

Tympanometry

	RT	LT
Canal volume (mL)	1.6	1.5
Static admittance (mL): Rt	0.9	Lt 0.7
Peak pressure (daPa): Rt	–20	Lt –30
Type: Rt	A	Lt A

FIGURE 10.19 Audiometric results for the patient with ASR threshold results at 1,000 Hz in Figure 10.18.

screening programs based solely on otoacoustic emissions, ASR thresholds should be tested to help rule out ANSD.

Extra-axial Brainstem Disorders

Tumors such as meningiomas, arising from the lining of the brain, that are located in the cerebellar pontine (CP) angle of the brainstem may exert pressure on CN VIII fibers. This type of extra-axial (outside the brainstem itself) lesion would result in a sound effect similar to an acoustic neuroma (Figure 10.18) (Jerger & Jerger, 1975). However, a meningioma may exist in the CP angle and not cause auditory symptoms if the CN VIII fibers are not compromised.

Intra-axial Brainstem Disorders

Lesions occurring within the brainstem (intra-axial) may affect the ASR thresholds if the reflex pathways are compromised (Gelfand, 1984; Jerger & Jerger, 1977). If the crossed fibers are compromised, a pattern as shown in Figure 10.20 (audiogram in Figure 10.21) may exist, which Hannley (1986) referred to as the "horizontal pattern" in that the contralateral reflexes were affected, but the ipsilateral reflexes were normal, thus absent reflexes occurred in a horizontal

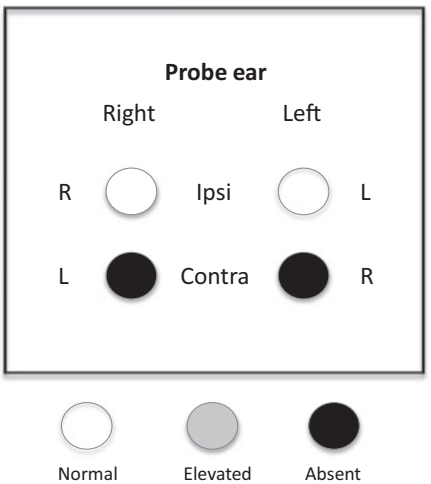


FIGURE 10.20 ASR results for a 1,000-Hz activator are shown for a patient with an intra-axial brainstem lesion. This horizontal pattern indicates that the brainstem is compromised at the point of nerve fiber crossover, thus rendering the contralateral reflexes absent, but leaving the ipsilateral reflexes intact.

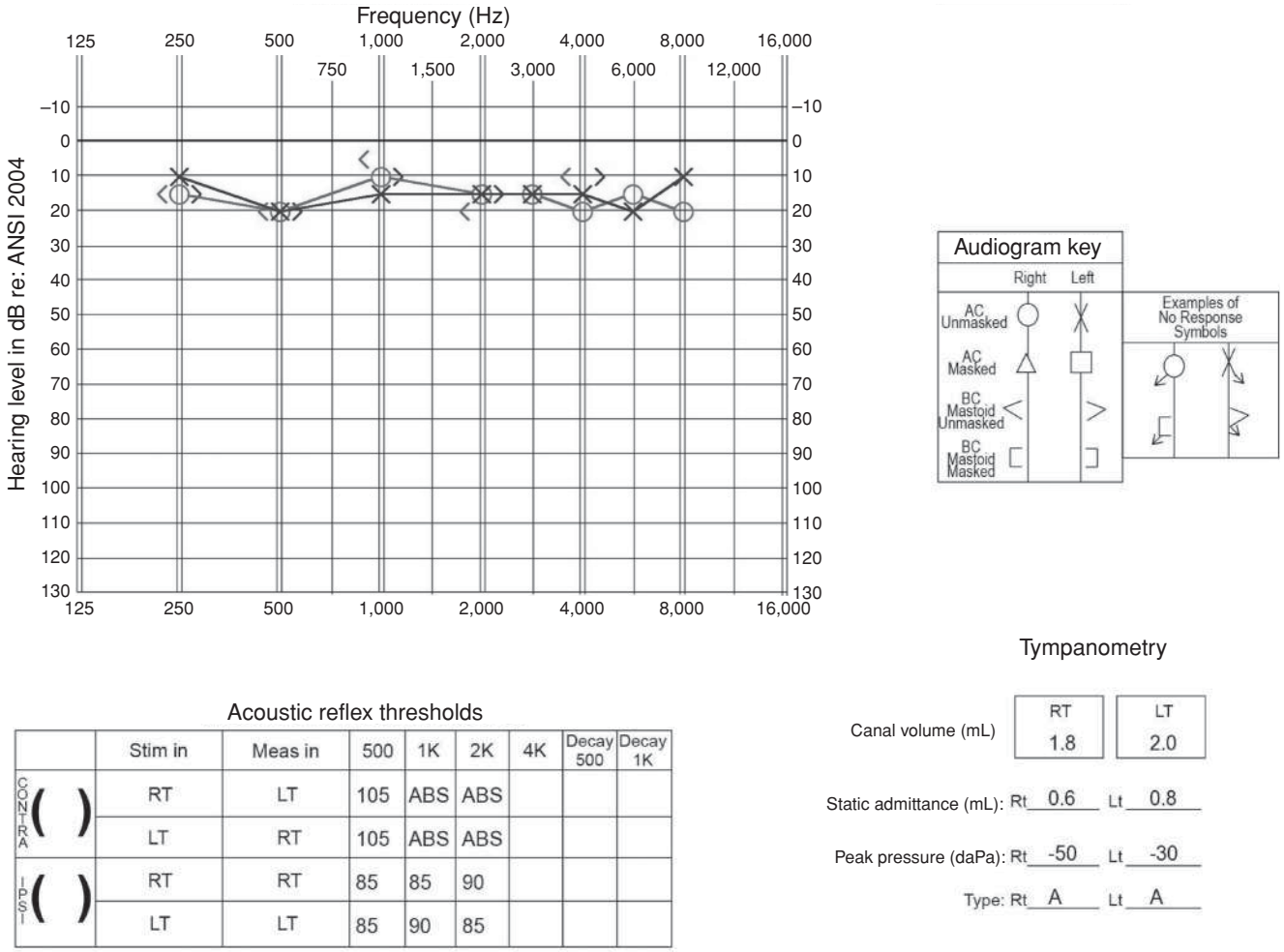


FIGURE 10.21 Audiometric results for the patient with ASR results at 1,000 Hz in Figure 10.20.

pattern. However, Cohen and Prasher (1988) reported that it was not uncommon in intra-axial brainstem lesions to have abnormal reflexes for both ipsilateral and contralateral stimulation. They referred to this as a "full house" pattern, which was observed along with abnormal ABR results bilaterally.

Demyelinating and Cortical Disorders

Reflex thresholds, reflex magnitude and temporal characteristics such as rise time and onset latency may be useful in the assessment of patients with multiple sclerosis (MS), a demyelinating disease (Jerger et al., 1986; Keith et al., 1987). However, Wiegand and Poch (1988) reported that in patients who are asymptomatic for the disease, ASR rise time and onset latency may not be a useful screening tool for MS.

ASR thresholds do not appear to be greatly affected by cortical disorders. For example, Gelfand and Silman (1982) reported on ASR thresholds in 14 patients with documented brain lesions including head trauma, stroke and anoxia who also had auditory processing deficits as confirmed by evaluations from speech language pathologists. All subjects had ASR thresholds consistent with their hearing loss when compared to individuals with normal hearing or sensory/neural loss who did not have cortical disorders. Downs and Crum (1980) reported on several cases of low ASR thresholds in patients with documented brain damage, with a suggestion that a release from central inhibition could have been responsible for the reduced ASR thresholds. However, this finding is not supported by other studies (Gelfand, 1984; Gelfand & Silman, 1982).

Superior Canal Dehiscence

A dehiscence of the superior semicircular canal may result in symptoms of vertigo, oscillopsia, or disequilibrium in response to sound or changes in ear pressure (Minor et al., 1998). Patients with superior canal dehiscence (SCD) may also experience elevated puretone air conduction thresholds and enhanced bone conduction threshold in the involved ear as the dehiscence acts as a third window for the inner ear, which can lower the cochlear input impedance and shunt sound pressure away from the cochlea resulting in an

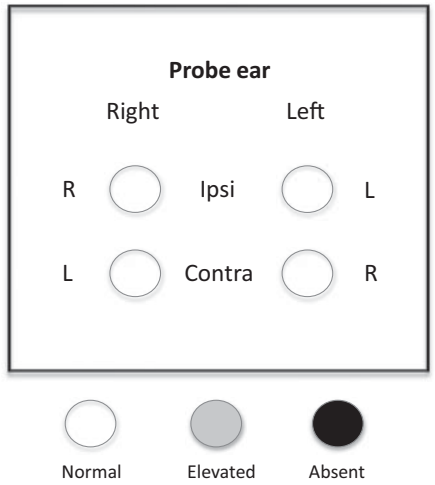
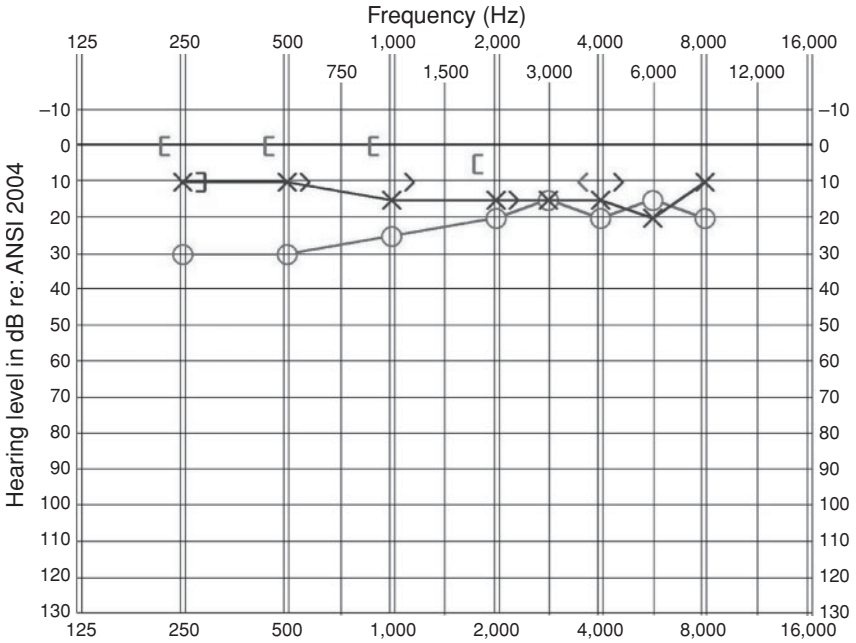
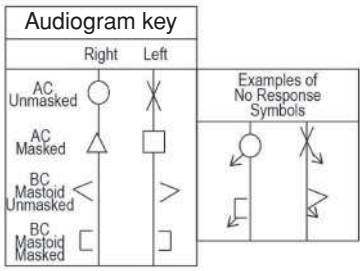


FIGURE 10.22 ASR results at 500 Hz for a subject with a right conductive hearing loss. Given the degree of air–bone gap one would expect absent reflexes with the probe in the right ear. However, this is a case of superior canal dehiscence with a normal middle ear. The presence of the reflexes helps distinguish this case from otosclerosis, which would have a similar hearing loss but absent reflexes.

air conduction hearing loss that appears to be conductive in nature (Rosowski et al., 2004). In some patients the only presenting symptom for SCD may be "conductive" hearing loss in the involved ear with larger air–bone gaps in the low frequencies and no vestibular symptoms, which mimics otosclerosis (Merchant et al., 2007; Mikulec et al., 2004).

The ASR threshold is one test that can help to distinguish SCD from otosclerosis; the presence of acoustic reflexes at normal levels with the probe in the involved ear is an indication that the middle ear is not the source of the "conductive" hearing loss. These patients may also present with low thresholds for vestibular evoked myogenic potentials (VEMP) (Banerjee et al., 2005; Zuniga et al., 2013). Figure 10.22 shows the ASR result at 500 Hz for a patient with a mild CHL in the low frequencies on the right (audiogram in Figure 10.23). Note that ASR thresholds are within normal limits in the presence of the conductive loss for ipsilateral and contralateral stimulation. This paradoxical ASR finding is part of the pattern of findings that aid in the diagnosis of SCD along with low VEMP thresholds and radiological evidence (see also Chapter 20 in this volume on VEMP results in SCD).





Acoustic reflex thresholds

		Stim in	Meas in	500	1K	2K	4K	Decay 500	Decay 1K
CONTRA	( )	RT	LT	85	90	85			
		LT	RT	85	85	90			
IPSI	( )	RT	RT	85	85	85			
		LT	LT	85	90	85			

Tympanometry

	RT	LT
Canal volume (mL)	1.8	1.6

Static admittance (mL): Rt 0.5 Lt 0.4

Peak pressure (daPa): Rt –20 Lt –20

Type: Rt A Lt A

FIGURE 10.23 Audiometric results for the patient whose ASR results at 500 Hz are shown in Figure 10.22.

Functional Hearing Loss

As indicated above, median ASR thresholds are expected to be within normal limits for cochlear hearing loss ≤60 dB HL, so for suspected functional losses in this range ASR thresholds will not provide objective evidence to support the suspicion. In these cases, especially for losses in the upper end of this range, objective evidence provided by otoacoustic emissions may prove to be more useful (see Chapter 19 this volume). Tonal ASR thresholds occurring below the admitted puretone threshold are an obvious red flag for functional hearing loss. However, the question remains, how low is too low if the ASR threshold occurs at a low sensation level above the puretone threshold? Gelfand et al. (1990) suggested using published 10th percentiles of ASR thresholds at 500, 1,000 and 2,000 Hz (lower curves in Figure 10.5) as cutoff values for determining the likelihood of functional hearing loss. ASR thresholds falling below 10th percentile values for a given level of hearing loss are considered suspect for functional hearing loss. Gelfand (1994) found this 10th percentile approach to successfully identify about 85% of 74 ears with functional hearing loss having admitted thresholds ≥60 dB HL at all three frequencies, while maintaining a false-positive rate for 50 ears with true sensory/neural hearing loss between 5% and 7%.

Acoustic Reflex Adaptation

The acoustic reflex adapts when constantly evoked for a period of time. This relaxation of the reflex results in the admittance change induced by the ASR returning to baseline or decaying over time. Adaptation of the reflex occurs in normal hearing subjects depending on the duration of exposure. There is a frequency dependence of this effect that increases in adaptation with frequency, for example, at 4,000-Hz RD for tones presented at 10 dB above reflex threshold decays to 50% of maximum in less than 10 seconds in subjects with normal hearing (Anderson et al., 1969; Wilson et al., 1984). Anderson et al. (1969) proposed using a criterion RD of 50% of original amplitude in 5 seconds at 500 and 1,000 Hz as a criterion for abnormal RD and an indication for retrocochlear site of lesion. Other studies have suggested looking for a decay of 50% within a 10-second window (Jerger et al., 1974b; Olsen et al., 1981). Hirsch and Anderson (1980a, 1980b) recommended looking at adaptation at 500 and 1,000 Hz as a graded finding as follows:

RD^{+++} if the reflex amplitude declines ≥50% in 5 seconds at 500 and 1,000 Hz. This is positive sign of retrocochlear lesion.

RD^{++} if the reflex amplitude declines ≥50% in 5 seconds at 1,000 Hz but not 500 Hz. This is a questionable sign of retrocochlear lesion.

RD^{+} if the reflex amplitude declines <50% within 5 seconds at 500 and 1,000 Hz. This is not a significant retrocochlear sign.

Since the proposed method of determining RD depends on an activator presentation for 5 to 10 seconds at 10 dB above the ASR threshold it calls into question the resultant high level of sound exposure during the test. Hunter et al. (1999) reported on a case of hearing loss that was caused by the RD testing in a patient whose ASR threshold at 1,000 Hz was 110 dB HL. RD testing was conducted at 120 dB HL for 10 seconds in each ear and resulted in permanent threshold shift at 1,000 Hz of 20 to 30 dB. A similar finding was reported by Arriaga and Luxford (1993). Hunter et al. recommend a maximum safe presentation level for RD stimuli of 115 dB SPL, but suggested that manufacturers include an in-the-ear measure of SPL for activator presentation to help account for inter-patient variability in real-ear stimulus presentation levels.

New Methods of Measuring the ASR Threshold

Several recent studies have investigated new methods of measuring the ASR threshold. Neumann et al. (1996) utilized standard otoacoustic emissions recording techniques to measure ASR threshold. In this method the probe stimulus and activator stimulus are one and the same. Two identical 100 ms tone bursts were presented with a 10 ms interstimulus interval with a repetition rate of one per second. Since the latency of the ASR is on the order of 100 ms, it was assumed that the first tone burst was not affected by an ASR that might have been elicited by the tone; however, the second 100 ms tone burst would be affected by stapedius muscle contraction. The difference in microphone response for the two tone bursts averaged across a number of presentations was taken as a measure of the ASR. The level of the tones was varied until a difference between the two tones was reliably detected. This method allowed for an ASR threshold for normal hearing subjects to be detected at a level 8 dB lower than traditional methods. In addition, for 5 out of 10 subjects with sensory/neural hearing loss, the new method was able to detect an ASR when the ASR was absent for traditional methods.

Several studies have used wideband acoustic immittance (WAI) to measure the ASR threshold (see Schairer et al., 2013 for a review). For an introduction to WAI, see Chapter 9 by Hunter and Sanford in this volume. Feeney and Keefe (1999) were the first to report on this method for measuring contralateral reflex thresholds using a WAI system developed by Keefe et al. (1992). In that study 40 ms wideband chirps were presented at ambient pressure as the probe stimulus and 1,000- or 2,000-Hz contralateral tones were used as reflex activator stimuli for three subjects. The tones were varied in level ±8 dB relative to the clinical ASR threshold in 2-dB steps. WAI obtained during the baseline measurement in quiet was subtracted from the WAI obtained during the presentation of the contralateral activator stimulus. The study demonstrated that the ASR could

be measured using this technology and that the ASR threshold appeared to be as much as 8 dB lower using the WAI method. The same method was used by Feeney and Keefe (2001) but with a white noise contralateral activator. Measurements on seven subjects revealed lower ASR thresholds by about 18 dB with the WAI method using statistical tests of the magnitude and shape of the shift across frequency in WAI to detect the ASR threshold.

A statistical method was also used by Feeney et al. (2003) to measure the contralateral reflex threshold for 1,000- and 2,000-Hz tones for 34 adults with normal hearing. The average reflex thresholds measured with this method were 12 and 14 dB lower than for a clinical method for the 1,000- and 2,000-Hz tones, respectively. Feeney et al. (2004) used the method to develop an ipsilateral wideband test using a 4,000-Hz activator and wideband probe. In this study the WAI method resulted in ASR thresholds that were 3 dB lower than with a clinical method.

Schairer et al. (2007) developed an automated system to use WAI to measure ipsilateral ASR thresholds using a wideband click as the probe presented alternately with 1,000- and 2,000-Hz tones or BBN activators. They reported ASR thresholds to be from 2.2 to 9.4 dB below clinical ASR thresholds depending on the activator stimulus. An automated WAI method for assessing ASR thresholds in newborns was recently reported by Keefe et al. (2010). In that study a combination of WAI middle ear tests and ASR thresholds predicted newborn hearing screening outcomes.

Although these new methods have resulted in lower ASR thresholds than clinical tests, which may result in the measurement of the ASR in a greater proportion of patients, more research is needed to evaluate the methods for various ages and with various degrees and types of hearing loss.

CONCLUSIONS

The ASR test battery is an established part of the clinical armamentarium of the audiologist. No other test in the toolbox can provide more information about a variety of components that contribute to the human auditory system: Middle ear, cochlea, CN VIII, brainstem, CN VII, and stapedius muscle. A normal ASR test can help confirm normal function in any of these components, whereas an abnormal ASR test can help point to a lesion. The addition of ASR test results with other physiological and behavioral tests provides unique information that strengthens the test battery and provides a cross-check of results.

ACKNOWLEDGMENTS

The authors thank Angela Garinis for helpful comments on a draft of the chapter, and Dan Putterman and Monica Feeney for assistance with graphics. The content of this chapter does not represent the views of the Department of Veterans Affairs or of the United States Government.

FOOD FOR THOUGHT

1. A patient with a suspected right vestibular schwannoma has been referred to you for a hearing evaluation. What symptoms might the patient report? What results might you see on a standard clinical test battery, including puretone thresholds, speech discrimination scores, and immittance (specifically, what ASR pattern would you expect)? What special tests would you recommend and why? Assume that a right vestibular schwannoma was eventually diagnosed and the neurotologist recommended surgical removal of the tumor which would likely result in loss of most or all of the hearing on the right side. What amplification options would be appropriate for the patient after the surgery? (Assume two scenarios, one in which the hearing in the left ear is normal, and one in which there is moderate hearing loss in the left ear).
2. You are opening your own private practice, and you need to define your immittance test battery protocol. Assume that you will test patients from newborns to adult. What test equipment would you purchase? Define the age-specific probe tones, activators, and normative data that you would use (and identify conditions for which normative data may not be available). Think about your report templates—how will describe your results for ASR present when expected, present when not expected, etc.?
3. You are unable to obtain behavioral test results for a 3-year-old child, except for speech awareness at 40 dB HL in the sound field. She is very cooperative for the physiological test battery and has normal otoacoustic emissions in one ear, absent in the other, and normal tympanometry bilaterally at 226 Hz. What additional information about this child could you obtain with ipsilateral and contralateral ASR thresholds? If the ipsilateral and contralateral ASR thresholds were normal at 1,000 Hz, would you consider the child to have normal hearing? Why or why not?

REFERENCES

Aiken SJ, Andrus JN, Bance M, Phillips DP. (2013) Acoustic stapedius reflex function in man revisited. *Ear Hear.* 34(4), e38–e51.

Alford BR, Jerger JF, Coats CA, Peterson CR, Weber SC. (1973) Neurophysiology of facial nerve testing. *Arch Otolaryngol.* 97(2), 214–219.

American National Standards Institute. (2004) *Method for manual puretone threshold audiometry* (ANSI S3.21–2004). New York: American National Standards Institute.

American National Standards Institute. (2012) *Specifications for Instruments to Measure Aural Acoustic Impedance and Admittance (aural acoustic immittance)* (ANSI S3.39–1987–R2012). New York: American National Standards Institute.

American Speech-Language-Hearing Association. (1990) Guidelines for screening for hearing impairment and middle-ear disorders. *ASHA.* 32(suppl 2), 17–24.

Anderson H, Barr B, Wedenberg E. (1969) Early diagnosis of 8th-nerve tumours by acoustic reflex tests. *Acta Otolaryngol Suppl.* 263, 232–237.

Arriaga MA, Luxford WM. (1993) Impedance audiometry and iatrogenic hearing loss. *Otolaryngol Head Neck Surg.* 108, 70–72.

Banerjee A, Whyte A, Atlas MD. (2005) Superior canal dehiscence: Review of a new condition. *Clin Otolaryngol.* 30(1), 9–15.

Battmer R, Laszig R, Lehnhardt E. (1990) Electrically elicited stapedius reflex in cochlear implant patients. *Ear Hear.* 11(5), 370–374.

Berlin CI, Hood LJ, Morlet T, Wilensky D, St. John P, Montgomery E, et al. (2005) Absent or elevated middle ear muscle reflexes in the presence of normal otoacoustic emissions: A universal finding in 136 cases of auditory neuropathy/dys-synchrony. *J Am Acad Audiol.* 16(8), 546–553.

Borg E. (1973) On the neuronal organization of the acoustic middle ear reflex. A physiological and anatomical study. *Brain Res.* 49, 101–123.

Borg E, Counter SA, Rosler G. (1984) Theories of middle-ear muscle function. In: Silman S, ed. *The Acoustic Reflex: Basic Principles and Clinical Applications.* Orlando: Academic Press, pp 63–99.

Borg E, Moller AR. (1967) Effect of ethyl alcohol and pentobarbital sodium on the acoustic middle ear reflex in man. *Acta Otolaryngol.* 64, 415–426.

Borg E, Nilsson R, Liden G. (1982) Fatigability of the stapedius reflex in industrial noise. A field study. *Acta Otolaryngol.* 94(5–6), 385–393.

Brask T. (1979) The noise protection effect of the stapedius reflex. *Acta Otolaryngol Suppl.* 360, 116–117.

Caner G, Olgun L, Gultekin G, Balaban M. (2007) Optimizing fitting in children using objective measures such as neural response imaging and electrically evoked stapedius reflex threshold. *Oto and Neurotol.* 28, 637–640.

Clemis JD, Samo CN. (1980) The acoustic reflex latency test: Clinical application. *Laryngoscope.* 90(4), 601–611.

Cohen M, Prasher D. (1988) The value of combining auditory brainstem responses and acoustic reflex threshold measurements in neuro-otological diagnosis. *Scand Audiol.* 17(3), 153–162.

DiGiovanni JJ, Ries DT. (2007) Stapedial reflex and ears with high static acoustic admittance. *Am J Aud.* 16, 68–74.

Downs DW, Crum MA. (1980) The hyperactive acoustic reflex. Four case studies. *Arch Otolaryngol.* 106(7), 401–404.

Feeney MP, Keefe DH. (1999) Acoustic reflex detection using wideband acoustic reflectance, admittance, and power measurements. *J Speech Lang Hear Res.* 42, 1029–1041.

Feeney MP, Keefe DH. (2001) Estimating the acoustic reflex threshold from wideband measures of reflectance, admittance, and power. *Ear Hear.* 22, 316–332.

Feeney MP, Keefe DH, Marryott LP. (2003) Contralateral acoustic reflex thresholds for tonal activators using wideband energy reflectance and admittance. *J Speech Lang Hear Res.* 46, 128–136.

Feeney MP, Keefe DH, Sanford CA. (2004) Wideband reflectance measures of the ipsilateral acoustic stapedius reflex threshold. *Ear Hear.* 25, 421–430.

Feeney MP, Sanford CA. (2008). Middle-ear measurement. In: Madell J, Flexer C, *Pediatric Audiology: Diagnosis, Technology, and Management.* New York: Thieme; pp 115–122.

Forquer BD. (1979). The stability of and the relationship between the acoustic reflex and uncomfortable loudness levels. *J Am Aud Soc.* 5, 55–59.

Fortnum H, O'Neill C, Taylor R, Lenthall R, Nikolopoulos T, Lightfoot G, et al. (2009) The role of magnetic resonance imaging in the identification of suspected acoustic neuroma: a systematic review of clinical and cost effectiveness and natural history. *Health Technol Assess.* 13(18): iii–iv, ix–xi, 1–154.

Gantz BJ, Rubinstein JT ,Gidley P, Woodworth GG.(1999) Surgical management of Bell's palsy. *Laryngoscope.* 109(8), 1177–1188.

Gelfand SA. (1984) The contralateral acoustic reflex. In: Silman S, ed. *The Acoustic Reflex: Basic Principles and Clinical Applications.* Orlando, Academic Press; 137–186.

Gelfand SA. (1994) Acoustic reflex threshold tenth percentiles and functional hearing impairment. *J Am Acad Audiol.* 5(1), 10–16.

Gelfand SA. (2009) Chapter 10: The acoustic reflex. In: Katz J, Medwetsky L, Burkard R, Hood L. eds. *Handbook of Clinical Audiology.* 6th ed. 189–221. Philadelphia: Lippincott Williams & Wilkins.

Gelfand SA, Silman S (1982) Acoustic reflex thresholds in brain-damaged patients. *Ear Hear.* 3(2), 93–95.

Gelfand SA, Schwander T, Silman S. (1990) Acoustic reflex thresholds in normal and cochlear-impaired ears; effects of no-response rates on 90th percentiles in a large sample. *J Speech Hear Disord.* 55, 198–205.

Gerhardt KJ, Hepler EL Jr. (1983) Acoustic-reflex activity and behavioral thresholds following exposure to noise. *J Acoust Soc Am.* 74(1), 109–114.

Hannley M. (1986) *Basic Principles of Auditory Assessment.* San Diego: College Hill Press.

Hirsch A, Anderson H. (1980a) Elevated stapedius reflex threshold and pathologic reflex decay. Clinical occurrence and significance. *Acta Otolaryngol Suppl.* 368, 1–28.

Hirsch A, Anderson H. (1980b) Audiologic test results in 96 patients with tumours affecting the eighth nerve. A clinical study with emphasis on the early audiological diagnosis. *Acta Otolaryngol Suppl.* 369, 1–26.

Hodges AV, Butt S, Dolan-Ash S, Balkany TJ. (1999) Using electrically evoked auditory reflex thresholds to fit the CLARION cochlear implant. *Ann Otol Rhinol Laryngol.* 177, 64–68.

Hodges AV, Butts SL, King JE. (2003) Chapter 4: Electrically evoked stapedial reflexes: Utility in cochlear implant patients. In: Cullington HE, ed. *Cochlear Implants: Objective Measures.* pp 81–95. Philadelphia: WHurr Publishers; pp 81–95.

Hunter LL, Ries DT, Schlauch RS, Levine SC, Ward WD. (1999) Safety and clinical performance of acoustic reflex tests. *Ear Hear.* 20, 506–514

Jerger J, Anthony L, Jerger S, Mauldin L. (1974a) Studies in impedance audiometry. 3. Middle ear disorders. *Arch Otolaryngol.* 99(3), 165–171.

Jerger J, Harford E, Clemis J, Alford B. (1974b) The acoustic reflex in eighth nerve disorders. *Arch Otolaryngol.* 99, 409–413.

Jerger S, Jerger J. (1975) Extra- and intra-axial brain stem auditory disorders. *Audiology.* 14(2), 93–117.

Jerger S, Jerger J. (1977) Diagnostic value of crossed vs uncrossed acoustic reflexes: Eighth nerve and brain stem disorders. *Arch Otolaryngol.* 103(8), 445–453.

Jerger J, Oliver TA, Rivera V, Stach BA. (1986) Abnormalities of the acoustic reflex in multiple sclerosis. *Am J Otolaryngol.* 7(3), 163–176.

Kankkunen A, Liden G. (1984) Ipsilateral acoustic reflex thresholds in neonates and in normal-hearing and hearing-impaired pre-school children. *Scand Audiol.* 13, 139–144.

Keefe DH, Fitzpatrick D, Liu YW, Sanford CA, Orga MP. (2010) Wideband acoustic-reflex test in a test battery to predict middle-ear dysfunction. *Hear Res.* 263(1–2), 52–65.

Keefe DH, Ling R, Bulen JC. (1992) Method to measure acoustic impedance and reflection coefficient. *J Acoust Soc Am.* 91, 470–485.

Kei J. (2012) Acoustic stapedial reflexes in healthy neonates: Normative data and test-retest reliability. *J Am Acad Audiol.* 23(1), 46–56.

Keith RW, Garza-Holquin Y, Smolakm L, Pensak ML. (1987) Acoustic reflex dynamics and auditory brain stem responses in multiple sclerosis. *Am J Otol.* 8(5), 406–413.

Lyons MJ. (1978) The central location of the motor neurons to the stapedius muscle in the cat. *Brain Res.* 143, 437–444.

Martin FN, Coombes S. (1974) Effect of external ear canal pressure on the middle-ear muscle reflex threshold. *J Speech Hear Res.* 17, 526–530.

Mazlan R, Kei J, Hickson L. (2009) Test-retest reliability of the acoustic stapedial reflex test in healthy neonates. *Ear Hear.* 30, 295–301.

Merchant SN, Rosowski JJ, McKenna MJ.(2007) Superior semicircular canal dehiscence mimicking otosclerotic hearing loss. *Adv Otorhinolaryngol.* 65, 137–145.

Metz O. (1952) Thresholds of reflex contractions of muscles of middle ear and recruitment of loudness. *Arch Otolaryngol.* 55, 536–543.

McMillan PM, Bennett MJ, Marchant CD, Shurin PA. (1985a) Ipsilateral and contralateral acoustic reflexes an neonates. *Ear Hear.* 6, 320–324.

McMillan PM, Marchant CD, Shurin PA. (1985b) Ipsilateral acoustic reflexes in infants. *Ann Otol Rhinol Laryngol.* 94, 145–148.

Mikulec AA, McKenna M, Ramsey MJ, Rosowski JJ, Hermann BS, Rauch SD, et al. (2004) Superior semicircular canal dehiscence presenting as conductive hearing loss without vertigo. *Otol Neurotol.* 25(2), 121–129.

Minor LB, Solomon D, Zinreich JS, Zee DS.(1998) Sound- and/or pressure-induced vertigo due to bone dehiscence of the superior semicircular canal. *Arch Otolaryngol Head Neck Surg.* 124(3), 249–258.

Neumann J, Uppenkamp S, Kollmeier B. (1996). Detection of the acoustic reflex below 80 dB HL. *Audiol Neurootol.* 1(6), 359–369.

Newton JR, Shakeel M, Flatman S, Beattie C, Ram B. (2010) Magnetic resonance imaging screening in acoustic neuroma. *Am J Otolaryngol.* 31(4), 217–220.

Nikolopoulos TP, Fortnum H, O'Donoghue G, Baguley D. (2010) Acoustic neuroma growth: A systematic review of the evidence. *Otol Neurotol.* 31(3), 478–485.

Olsen WO, Stach BA, Kurdziel SA. (1981) Acoustic reflex decay in 10 seconds and in 5 seconds for Meniere's disease patients and for VIIIth nerve tumor patients. *Ear Hear.* 2(4), 180–181.

Osterhammel D, Osterhammel P. (1979) Age and sex variations for the normal stapedial reflex thresholds and tympanometric compliance values. *Scand Audiol.* 8, 153–158.

Phillips DP, Stuart A, Carpenter M. (2002) Re-examination of the role of the human acoustic stapedius reflex. *J Acoust Soc Am.* 111(5Pt 1), 2200–2207.

Qui WW, Stucker F. (1998) Characteristics of acoustic reflex latency in normal-hearing subjects. *Scand Audiol.* 27, 43–49.

Rosowski JJ, Songer JE, Nakajima HH, Brinsko KM, Merchant SN. (2004) Clinical, experimental, and theoretical investigations of the effect of superior semicircular canal dehiscence on hearing mechanisms. *Otol Neurotol.* 25(3), 323–332.

Rousch J, Tait C. (1985) Pure tone and acoustic immittance screening of preschool aged children: An examination of referral criteria. *Ear Hear.* 6, 245–249.

Schairer KS, Ellison JC, Fitzpatrick D. (2007) Wideband ipsilateral measurements of middle-ear muscle reflex thresholds in children and adults. *J Acoust Soc Am.* 121, 3607–3616.

Schairer KS, Feeney MP, Sanford CA. (2013) Acoustic reflex measurement. *Ear Hear.* 34(7suppl 1), 43s–47s.

Sells JP, Hurley RM, Morehouse CR, Douglas JE. (1997) Validity of the ipsilateral acoustic reflex as a screening parameter. *J Am Acad Audiol.* 8, 132–136.

Silman S, Gelfand SA. (1981) The relationship between magnitude of hearing loss and acoustic reflex threshold levels. *J Speech Hear Disord.* 46, 312–316.

Silman S, Silverman CA, Gelfand SA, Lutolf J, Lynn DJ. (1988) Ipsilateral acoustic-reflex adaptation testing for detection of facial-nerve pathology: Three case studies. *J Speech Hear Disord.* 53(4), 378–382.

Silverman CA, Silman S, Miller MH. (1983) The acoustic reflex threshold in aging ears. *J Acoust Soc Am.* 73, 248–255.

Simon U, Pirsig W. (1973) Influence of chlorpromazine on audiometric tests. *Scand Audiol.* 3, 99–105.

Sprague BH, Wiley TL, Goldstein R. (1985) Tympanometric and acoustic-reflex studies in neonates. *J Speech Hear Res.* 28, 265–272.

Starr A, Sininger YS, Pratt H. (2000) The varieties of auditory neuropathy. *J Basic Clin Physiol Pharmacol.* 11(3), 215–230.

Voss SE, Rosowski JJ, Merchant SN, Thornton AR, Shera CA, Peake WT. (2000) Middle ear pathology can affect the ear-canal sound pressure generated by audiologic earphones. *Ear Hear.* 21(4), 265–274.

Weatherby LA, Bennett MJ. (1980) The neonatal acoustic reflex. *Scand Audiol.* 9, 103–110.

Wiegand DA, Poch NE. (1988) The acoustic reflex in patients with asymptomatic multiple sclerosis. *Am J Otolaryngol.* 9(5), 210–216.

Wiley TL, Oviatt DL, Block MG. (1987) Acoustic-immittance measures in normal ears. *J Speech Hear Res.* 30, 161–170.

Wilson RH. (1979) Factors influencing the acoustic-immittance characteristics of the acoustic reflex. *J Speech Hear Res* 22, 480–499.

Wilson RH, Margolis RH. (1999) Acoustic reflex measurements. In: Musiek FE, Rintelmann WF, eds. *Contemporary Perspectives in Hearing Assessment.* Zug, Switzerland: Pearson; p 142.

Wilson RH, McCullough JK, Lilly DH. (1984) Acoustic-reflex adaptation: Morphology and half-life data for subjects with normal hearing. *J Speech Hear Res.* 27(4), 586–595.

Wormald PJ, Rogers C, Gatehouse S. (1995) Speech discrimination in patients with Bell's palsy and a paralysed stapedius muscle. *Clin Otolaryngol Allied Sci.* 20(1), 59–62.

Zuniga MG, Janky KL, Nguyen KD, Welgampola MS, Carey JP. (2013) Ocular versus cervical VEMPs in the diagnosis of superior semicircular canal dehiscence syndrome. *Otol Neurotol.* 34(1), 121–126.

CHAPTER 11

Introduction to Auditory Evoked Potentials

Robert Burkard and Manuel Don

INTRODUCTION

The seventh edition of the Handbook of Clinical Audiology includes eight chapters specifically dealing with auditory-evoked potentials (AEPs). The chapters range from the very short latency response components using electrocochleography (EcochG) to long-latency responses. These responses vary in terms of generators, time epochs, stimulus, and response dependencies, as well as clinical applications. However, they share a number of commonalities, in terms of the use of differential amplification and time-domain signal averaging to observe a response that is small when compared to the magnitude of the background electroencephalographic (EEG) activity. The present chapter will (1) present a brief overview of the central auditory nervous system from eighth nerve through brainstem; (2) provide a brief overview of the various AEPs, and their clinical applications; (3) present an introduction to the instrumentation and principles underlying the acquisition of AEPs; (4) review the normative aspects of the auditory brainstem response (ABR) as an introduction to the clinical applications of the ABR presented in Chapters 13, 14, and 16; and (5) describe two relatively new techniques, involving novel stimulus manipulations, designed to improve the ABR for site-of-lesion testing and for hearing screening/threshold estimation.

THE EIGHTH NERVE AND AUDITORY BRAINSTEM

For threshold estimation, it is not very important to know the generators of a particular AEP peak. In contrast, for such clinical applications as site-of-lesion testing (see Chapter 13), or intraoperative monitoring (see Chapter 16), knowledge of the generators of a particular peak can be invaluable. Early work focusing on the ABR peak generators (e.g., Buchwald and Huang, 1975) used lesion studies in animals (cats) and related the loss of peaks or changes in amplitude to the level of the lesion. This led to a mapping of a given ABR peak to a specific generator. This mapping was problematic for two reasons. First, other than for the first peak (generated by the eighth nerve), multiple regions of the auditory nervous system are activated in a temporally overlapping fashion, making a one-to-one mapping of peak to generator impossible. Second, even though in the animal studies it was often possible to identify the dominant contributor to a given peak, the generalization to humans was confounded by the unusually long auditory nerve in humans compared to other mammals (Moller, 1994). Despite these difficulties, our current knowledge of ABR peak generators allows us to make useful clinical decisions for site-of-lesion testing, as well as to interpret changes in the ABR during intraoperative monitoring.

The auditory nerve, one branch of the vestibulocochlear (eighth) cranial nerve, projects from the hair cells to the cochlear nuclei (CN). According to Spoendlin (1972), 90% to 95% of auditory nerve fibers are type I fibers, and 5% to 10% are type II fibers. Type I afferent dendrites innervate inner hair cells (IHCs), whereas type II afferents innervate outer hair cells (OHCs). Type I fibers are bipolar and heavily myelinated, whereas type II afferents are sparsely myelinated and are pseudomonopolar (i.e., the axon and dendrites arise from a common neurite arising from the cell body, and then the two processes split and project in different directions). The cell bodies of both type I and type II auditory peripheral afferents are contained in the spiral ganglion. Based on number alone, it is clear that IHCs and type I afferents deliver most of the auditory information to the central auditory nervous system. The auditory nerve passes through the internal auditory meatus of the cochlea, and upon entering the posterior fossa, it projects to the lateral aspect of the brainstem, near the pontomedullary junction. These fibers bifurcate and terminate in the CN of the caudal pons. There are three divisions of the CN: The anteroventral cochlear nucleus (AVCN), the posteroventral cochlear nucleus (PVCN), and the dorsal cochlear nucleus (DCN). Each of these nuclei is tonotopically organized (i.e., each subnucleus has a map relating place to a specific best or characteristic frequency). Best or characteristic frequency is the frequency to which that unit responds at the lowest sound pressure level. Indeed, a tonotopic organization is a characteristic of many auditory nuclei. Each of the various subnuclei of the CN has unique anatomical cell types, unique electrophysiological response patterns, and unique connections. Auditory nerve fibers are uniform in cell type (mostly type I afferents) and present fairly uniform response properties (sustained responses showing adaptation, but differing in terms of best frequency

and spontaneous discharge rate). However, at the level of the CN such homogeneity is no longer seen. Multiple cell types are present in the CN, with input from various structures. Similarly, response characteristics vary greatly, reflecting, among other things, differing membrane properties and combinations of excitatory and inhibitory input. The details of these response properties go well beyond the scope of the present review. Detailed reviews of the anatomy and physiology of the auditory nervous system can be found in Webster et al. (1992) and Popper and Fay (1992).

The three subnuclei of the CN have major projections (called acoustic striae) to more rostral regions of the brainstem: The dorsal acoustic stria arises from the DCN and projects to the contralateral inferior colliculus (IC). The ventral acoustic stria projects from the AVCN to the superior olivary complex (SOC) bilaterally. The intermediate acoustic stria projects from the PVCN to the ipsilateral SOC. The SOC is composed of multiple subnuclei. The periolivary nuclei are integral to the descending auditory system, which projects to the OHCs or the type I afferents beneath the IHCs via the crossed and uncrossed olivocochlear bundles. This descending system is involved in the suppression of otoacoustic emissions (OAEs) (see Chapter 19). In terms of the ascending system, three SOC subnuclei are of importance: the medial superior olive (MSO), the lateral superior olive (LSO), and the medial nucleus of the trapezoid body (MNTB). The SOC is where inputs from both ears first converge. Information from the contralateral and ipsilateral CN projects to medial and lateral dendritic tufts of the MSO, respectively. Single-unit responses from the MSO show similar tuning curves from both ears, suggesting convergence of excitatory input from similar best frequency regions from the right and left CN. For example, the right LSO receives direct input from the right CN, whereas input from the left CN crosses midline to the right MNTB, and then to the right LSO. There is evidence that the input from the MNTB is inhibitory. Both MSO and LSO are tonotopically organized. The SOC clearly adds binaural processing to the monaural CN input.

From the SOC, the major output is via the lateral lemniscus (LL). There are two subnuclei of the LL: the dorsal nucleus of the LL (DNLL) and the ventral nucleus of the LL (VNLL). The LL terminates in the IC. The IC are on the dorsal aspect of the midbrain and appear as a pair of protuberances just below the paired superior colliculi. There are several subnuclei of the IC, but the main division is the central nucleus. The central nucleus of the IC is tonotopically organized, with a laminar arrangement. You can visualize the isofrequency (same frequency) laminae like the layers of an onion, with each layer of onion having a narrow range of best frequencies. The other regions of the IC have units with broader tuning curves, making determination of their frequency organization complicated. These other divisions appear to respond to not only auditory input, but also to somatosensory and visual inputs.

Now that we know something about the auditory nerve and brainstem, we can briefly provide a listing of the primary generators of the various ABR peaks. The human ABR is composed of a series of up to seven vertex-positive peaks that occur within 10 ms of stimulus onset to moderate-level click stimuli in adults. The first five peaks have received the most attention scientifically and clinically. Based largely on the studies performed by Moller and Jannetta during intraoperative monitoring, we can assign the following peak generators: Wave I arises from the distal auditory nerve, Wave II arises from the proximal eighth nerve, Wave III is primarily generated by the CN, Wave IV appears to be generated by the SOC, Wave V (the peak) appears to emanate from the LL, whereas the trough following Wave V comes predominantly from the IC (Moller, 1994).

AN OVERVIEW OF AUDITORY-EVOKED POTENTIALS

If you were to simultaneously measure the responses from all auditory nervous system structures following presentation of an acoustic stimulus, you would record activity in the cochlea, auditory nerve, auditory brainstem, thalamus, and auditory cortex. Multiple brain regions would be activated at the same time. However, it would be true that, generally speaking, the more caudal structures in the auditory nervous system would have shorter onset latencies than the more rostral structures. This latency increase for more rostral structures is the result of the finite action potential conduction velocity and the delay as the activity passes through chemical synapses. Although we have no noninvasive way of recording from these various auditory nuclei directly, it is possible to record a series of responses from the scalp (using noninvasive surface electrodes) which have latencies ranging from one one-thousandth to several tenths of a second. A millisecond (one-thousandth of a second) and a microsecond (one-millionth of a second) will be convenient time units. Microvolts (one-millionth of a volt) will be convenient amplitude units. Because of the progressive latency increase of responses from more rostral auditory structures, it is popular to classify AEPs by their response time following the onset of a transient stimulus (e.g., a click).

EcochG refers to the responses from the cochlea and auditory nerve, using a recording electrode located in close proximity to the inner ear. Two responses arise from the hair cells: the cochlear microphonic (CM) and the summating potential (SP) (Dallos, 1973; Davis, 1976). Each has a very short latency (1 ms or so), which is basically the delay from stimulus onset to hair cell excitation. The CM has the same waveform as the stimulus, and so a 2,000-Hz toneburst will produce a CM with spectral energy primarily at 2,000 Hz. The SP is a direct current (DC) response, which continues for the duration of the eliciting stimulus. The response from the acoustic portion of the eighth cranial nerve is

called either the whole-nerve action potential (WNAP) or the compound action potential (CAP). The first two negative peaks of the CAP are labeled N_1 and N_2. Unlike the CM and SP, which continue for the duration of the stimulus, the CAP occurs at stimulus onset (and sometimes offset). Many AEPs (CAP, ABR, middle latency response: MLR, slow vertex potential: SVP) are responsive to the stimulus onset. The CAP has a latency that is roughly 1 ms longer than the CM or SP, which is the result of the synaptic delay from hair cell depolarization to the onset of auditory nerve fiber discharge. Unlike the other AEPs, the EcochG responses are typically NOT measured with scalp electrodes, but rather from electrodes placed in the ear canal, on or near the tympanic membrane, or on the promontory or round window of the inner ear (see Chapter 12).

The ABR includes a series of five to seven peaks arising from auditory nerve and brainstem structures (Moller, 1994), occurring within 10 ms of the onset of a moderate-intensity click stimulus in otologically, audiologically, and neurologically intact adults. Most investigators label the peaks with capital Roman numerals (I through VIII), following the convention established by Jewett and Williston (1971). An ABR from a normal adult is shown in Figure 11.1. The ABR is, without question, the most clinically useful AEP at the present time. It can be used for estimating hearing threshold (Chapter 14) and differential diagnosis of peripheral and central abnormalities (Chapter 13) and for intraoperative monitoring (Chapter 16).

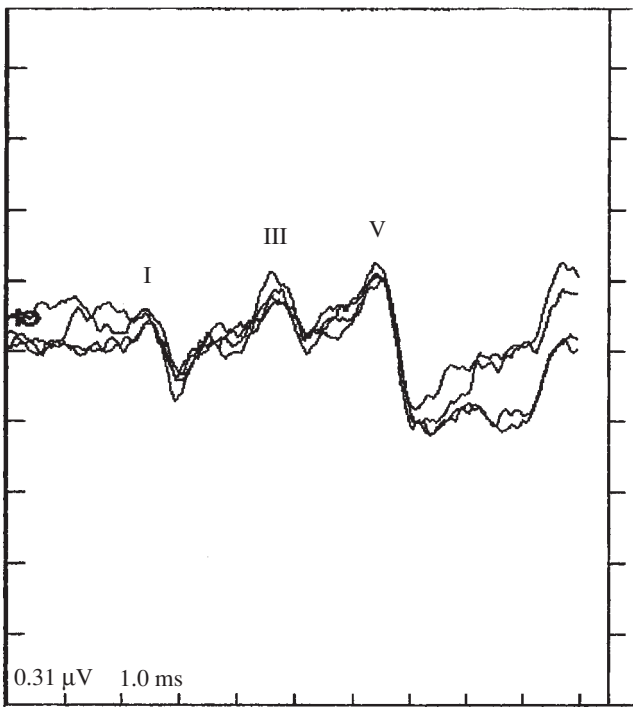


FIGURE 11.1 An example of the human ABR. Waves I, III, and V are labeled. [Reprinted from Burkard R, Secor C. [2002] Overview of auditory evoked potentials. In: Katz J, ed. *Handbook of Clinical Audiology*. 5th ed. Baltimore, MD: Lippincott, Williams & Wilkins; pp 233–248, with the permission of Lippincott Williams & Wilkins.]

MLRs are typically recorded over a time window of 80 to 100 ms. Generators are thought to include thalamus and auditory cortex (Chapter 17; Kraus et al., 1994; Moller, 1994). Unlike EcochG and ABR, the MLR appears to be affected by subject variables such as attention and arousal. Peak-labeling nomenclature varies somewhat with investigator, but the polarity of the peak is typically indicated by a capital P or N (positive or negative), subscripted by small-case early letters of the alphabet (e.g., P_a and N_b). The earliest MLR peaks may be labeled P_o and N_o. AEP responses occurring beyond ~75 ms are collectively called the SVP or late component responses. These responses are most commonly labeled with a capital N or P, to indicate peak polarity and subscripted with an Arabic number indicating which SVP peak of that polarity it is (e.g., P_1 is the first positive SVP peak). Alternatively, an Arabic number indicating average peak latency may be used as a subscript. For example, N_{200} would be the negative peak with a mean latency of approximately 200 ms. The SVP is affected by attention and arousal (see Chapter 18). The term event-related potential (ERP) is used when referring to long-latency responses that are strongly dependent on the attention and arousal of the subject (Kraus and McGee, 1994). Innovative paradigms have been developed to study ERPs. One commonly used paradigm is the oddball paradigm. In its simplest application, the oddball paradigm involves the use of two different stimuli, one that occurs frequently and another that occurs infrequently. Responses to the frequent stimulus are averaged in one memory array, whereas the responses to the infrequent stimuli are averaged in a second memory array. Often, there are differences in the responses to the frequent and infrequent stimuli, with these differences depending on whether the subject is attending to the stimuli or not. Differences can also be due to the acoustical differences between the rare and frequent stimuli, an effect that can be controlled by using the same stimuli as both the "frequent" and the "rare" stimulus in different runs. If attending, there is an additional response called the P_3 or P_{300} response to the infrequent stimulus. Thus, the P_{300} can be used to evaluate the effects of attention on the ERP response. Differences to the rare and frequent stimuli can also be seen in the response known as the mismatch negativity (MMN), and this occurs even when the subject is not actively attending to the stimuli. Both the P_{300} and the MMN are discussed in Chapter 18.

A relative newcomer to the family of AEP responses is the auditory steady-state response (ASSR). This response represents a sustained response and can be elicited by trains of clicks, noisebursts, or tonebursts, as well as amplitude-modulated (AM) and/or frequency-modulated (FM) stimuli. This response has been extensively investigated in recent years (see Chapter 15).

CLINICAL APPLICATIONS OF AEPS

There are a number of well-established clinical uses of AEPs. First, AEPs can be (and are) used for hearing screening and to estimate hearing thresholds of difficult-to-test populations. The ABR is currently the most popular AEP for hearing screening and threshold estimation in the United States (see Chapter 14). However, in recent years there has been a lot of interest in applying the ASSR to the problem of threshold estimation (see Chapter 15). EcochG is more invasive than the ABR (or ASSR), especially if a transtympanic electrode is used, whereas the middle and long-latency responses have the disadvantage of being at least somewhat dependent on attention and arousal (see Chapters 17 and 18). There are clear advantages (in terms of expense and time) in using OAEs rather than ABRs (or, perhaps, ASSRs) for hearing screening. However, OAEs appear to be less than optimal for threshold estimation, and OAEs in isolation will not allow identification of those patients with auditory neuropathy (see Chapters 14 and 19).

AEPs are also used for site-of-lesion testing. For example, the ABR is useful for differentiating conductive, sensory, and retrocochlear disorders (see Chapter 13). Recent studies have revealed a clinical entity termed "auditory neuropathy" (Hood, 1998; Starr et al., 1996). Subjects with auditory neuropathy often have abnormal or missing ABRs, missing acoustic reflexes, normal OAEs but missing contralateral suppression of OAEs, and speech discrimination (especially in noise) that is commonly poorer than predicted by the threshold audiogram. These patients show no evidence of CNS lesions by conventional imaging modalities. The diagnosis is made based on the unusual pattern of audiologic results (including OAEs and AEPs), not on diagnostic imaging procedures. Auditory neuropathy is discussed in more detail in Chapter 12.

AEPs (primarily EcochG and ABR) are also used for intraoperative monitoring (see Chapter 16). For intraoperative monitoring, the patient's baseline responses are used as the basis for monitoring changes in inner ear/auditory nerve and/or brainstem function during surgery. Degradations in the response (such as increases in peak latency and/or decreases in amplitude) are used to warn the surgeon that damage is being done to the auditory system, thus giving the surgeon the opportunity to modify their procedures to preserve auditory and/or neural function.

The above list of clinical uses of AEPs is not exhaustive, but does reflect the most common uses of AEPs in clinical audiology today. AEPs are now being used in the mapping of cochlear implants in the pediatric population. It is likely that ERPs such as the P_{300} and the MMN will find a place in the diagnosis and perhaps in the efficacy of treatment of such complex clinical entities as central auditory processing disorder.

THE TECHNICAL DETAILS

It is necessary to present an acoustic (or perhaps an electrical) stimulus to elicit an AEP. To record the AEP, the electrical response must be recorded from the human scalp (or, for EcochG, from the ear canal, tympanic membrane, or round window/promontory). Electrodes serve as the interface between the scalp (or ear) and the electronic instrumentation. Because AEPs are small in amplitude, we must use special amplifiers (called bioamplifiers) to make these signals large enough for further signal processing. It is also common to get rid of undesirable electrical activity by the use of filtering. Finally, the scalp-recorded electrical activity must be converted into a binary format so that it can be utilized by a digital computer. The device that accomplishes this transformation is an analog-to-digital converter (ADC). Once in binary form, the data can be manipulated by a digital computer. This manipulation can include additional filtering (called digital filtering), the elimination of responses deemed to be too noisy (called artifact rejection), and a synchronization of stimulus onset and response recording that we will refer to as time-domain signal averaging. In the sections that follow, we will consider electrodes, bioamplifiers, filters, artifact rejection, analog-to-digital conversion, and time-domain signal averaging. We will also consider the stimuli used to elicit AEPs.

Response Recording

In the clinic, surface (scalp) electrodes and an electrolytic paste or gel typically serve as the interface between the biologic world (the scalp) and the electrical world (the bioamplifier input). Needle electrodes can also be used: These are placed subdermally (under the skin), and the ions present in your body fluids facilitate the transfer of the electrical signal from the tissue to the electrode. Various types of electrodes, both disposable and reusable, are commercially available. Some are special purpose, but most are usable for a variety of recording situations. The locations of electrode placement have been standardized by the development of the international 10–20 system (Jasper, 1958). For the international 10–20 system, left scalp locations are subscripted with odd numbers, whereas right scalp locations are subscripted with even numbers. Here we will briefly review those scalp locations commonly used for AEP recordings. The labels for the left and right mastoid are A_1 and A_2, respectively. F_{pz} is the nasion (bridge of the nose), O_z is the middle of the occipital lobe, and F_z is the middle of the forehead. The vertex (Cz) is halfway between nasion and inion, and midway between the ear canals.

A critical step when applying electrodes is the scalp preparation. Cleaning the scalp by abrasion, using alcohol or other skin-preparation materials, removes dead skin and oils (see Chapter 46 for further information on infection control).

You should measure the interelectrode impedance following electrode application, using either the impedance-testing function that comes packaged with commercially available AEP systems or using a handheld portable electrode impedance meter. Low electrode impedances are desirable (some sources state 5 kΩ or less), but it is sometimes difficult to achieve such low electrode impedances. For example, if you use a tympanic membrane electrode, it is often impossible to achieve a low electrode impedance. High electrode impedance increases the noise floor of the recording and will often result in an increased magnitude of the line frequency and its harmonics (60 Hz and its harmonics in the United States; 50 Hz and its harmonics in Europe and elsewhere). For differential recordings, which are used in most AEP applications, differences in interelectrode impedances can compromise the minimization of common-mode noise (see below).

Three electrodes are used for each recording channel when using a differential bioamplifier. The three leads are referred to as the noninverting, inverting, and the common leads. The noninverting lead is sometimes called the positive or the active lead. The inverting lead is often referred to as the negative or the reference lead. The common lead is sometimes called the ground. In a differential amplifier, the voltages seen by the noninverting and the inverting leads are relative to the common electrode. Another term for voltage is the potential difference, telling us that the voltage of one lead must be expressed relative to the voltage of a second lead. For differential amplification, the voltage from the inverting-common channel is subtracted from the voltage of the noninverting-common channel. Indeed it is this subtraction (or voltage difference) that is the basis of the term "differential amplification." Following this voltage subtraction, the remainder is amplified. Differential amplification substantially reduces noise that is common to the inverting and noninverting leads. This noise, in fact, is called common-mode noise. Why is this useful? Let us imagine that we are recording in a room with a lot of 60-Hz electrical activity (e.g., from the lights). This 60-Hz line noise will often be of similar magnitude and phase at the noninverting and inverting leads. This voltage that is "common" to both of these leads will be "subtracted" prior to amplification. The common-mode rejection ratio (CMRR) is a value that tells us how well "common-mode" activity is eliminated. Let us say we have a bioamplifier gain (V_{out}/V_{in}) of 100,000. If we put 10 μV into the noninverting lead and short the inverting lead (or vice versa), we essentially eliminate the differential amplification, producing a monopolar or single-ended bioamplifier, whose output would be 1 V (1,000,000 μV). If we now apply the same 10 μV into both the inverting and noninverting leads of a differential amplifier, because of the subtraction process used in differential amplification, the output voltage is much smaller, let us say 10 μV. To calculate the CMRR, you divide the differential voltage by the single-ended voltage, and you can convert this to a decibel value by taking 20 times the base 10 logarithm of this ratio:

$$\begin{aligned}\text{CMRR} &= 20 \log_{10}(1{,}000{,}000\ \mu\text{V}/10\ \mu\text{V}) \\ &= 20 \log_{10}(100{,}000) = 100\ \text{dB}\end{aligned}$$

The larger the dB value, the better the CMRR.

Anywhere from 1 to 256 (or more) bioamplifier channels can be used for AEP recordings. In most clinical applications, a small number of channels are used, perhaps 1 to 4. For ABR recordings, it is common to use two recording channels, called the ipsilateral montage and the contralateral montage. For left ear stimulation, the ipsilateral channel might be vertex (noninverting), left mastoid (inverting), and forehead (common), whereas the contralateral channel would be vertex (noninverting), right mastoid (inverting), and forehead (common).

The Digital World

At the heart of evoked potential instruments is a digital microprocessor. However, the bioamplifier output (discussed above) is a continuous (analog) voltage. The analog voltage coming out of the bioamplifier must be converted into a digital form, via a process called analog-to-digital conversion.

Figure 11.2 shows an analog representation of a sinusoid. An analog signal has a voltage value at all moments in time. For example, there is a signal at 1.5 ms and at 1.5001 ms. Also, the voltage can assume any value within a specified voltage range (e.g., ±1 V). For example, the signal can have a voltage of 5 mV and 4.99999 mV. When our analog voltage is converted to a digital format, only a finite number of voltage values can be recorded, and we can only sample the voltage at specific time intervals. We will describe these sampling processes below, but first we will describe the base 2 (or binary) number system.

Binary Number System

In our base 10 (or decimal) number system, each place can represent 10 different values (0 through 9). In the base 2 or binary number system, each place can represent only two values: 0 or 1. In the base 10 system, the rightmost (integer) number is multiplied by 10^0, with the exponent increasing by 1 for each number as we move to the left. The reader who is not familiar with exponents should refer to Speaks (1996). Although we do not think about this much, the number 25 can be described as

$$25 = (2 \times 10^1) + (5 \times 10^0)$$

In a binary number, the rules are the same, except that we can only use 1s and 0s. What is the decimal equivalent of the binary number 1000111?

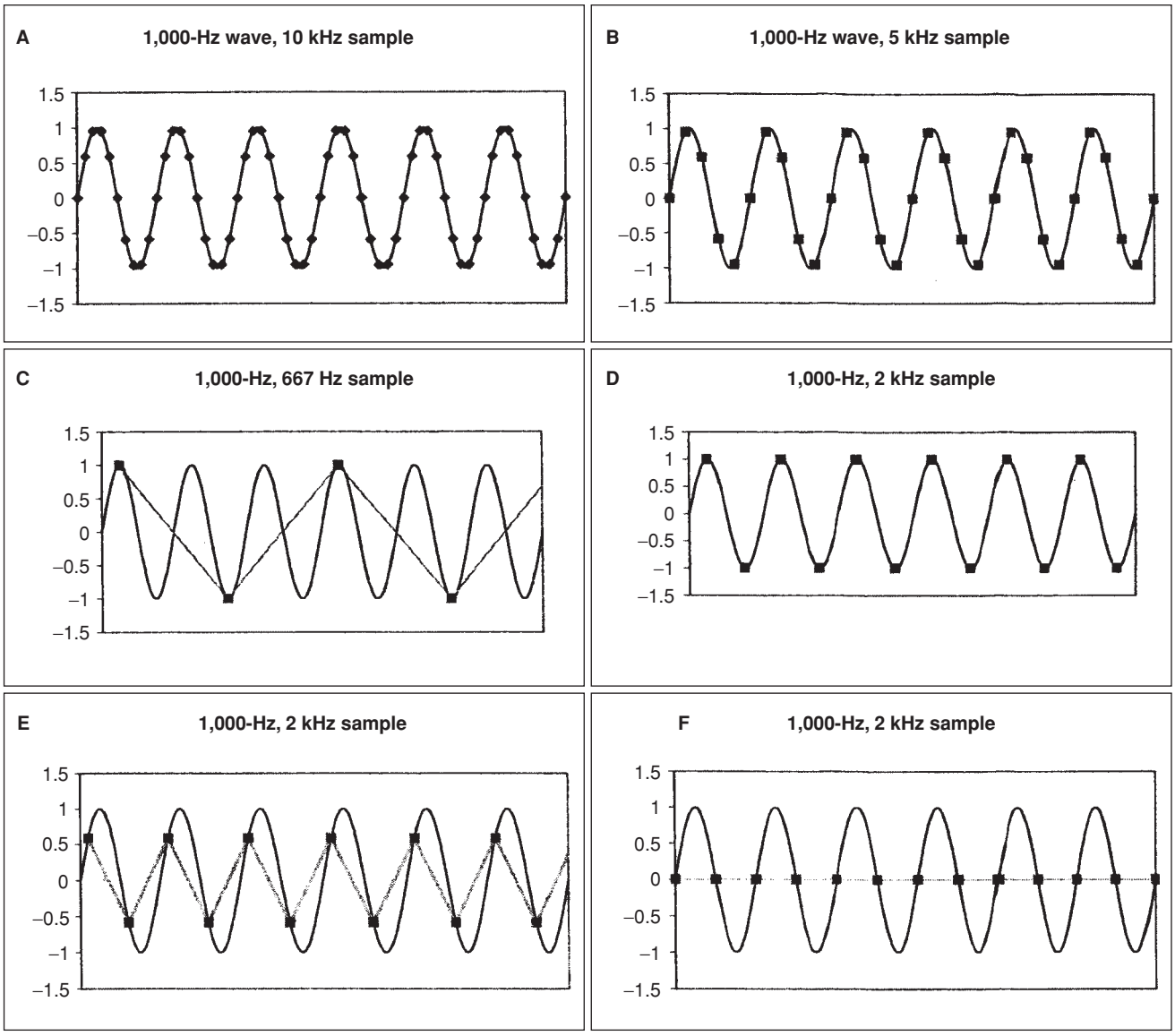


FIGURE 11.2 An analog representation of a sinusoid is shown. The time window shown represents 5 ms. Each subfigure shows an analog representation of a 1,000-Hz sinusoid, sampled at 10,000, 5,000, 667, 2,000, 2,000, and 2,000 Hz, respectively. [Reprinted from Burkard R, Secor C. [2002] Overview of auditory evoked potentials. In: Katz J, ed. *Handbook of Clinical Audiology*. 5th ed. Baltimore, MD: Lippincott Williams & Wilkins; pp 233–248, with the permission of Lippincott Williams & Wilkins.]

$$1000111 = (1 \times 2^6) + (0 \times 2^5) + (0 \times 2^4) + (0 \times 2^3) + (1 \times 2^2) + (1 \times 2^1) + (1 \times 2^0) = 64 + 0 + 0 + 0 + 4 + 2 + 1 = 71$$

As you can see, the rules of binary numbers are the same as those of decimal numbers, except that we can only use 0s and 1s. If each binary digit (called a bit) can only represent two values, then we need to use more than one bit to represent numbers greater than 1: In our example above, the two-place number 71 in decimal is represented in binary by the seven-place number 1000111. We refer to each of these binary places as a bit, and hence 1100010 is a seven-bit binary number. An n-bit binary number can assume 2^n values. Thus, a seven-bit number can assume 2^7 or 128 values. The smallest value this seven-bit number can assume is 0 (0000000), whereas the largest is 127 (1111111 = 64 + 32 + 16 + 8 + 4 + 2 + 1 = 127). When we need to represent a greater number of possible values in the binary system, we must increase the number of bits.

Analog-to-Digital Conversion

An ADC is a device that changes the continuous activity of the real world (such as brain wave activity) into the binary coding used in the digital world. For practical reasons, it is

impossible to sample an infinite number of values. When we convert analog signals into a digital format we cannot sample the signal at all possible times, and we must round off the amplitude values. Thus, with analog-to-digital conversion, we lose information, but we gain the versatile processing capabilities of the digital computer.

The amplitude "round off" mentioned above is known as quantization. When the amplitude of a continuous (analog) signal is "rounded off" in the digital world, there is often a difference between the original analog value and the digital value. This difference is called quantization error. The magnitude of this quantization error is affected by a number of factors. One of these factors is the dynamic range of the ADC. The dynamic range is the largest voltage that can be accurately digitized. Let us say your ADC has a dynamic range of ±1 V. A second factor in determining quantization error is the number of bits of the ADC. Let us assume that we have a ±1 V dynamic range and a 12-bit ADC. A 12-bit ADC provides 4,096 (e.g., 2^{12}) possible values. As the total voltage range is 2 V (from −1 to +1 V) and a 12-bit ADC encodes 4,096 values, the resolution of this system is (with x = number of bits):

$$\text{ADC resolution} = \text{voltage range}/2^x = 10\,\text{V}/2^{12} = 12\,\text{V}/4{,}096 = 0.000488\,\text{V} = 0.488\,\text{mV}.$$

In this example, the resolution between two sequential points is 0.488 mV. The quantization error becomes relevant when we want to separate out amplitudes that are less than this ADC resolution. Assuming that the ADC rounds down to the largest value it has exceeded, the smallest nonzero amplitude this ADC can encode (called the least significant bit, LSB) must be more negative than −0.488 mV or more positive than +0.488 mV. You are probably wondering how this might be a useful resolution, as all EEG activity is quite a bit less than half a millivolt, and hence all electroencephalogram (EEG) voltages would round off to zero. For example, let us assume that off-the-scalp EEG voltage for a subject is ±50 μV (±0.05 mV) and your evoked potential amplitude is 1 μV. How close will you estimate this value in the proposed digital system? Without amplification (as stated above) because 1 μV is less than the quantization range of ±0.488 mV, you will estimate the amplitude as 0 mV. The quantization error in estimating the peak EEG amplitude in this case is 1 μV (1 μV to 0 μV) and produces a significant estimation error. How do you reduce this unacceptable error? First, we could use an ADC with more bits. Sixteen-bit ADCs are currently available at a reasonable cost. This would produce a resolution of $2\,\text{V}/2^{16} = 2{,}000\,\text{mV}/65{,}536 = 0.0305$ mV, or 30.5 μV. With this 16-bit system, 1 μV is still digitally approximated as 0 μV, again giving a quantization error of 1 μV. In other words, this did not help one bit (bad joke, but after all, this is digital electronics we are talking about here). One can get 24-bit ADCs, which provide in excess of 16 million values (16,777,216). In this case, the digital approximation is $2\,\text{V}/2^{24}$, with a resolution of ~0.12 μV; the digital approximation is this example would be 0.96 μV, with a resulting quantization error of 0.04 μV—a much more acceptable "round-off" error.

An alternative to using a very high resolution ADC (above) is to provide bioamplifier gain. All commercially available evoked potential systems provide variable bioamplifier gain. With the use of a bioamplifier, we can amplify the signal of interest so that it occupies most of the ADC voltage range, and in doing so we improve the amplitude resolution of our digital system. For our ±1 V ADC range with 10-bit resolution, let us amplify the signal by 10,000 times. In this case, the full-scale off-the-scalp voltage that the ADC can resolve is 2 V/10,000 = 0.002 mV (note that this equals a total specified EEG range of ±100 μV). Because this 0.2 mV range is encoded by the 10-bit ADC (representing 1,024 values), our off-the-scalp resolution is now 0.2 mV/1,024 = 0.195 μV and our digital estimate of the 1 μV peak amplitude is 0.975 μV, with an error of 0.025 μV. To summarize, we can reduce quantization error using several strategies: reduce the ADC voltage range, increase the number of bits of the ADC, and/or increase bioamplifier gain.

The second limitation of a digital system arises because we sample the continuous analog response at periodic time intervals. The solid line in Figure 11.2A shows a 1,000-Hz analog sine wave. The dots represent the results of digitally sampling this 1,000-Hz sine wave every 0.1 ms. This is called the sampling period. The inverse of the sampling period is the sampling frequency (1/0.1 ms = 1/0.0001 s = 10,000 Hz). Figure 11.2B shows the same 1,000-Hz sine wave, but now sampled half as often: every 0.2 ms. If we visually connect the dots, we can visualize a signal that follows the periodicity of the sine wave. If we sample even less frequently, let us say every 1.5 ms (Figure 11.2C), we no longer adequately resolve the period of this sine wave. We now see a sine wave with a longer period (i.e., a lower frequency) than our analog 1,000-Hz sine wave. Digitally sampling an analog signal at too large a sampling period (or too low a sampling frequency) results in an inaccuracy in the estimate of the frequency of the analog signal. This inaccurate (false) frequency is referred to as an aliased frequency. The process leading to this false or inaccurate frequency is called aliasing. The Nyquist theorem can be used to avoid aliasing. According to the Nyquist theorem, to avoid aliasing, we must have a sampling frequency that is greater than twice the highest frequency in the analog signal that we are digitizing. The lowest digitization (sampling) frequency that can be used to accurately represent the frequency of a given analog signal is called the Nyquist rate. In our example, from Figure 11.2, we must sample at just above 2,000 Hz to adequately represent our 1,000-Hz sine wave. Figure 11.2D shows what might happen if we digitize at exactly 2,000 Hz (i.e., at exactly twice the frequency of the analog signal). In this case, when the sampling occurs at or near the peaks of the sine wave, we can resolve the frequency of the sine

wave. Although this sine wave looks more like a triangular wave, the frequency does appear appropriate. Figure 11.2E shows the same 2,000-Hz sampling frequency, but now the sampling times are slightly shifted in time from that shown in Figure 11.2D. You can see that the frequency of the 1,000-Hz sine wave is accurate, but the peak amplitude is underestimated. Figures 11.2D and 11.2E show that sampling at exactly twice the frequency of the analog signal can adequately resolve the frequency of interest. Why, then, does the Nyquist theorem state that the sampling frequency must be just above twice the highest frequency in the analog signal? This is answered by referring to Figure 11.2F, where the 1,000-Hz sine wave is digitally sampled at 2,000 Hz, but now these sample times occur at the zero crossings, and not at or near the peaks. This produces a flat line (i.e., a 0-Hz signal). As this 0-Hz frequency is clearly different (lower) than the analog signal frequency of 1,000 Hz, this example demonstrates aliasing. To avoid aliasing, we must sample at a little more than twice the highest frequency in our signal. It is wise to sample at two times the Nyquist rate (i.e., four times the highest frequency in the analog signal of interest), just to err on the side of caution. Using an example relevant to AEP work, if we know our ABR has substantial energy from 100 to 3,000 Hz, then the Nyquist rate is just above 6,000 Hz, and this sampling rate will be adequate to prevent aliasing. If we double the Nyquist rate, then the desired sampling frequency is ~12,000 Hz.

Now we know that using a sampling frequency that is too low can result in aliasing. Are there any problems with using a very high sampling frequency? Using a sampling frequency that is well above that needed to prevent aliasing will result in larger data files. This is most easily understood by a numerical example. We want to obtain an ABR for an infant, and we know that we should look at a response time window (time epoch) of 20 ms. Assuming there is little energy at 1,500 Hz or above in an infant ABR. In this example, the Nyquist rate is just above 3,000 Hz, and if we double this to prevent aliasing, we should digitize at ~6,000 Hz (a sampling period of 167 μs). If we sample every 167 μs, then we will have 120 data points in our 20-ms time window:

$$\text{Response time epoch} = \{\text{sampling period}\} \times \{\text{number of data points}\}.$$

This can be rewritten as

$$\text{Number of data points} = \{\text{response time epoch}\}/\{\text{sampling period}\}$$

Plugging our numbers from the example into this equation yields

$$\text{Number of data points} = 20\ \text{ms}/167\ \mu\text{s} = 120\ \text{data points}$$

We would only need to store 120 data points for this ABR waveform. If we doubled the sampling frequency (i.e., four times the Nyquist rate) this would require a sampling period of 83.5 μs, and a grand total of 240 data points in our memory array. Now let us sample as fast as we can. We construct a digitizer that can sample at 1 GHz (a billion samples per second). A 1 GHz sampling frequency is a sampling period of 1 ns, or 0.001 μs. The number of points in our evoked potential memory array would be 20 ms = 20,000 μs/0.0001 μs = 20,000,000 data points. You would fill up a 10 GB hard disk after collecting roughly 500 ABRs. If you had a busy clinic, you could fill up the hard drive quite quickly. You should digitize at a high enough rate to safely avoid aliasing, but not so fast as to have issues with data storage. Most commercial AEP units limit the number of data points that can be used to obtain a response. Let us say that your memory array is limited to 1024 (2^{10}) words of memory. For a 20-ms time window, your sampling period is 20 ms/1,024 = 20 μs, or a sampling frequency of 50,000 Hz. In this example, aliasing is not a problem, unless you suspect that there is energy in the response that exceeds 50,000/2 = 25,000 Hz. In some instances, you will not know exactly the Nyquist frequency of the response you are measuring. In these cases, using a filter that eliminates energy at and above a frequency where aliasing could be an issue is warranted. In our example, using a filter that eliminates energy at and above 25,000 Hz would solve the problem. This type of filter is called a low-pass filter. When the purpose of a low pass is to prevent aliasing, then this low-pass filter is called an antialiasing filter.

Digital-to-Analog Conversion

In most (perhaps all) current commercially available AEP systems, the acoustic stimuli used to elicit an evoked potential are digitally generated by the computer. These digital signals are converted to analog signals by a device called a digital-to-analog converter (DAC). Aliasing can also arise with digital-to-analog conversion. To avoid aliasing, the output voltage of the DAC can be low-pass filtered at the appropriate frequency (i.e., less than half the digitization frequency) by a device called an anti-imaging filter. It should be noted that the earphones used for clinical AEP measures generally have low-pass cutoffs less than 10,000 Hz and that these transducers may serve as anti-imaging filters if DAC rates above 20 kHz are used.

Noise Reduction

For human recordings, AEP amplitude is typically much smaller than that of the background noise. Any unwanted electrical activity will be called noise. For AEP purposes, this noise can be composed of both periodic and aperiodic activity and can be of both biologic and nonbiologic origin. Biologic sources of noise include muscle activity and the

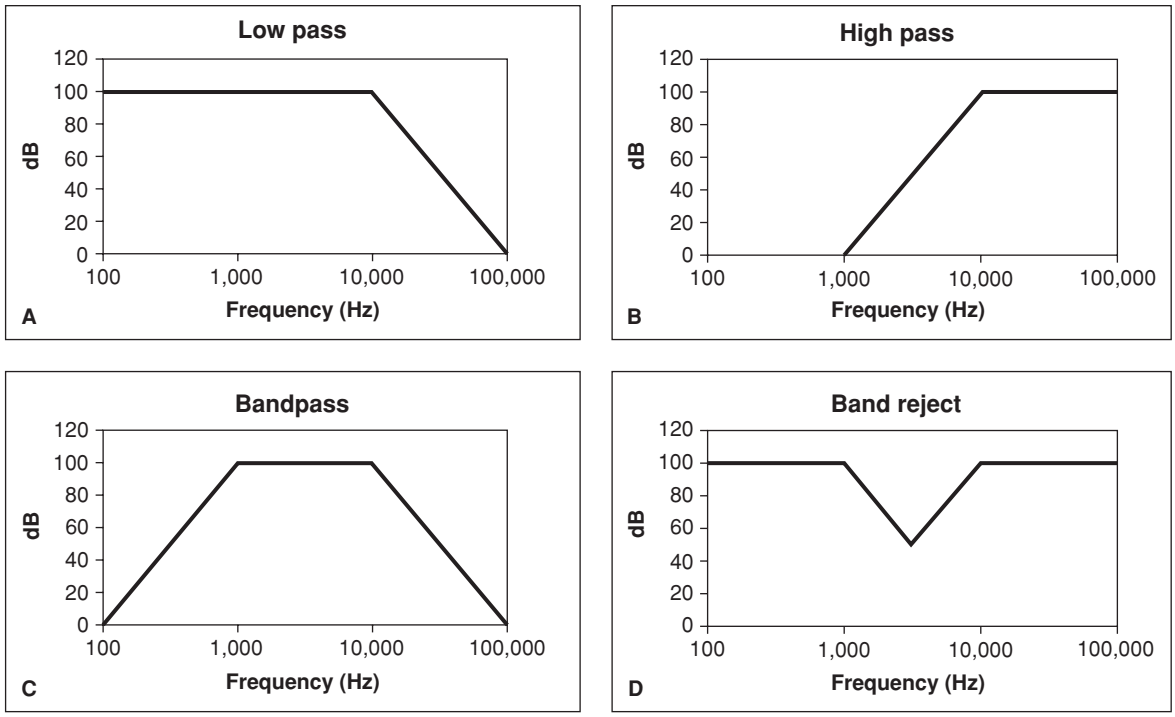


FIGURE 11.3 Filter response types are schematized: Low pass [**A**], high pass [**B**], bandpass [**C**], and band reject [**D**]. [Reprinted from Burkard R, Secor C. [2002] Overview of auditory evoked potentials. In: Katz J, ed. *Handbook of Clinical Audiology*. 5th ed. Baltimore, MD: Lippincott Williams & Wilkins; pp 233–248, with the permission of Lippincott Williams & Wilkins.]

EEG, both of which represent aperiodic noise. Nonbiologic sources of noise include aperiodic noise arising from the bioamplifier and periodic (60 Hz, or 50 Hz for our European and other readers) line-power noise. The 60-Hz (or 50-Hz) noise may arise from within the AEP system itself, but often this noise arises from the room where the AEP is being performed. This may be generated by overhead lights or be emitted by other electrical equipment in the room. Such 60-Hz (or 50-Hz) electrical noise is generated in the room, and the subject acts like an antenna. If this line noise is picked up by the electrodes on the subject's scalp, it is amplified (with the biologic activity). A few microvolts of line noise become a few volts, if a gain of 1,000,000 is used by the bioamplifiers.

How do you reduce unwanted electrical noise? First, low electrode impedances can reduce 60-Hz (or 50-Hz) line noise. Second, making the subject comfortable and encouraging them to sleep (if that does not negatively affect the response of interest) can substantially reduce noise arising from muscle activity. Third, differential amplification (i.e., using three electrode leads per recording channel) can reduce common-mode noise. Fourth, filtering the output of the bioamplifier can reduce noise. Finally, signal averaging reduces unwanted background noise. We have previously discussed electrode application and the use of differential amplification. In the following paragraphs we will provide more detail on the use of filtering and signal averaging to reduce unwanted noise.

Filtering involves eliminating noise that is outside the frequency range of the desired response. Most of the energy in, for example, the click-evoked ABR, is in the 30 to 3,000 Hz frequency range. Selectively eliminating electrical activity below 30 Hz and above 3,000 Hz will reduce the background noise with relatively minor changes to the ABR. Figure 11.3 shows the four basic types of filter functions. A low-pass filter (Figure 11.3A) reduces a signal above a given frequency, but allows lower frequency energy to pass through. A high-pass filter (Figure 11.3B) reduces a signal below a specified frequency, but passes signals above that frequency. A band-pass filter (Figure 11.3C) passes energy between two cutoff frequencies, but reduces energy above and below this frequency band. Finally, a band-reject or notch filter (Figure 11.3D) reduces energy between two cutoff frequencies, but passes energy above and below this band. The cutoff frequency (or half-power point) of a filter is the frequency at which the voltage at the filter output is reduced to 70.7% of the input (or is −3 dB). The rejection rate, or filter skirt, of a filter refers to how fast the voltage is reduced outside of the passband. This is often reported in decibels per octave. If the voltage of a signal is reduced by half (−6 dB) when the frequency is doubled (one octave is a doubling of frequency), then the filter rolls off at a rate of 6 dB/octave. Many filters roll off in integer multiples of 6 dB/octave, and each of these 6 dB/octave multiples is referred to as a pole. Thus, a 48 dB/octave filter is called an eight-pole filter.

It is important that you know the spectrum (frequency content) of the AEP you are recording, so that when you filter, you do not throw the baby out with the bathwater (i.e., you greatly reduce the AEP while reducing the amplitude of the noise). As most review chapters on the various AEPs will provide suggested filter settings for recording that response, determining optimal AEP filter settings should be a simple matter of following the suggestions in these chapters.

Despite our best efforts with getting the subject to relax, obtaining low and balanced electrode impedances, and using differential amplification with judicious filtering, the AEP of interest will still usually be smaller than the ongoing background electrical noise. In others words, the AEP is at a poor signal to noise ratio (SNR). In many instances, the response is much smaller than the amplitude of the background noise, making it difficult or impossible to identify the AEP. To improve the SNR (to make the AEP visible), we use time-domain signal averaging. The theory underlying signal averaging invokes several assumptions about the signal (i.e., the AEP) and the noise (see Hyde, 1994 for a more detailed treatment of this issue):

1. The AEP is always the same in response to a constant stimulus.
2. The noise is truly random (i.e., it is constantly changing).
3. The signal (the AEP) and the noise are independent.

For time-domain signal averaging, we present the same stimulus repeatedly while summing the response in memory. The average response is created by dividing the summed voltage at each time point in the memory array by the number of stimuli presented. Differences in the statistical properties of the signal (the AEP) and the noise lead to improvement in the SNR. For signal averaging to work correctly, we must initiate the summation into the memory array at a constant time relative to stimulus presentation. We typically start signal averaging at the onset of the stimulus. If we do this, then, according to the first assumption above, the AEP is constant to each stimulus presentation. For example, let us say we have a 1-μV AEP that we sum over 16 stimulus presentations:

$$1+1+1+1+1+1+1+1+1+1+1+1+1+1+1+1=16\,\mu\text{V}.$$

The average amplitude is thus 16 μV/16 sweeps = 1 μV. This is expected, as we said the stimulus is constant for each presentation. Let us say the background noise is 4 μV. The SNR = 1 μV/4 μV = 0.25. The signal is small compared to the noise, and it will be difficult to see the AEP. Let us sum together the response to 16 "noise" time epochs. In this case, because the noise segments are random, we sum these noise estimates in a manner that differs from that used for the signal. We square each value, sum these squared values, and take the square root. The interested reader is referred to Hyde (1994) for a more detailed explanation. According to this formula

$$(4^2+4^2+4^2+4^2+4^2+4^2+4^2+4^2+4^2+4^2+4^2+4^2+4^2+4^2+4^2+4^2)^{1/2}=16\,\mu\text{V}.$$

The average noise is now 16 μV/16 sweeps = 1 μV, and the SNR is now 1 μV signal/ 1 μV noise = 1. Thus, signal averaging over 16 sweeps changed the SNR from 0.25 to 1: A factor of 4 improvement. Under optimal conditions, the SNR increases in proportion to the square root of the number of sweeps.

Recommendations for the number of sweeps used clinically (in chapters that follow, and elsewhere) are based on the experimental and clinical experience of the investigators, or after review of the relevant literature. These recommendations can only be considered guidelines. The number of stimulus presentations required to obtain a given response depends on response amplitude (which varies with the AEP, stimulation and recording parameters, and the particular subject), background noise amplitude (which is dependent on subject and recording factors), and the target SNR (which is dependent on particular clinical objectives). For threshold estimation, the SNR should be on the order of 1 or more, as you are only interested in whether a response is present or not. For determination of peak latencies and amplitudes, an SNR of 2 or greater is desirable, as you are trying to accurately estimate response variables, which will be influenced by poor SNR.

You might read in the literature that you should obtain 2,000 sweeps for an ABR. Such statements, however, can only be considered suggestions, that will not be optimal under some circumstances. For one subject, to a lower level click stimulus, you might record a response amplitude of 0.1 μV. Let us say there is a 10 μV background noise. The SNR is 0.01, and this only improves by a factor of 44.7 for 2,000 sweeps, and the SNR of the averaged response will still be less than 1 (0.447). In this example, the recommended number of sweeps is inadequate. In a different subject, when you present a high-level stimulus, your response might be 0.6 μV. This patient is quite relaxed and has 3 μV of noise, resulting in an SNR of 0.6/3 = 0.2. Presenting 2,000 sweeps, the SNR (under optimal conditions) is 8.94 (0.2 × 44.7), which is a better SNR than is required for most clinical purposes. You could have saved yourself some time by stopping data acquisition before the 2,000 stimuli had been presented. Although guidelines of sweep numbers are of course useful, a clear understanding of the principles underlying signal averaging can lead to online protocol changes that will allow more efficient data collection, as well as reduce the likelihood of stopping signal averaging before an interpretable AEP is obtained. There are techniques that allow online estimation of SNR, and stopping rules can be used that stop data acquisition when a criterion SNR (and/or probability level that a response is

present) is achieved or a maximum number of responses have been averaged. One such technique (called Fsp) is described later in this chapter.

NORMATIVE ASPECTS OF THE ABR

In this section we will review some normative aspects that form the basis for the clinical applications of the ABR that are reviewed in Chapters 13, 14, and 16. We will review the recommended recording parameters of the ABR, briefly mention several relevant subject variables, and in more detail review the effects of several stimulus variables on the ABR.

As shown in Figure 11.1, the ABR from a normal-hearing and neurologically intact young adult to a moderately high level click stimulus results in a series of five to seven positive peaks. The peak amplitudes are, in most instances, less than 1 μV.

Subject Variables

The ABR is not substantially influenced by attention or sleep state (Kuk and Abbas, 1989; Picton and Hillyard, 1974), which makes it optimal for evaluating patients who are unable or unwilling to cooperate, such as infants or young children.

There are several subject variables that can affect the latency and the amplitude of the ABR, including core temperature, gender, and age. A decrease in core temperature results in an increase in ABR peak latencies, an increase in interwave intervals (IWIs), and a decrease in peak amplitudes (Hall et al., 1988; Marshall and Donchin, 1981). It is important to monitor core temperature during intraoperative monitoring. This is because a patient's core temperature may be outside of the normal range during, for example, heart surgery (Stockard et al., 1978), and core temperature changes can have an influence on the interpretation of the results. Females generally show shorter response latencies and IWIs, as well as larger response amplitudes, than males (Kjaer, 1979; Schwartz et al., 1994). These sex differences cannot be explained by differences in head size alone, but rather appear to be attributable, at least in part, to differences in cochlear length. Human females have shorter cochleae than their male counterparts (Sato et al., 1991). Don et al. (1993), using the high-pass subtractive masking technique (see below), reported that shorter cochlear length results in greater traveling-wave velocity, which is believed to account for some of the gender-related differences noted above. Finally, in regard to subject age, ABR peak latencies, IWIs, and amplitudes have been shown to vary with the age of a subject. Infant peak latencies and IWIs are longer than those seen in adults (Cox, 1985). Older adult subjects have ABRs that are typically reported to be longer in latency and smaller in amplitude, as compared to younger adult subjects (Schwartz et al., 1994). However, many of these older subjects have a hearing loss, which makes it difficult to separate the changes in the ABR that occur as a result of advancing age from the changes that can occur from a hearing loss.

Another issue that is not yet fully understood is how age affects the ABR IWIs. Rowe (1978) reported that the I–III IWI increased with increasing age, whereas the III–V IWI remained unchanged. In contrast, Costa et al. (1990) found that the I–II and I–III IWIs actually decreased with increasing age. These inconsistencies can make it challenging to interpret ABR results, especially when considering peak latencies, as well as IWIs, which are often used for site-of-lesion testing. Perhaps the evaluation of the I–V interval using derived-band responses (see Eggermont and Don, 1986) could be profitably combined to shed further light on the complicated and inconsistent effects of aging on the ABR IWIs.

Stimulus Variables

As mentioned above, the ABR is sensitive to manipulations in stimulus parameters, including stimulus polarity, whether the stimuli are presented monaurally or binaurally, stimulus spectrum, level, and rate. We will review the effects of these stimulus factors on the ABR in the paragraphs that follow.

A number of studies have investigated how click polarity affects the human ABR. Hair cells are only excited by a deflection of the stereocilia in the direction of the basal body (i.e., the hair cells perform a half-wave rectification of the input signal). Simple models of the auditory system suggest that the rarefaction phase of a stimulus should be the most effective (see Hall, 1995, pp 143–144). Empirically, some studies have shown shorter peak latencies and larger peak amplitudes to rarefaction than to condensation clicks (e.g., Stockard et al., 1979). However, other studies indicated that not all subjects show these trends and that polarity effects are, at best, small and variable (Borg and Lofqvist, 1981; Schwartz et al., 1994). Don et al. (1996) provide an in-depth discussion of the challenges in determining whether there are truly latency changes with changes in stimulus polarity. ABRs to monaural stimuli are smaller than to binaural stimuli (Owen and Burkard, 1991). If an ABR is collected in response to monaural stimulation from each ear and summed, this "summed monaural" response typically has a larger Wave V amplitude than is seen when one actually binaurally stimulates the subject (Owen and Burkard, 1991). This amplitude reduction for the true binaural response, as compared to that of the "summed monaural" response, is often interpreted as evidence of binaural interaction in the ABR. Most studies of binaural interaction using the difference between the sum of the monaural responses and the binaural response ignore the possibility that there are neural elements that (as shown in animal work) can be driven by either ear. These neural elements, therefore, contribute

twice in the monaural sum. However, its doubled contribution is not considered in response to binaural stimulation. Thus, even with no binaural interaction, there will always be a difference between the sum of the monaural responses and the binaural response suggesting binaural interaction. This potential confound makes it difficult to use the magnitude of the difference as a quantitative measure of the amount of binaural interaction.

Click stimuli are broad in frequency, which results in stimulation of much of the cochlea. To stimulate a limited region of the cochlea, you must either use a narrow-spectrum stimulus (such as a toneburst), use a masking procedure, or use a combination of a narrow-spectrum stimulus and maskers. Low-frequency tonebursts will typically produce longer ABR peak latencies, because of the increased traveling-wave delay to more apical cochlear regions. When presented at high stimulus levels, low-frequency tonebursts may actually generate an ABR that arises from the higher frequency (more basal) regions of the cochlea (Burkard and Hecox, 1983b). To reduce this basal spread of activity in response to low-frequency stimuli, several masking procedures have been developed and investigated. These masking procedures include (1) click stimuli with notched noise (Pratt and Bleich, 1982); (2) click stimuli and high-pass subtractive masking (Don and Eggermont, 1978; Teas et al., 1962); (3) toneburst stimuli in high-pass noise (Kileny, 1981); and (4) toneburst stimuli in notched noise (Picton et al., 1979). We will not review this literature further in this chapter. An excellent review of this topic can be found in Stapells et al. (1994).

To estimate ABR threshold, the level of the stimulus must be varied. ABR peak latencies increase whereas peak amplitudes decrease with decreasing click levels. Waves I, II, and IV are often difficult to identify at moderate click levels and below. Wave V is often the only wave that can be identified near threshold, although in some cases Wave III can also be seen at and near ABR threshold. The slope of the Wave V latency/intensity function (i.e., the change in Wave V latency for a given change in stimulus level) to click stimuli is typically near −40 μs/dB in normal-hearing young adults (Burkard and Hecox, 1983a; Hecox and Galambos, 1974).

Click rate also influences the ABR. As the click rate increases, peak latencies and IWIs increase, whereas peak amplitudes decrease (Burkard and Hecox, 1987). It should be noted that Wave V amplitude does not always decrease linearly with increasing rate. In normal-hearing young adults, Burkard and Hecox (1983a) showed a small decrease in Wave V amplitude with increasing rate from 15 to 30 Hz, and little amplitude change from 30 to 90 Hz. Burkard et al. (1990) used 50 dB nHL clicks in a group of normal-hearing young adults. They reported a mean Wave V amplitude increase with increasing rate from 30 Hz (0.388 μV) to 90 Hz (0.409 μV). These results demonstrate that Wave V amplitude to moderate-level clicks is only modestly affected by increasing rate. As Wave V is the wave most often seen at near-threshold levels, using a relatively fast stimulus repetition rate (30 to 50 Hz) is optimal for threshold estimation, as this will reduce the amount of time it takes to obtain an ABR.

NOVEL STIMULUS MANIPULATIONS FOR CLINICAL APPLICATIONS OF THE ABR

The cochlear delay line, that is, the increased response latency with decreasing stimulus frequency, leads to an ABR whose peak latencies and amplitudes are dominated by the activity of the higher frequency portions of the cochlea. In this section, we discuss how several creative stimulus manipulations are used in the attempt to account for this delay. We will also discuss their possible clinical utility. Consider this brief inclusion of several stimulus manipulations that have possible clinical applications as a thought-provoking prelude to subsequent chapters that describe the clinical utility of the ABR (i.e., Chapters 13, 14, and 16).

High-pass Subtractive Masking and the Stacked ABR

Why Standard ABR Measures Cannot Detect Small Tumors

Two requirements for any ABR measure used for tumor detection are that (a) the tumor exerts sufficient pressure to desynchronize, block, or alter the conduction properties of eighth nerve elements and (b) the tumor affects a sufficient number of those neural elements. Obviously, any ABR measure will fail if either of these two requirements is not met. However, for standard ABR latency measures such as the I–V IWI and the interaural Wave V (IT5) delays, there is an additional, third requirement: The tumor must affect the activity of those neural elements that determine the peak latency of the brainstem response to the stimulus. In other words, normal standard ABR latency measures are determined by only a subset of auditory nerve fibers. In particular, the latency of the standard ABR is determined by the high-frequency fibers. The high failure rate in detecting small intracanalicular (in the internal auditory meatus) tumors is not surprising because normal standard ABR latencies are possible if the synchronous activity of the high-frequency fibers that determine the latency is not sufficiently compromised by the tumor. Thus, even if a small tumor affected a substantial number of activated neural elements representing mid-to-low frequencies, the peak latency may not change much because the activity of these elements does not determine the peak latency of the standard ABR. If standard ABR latency measures detect the tumor, then a sufficient number of the neural elements that determine the peak latency have been affected. If, however, the ABR latency measures miss the tumor, then an adequate number of the appropriate

neural elements were not affected. It is this possible failure to affect a sufficient number of appropriate neural elements that makes latency measures insensitive to some, but not all, small tumors. We hypothesize that the variable success of the standard ABR measures in detecting small tumors is due, in part, to the variable underlying neuroanatomic organization of eighth nerve fibers and the variable location and encroachment of small tumors.

If latency is often insensitive to the effects of small tumors, what about Wave V amplitude? Amplitude measures should be very sensitive to loss or desynchronization of eighth nerve activity. Many studies examined standard ABR amplitude measures and concluded that they are often too variable compared with latency measures. Two major contributors to this amplitude variability discussed above are (a) the residual noise in the average and (b) phase cancellation of activity related to progressive activation and response time variations across the cochlea. Standard ABR wave amplitude measures do not reflect all the neural activity from click stimulation because of phase cancellation. In particular, studies have shown that activity from low-frequency regions of the cochlea contributes little to the standard ABR Wave V amplitude (Don et al., 1994, 1997, 2005). Therefore, like standard latency measures, standard amplitude measures will miss tumors that do not sufficiently affect high-frequency fibers.

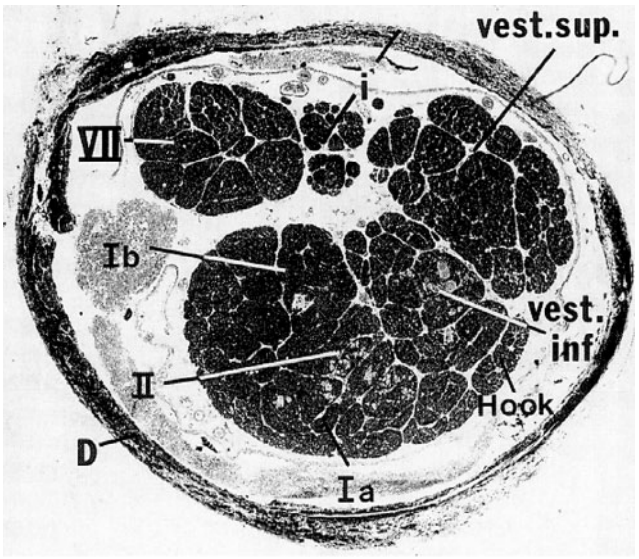


FIGURE 11.4 Tonotopic organization of the human auditory nerve. Transverse section through the internal auditory canal of an 8-year-old child, showing the position of the facial nerve (*VII*), the superior division of the vestibular nerve (*vest. sup.*), the inferior division of the vestibular nerve (*vest. inf.*), and the auditory nerve with the nerve fibers for the most basal end (*Hook*), for the lower basal turn (*Ia*), the upper basal turn (*Ib*), and the second and apical turns (*II*). (Reprinted from Spoendlin H, Schrott A. (1989) Analysis of human auditory nerve. *Hear Res*. 43, 25–38, with the permission of Elsevier Science.)

The Stacked ABR: A New Measure for Detecting Small Acoustic Tumors

If a new ABR measure is to be successful at detecting small acoustic tumors, it must avoid the main shortcomings described above. We hypothesize that to do so, it must be a measure of neural activity from all of the cochlea, not just the high-frequency regions. We have previously cited ABR studies demonstrating that high-frequency activity dominates the latency and amplitude responses, and we hypothesized that small tumors do not always affect these fibers. Is there neuroanatomic evidence to support this hypothesis?

Anatomic Considerations

Acoustic tumors generally arise from Schwann cells in the vestibular division of the eighth nerve in the internal auditory canal and eventually extend into the cerebellopontine angle. The tumors can arise from either the superior or inferior divisions of the vestibular nerve and encroach upon the cochlear nerve. To understand the effect of a small tumor on the cochlear nerve, we need to understand the tonotopic neuroanatomic organization of the fibers in the cochlear nerve. Figure 11.4 is adapted from Spoendlin and Schrott (1989). In this transverse section of the human internal auditory canal we see the seventh (facial) nerve (VII; upper left) and three divisions of the eighth (VIII) (auditory and vestibular) nerve. Clockwise from upper right, the divisions of the eighth nerve are as follows: First, the superior vestibular nerve; second, the inferior vestibular nerve; third, the auditory (cochlear) nerve. In the auditory nerve, high-frequency fibers arising from the lower and upper basal turns of the cochlea lie inferiorly (Ia) and superiorly (Ib), respectively. Fibers from the second and apical turns of the cochlea lie in the medial portion of the cochlear nerve (II), adjacent to the inferior vestibular nerve.

This figure clearly shows that if a tumor arose from that adjacent portion of the inferior vestibular nerve, it would affect the lower frequency fibers in the second and apical turns first. There are tumor patients with only low-frequency or upward-sloping hearing losses (Johnson, 1977). In some of those cases, it is possible that a tumor here might be partly responsible for such hearing losses. As can be seen in this figure, depending on where the vestibular schwannoma arises, high- or low-frequency fibers can be affected first. At the House Clinic in Los Angeles, approximately half the eighth nerve tumors originate from the inferior vestibular nerve, with the other half arising from the superior vestibular nerve (Dr. Fred Linthicum, personal communication). In addition, tumors do not always simply push against the nerve trunk, exerting pressure from the periphery of the trunk inward. Instead, there is strong evidence that the tumor often invades or infiltrates the nerve trunk. Studies have demonstrated that invasion of the cochlear nerve by solitary vestibular

schwannomas is common. Neely (1985) found invasion of the nerve trunk in all 22 cases he studied; Marquet et al. (1990) and Forton et al. (1990), in over 50% of their cases; and Dr. Fred Linthicum, in half of the cases in a series of 28 tumors from the House Clinic (personal communication). In particular, neurofibromatosis type II tumors typically infiltrate the nerve trunk. Therefore, fibers other than those on the surface of the trunk may be affected. Clearly, a measure of neural activity from all parts of the cochlea would be better in detecting small tumors than ABR measures confined to the high frequencies alone.

Description of the Stacked ABR

We mentioned above that amplitude measures should be able to reflect neural activity that has been desynchronized or blocked by a tumor. However, we presented several major problems with standard measures, in particular, the detrimental effect of varying SNR and the dependence of these measures on high-frequency activity because of phase cancellation of activity from lower frequency regions. Don et al. (1997, 2005) have proposed an ABR measure to circumvent these problems. This measure, the stacked ABR amplitude, is sensitive to neural fiber activity from all frequency regions of the cochlea. Thus, the measure will reflect the loss of synchronized neural fiber activity, no matter which fibers are compromised by a tumor, as long as the stimulus level is high enough to activate most of the neural fibers in all the frequency regions. Determining the stacked ABR amplitude requires the derived-band and stacked ABR techniques. A click stimulus is used to activate the whole cochlea, and the resulting response is separated into five frequency bands by using a high-pass masking technique with response subtraction (Don and Eggermont, 1978; Parker and Thornton, 1978a, 1978b). The ABRs representing these five frequency bands are called derived-band ABRs and are used in constructing the stacked ABR.

The derived-band ABR technique requires six stimulus conditions. These six stimulus conditions are noted in the first column on the far left of Figure 11.5 and from top

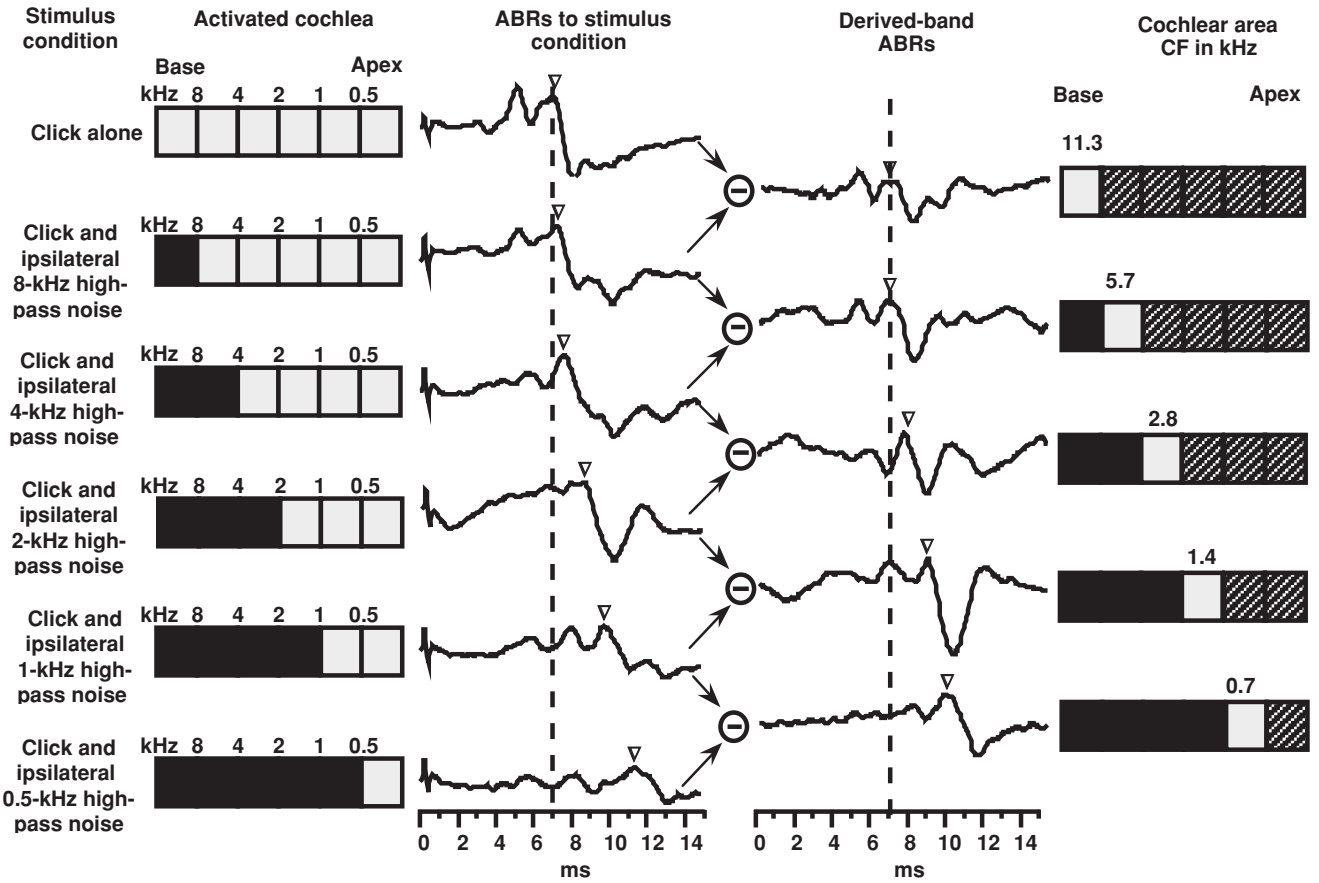


FIGURE 11.5 Schematic description of the high-pass masking and subtraction technique used to obtain derived-band ABRs. **First column:** The six stimulus conditions. **Second column:** Schematics of the areas of the cochlea stimulated for that condition. **Third column:** ABR waveforms to stimulus conditions. **Fourth column:** Derived-band ABR waveforms resulting from successive subtraction of conditions. **Fifth column:** Schematics of the octave-wide areas of the cochlea whose activity is represented in the derived-band ABRs. [See text for detailed explanation.]

to bottom are the presentation of clicks alone, followed by clicks presented with simultaneous ipsilateral high-pass masking pink noise with cutoff frequencies of 8, 4, 2, 1, and 0.5 kHz. A schematization of the cochlear regions activated (illustrated by the lightly shaded regions) by the unmasked and various high-pass masker cutoff conditions is shown in column 2. ABRs to the clicks without masking noise, and each of the five high-pass noise conditions, are shown in the third column from the left. Note the increase in ABR peak latency with decreasing high-pass masking noise cutoff frequency. If the ABR obtained from one high-pass masker cutoff condition is subtracted from the ABR obtained with the high-pass masker cutoff frequency that is one octave higher in frequency, you obtain the derived-band responses (shown in the fourth column from the left), which shows an increase in derived-band response latency with decreasing derived-band cutoff frequencies. For the high-pass responses, the ABR arises from all cochlear regions apical to the high-pass masker cutoff frequency. In contrast, the derived-band responses are thought to arise from the octave-wide cochlear region delimited by the two high-pass masker cutoff frequency conditions used to create the derived band (e.g., from the 4- to 8-kHz region when you subtract the 4-kHz high-pass condition from the 8-kHz high-pass condition; see Don et al. 2005). This octave-wide cochlear region for each derived band is illustrated by the lightly shaded regions in the rightmost column of Figure 11.5.

The theoretical center frequencies (CFs) of these derived bands are simply and arbitrarily defined as the geometric mean of the two cutoff frequencies of the stimulus conditions involved in the subtraction. Specifically, the theoretical CF for each derived band is computed as the square root of the product of the two successive high-pass filter cutoff frequencies used for the band. For example, the derived-band ABR resulting from subtracting the response to clicks + 4 kHz high-pass masking noise from the response to clicks + 8 kHz high-pass masking noise would have a theoretical CF of about 5.7 kHz ($\{4 \times 8\}^{1/2} \approx 5.7$). For the click-alone condition, 16 kHz is used for the calculations. Thus, the theoretical derived-band CFs for the five derived-band ABRs are 11.3, 5.7, 2.8, 1.4, and 0.7 kHz.

The delay in peak activation (ΔT) from different regions of the cochlea (Figure 11.5, fourth column from the left, labeled "Derived-band ABRs") demonstrates that the activity of the cochlea underlying the generation of the standard ABR is not synchronous in time, but is progressively delayed as more apical cochlear regions are activated. This clearly illustrates how activity from lower frequency regions is phase-canceled by activity from higher frequency regions. For example, the peak of Wave V in the 0.7-kHz derived-band ABR is in phase and canceled by the trough following the peak of Wave V in the 1.4-kHz derived-band ABR. Similar phase cancellation can be seen between other successive derived-band waveforms. As a result, the amplitude of the standard ABR to clicks alone does not reflect

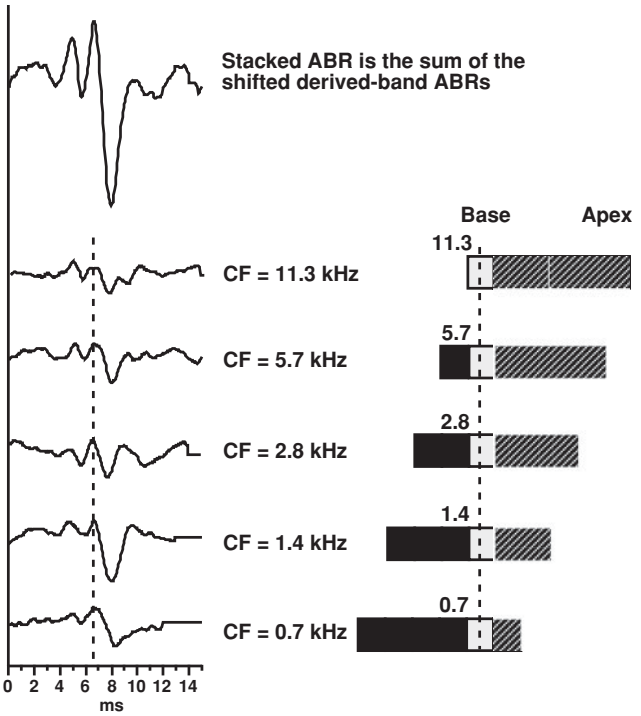


FIGURE 11.6 The stacked ABR is formed by shifting the derived bands to align the Wave V peaks, then adding the waveforms together. The Wave V amplitude of the stacked ABR is the new measure of interest.

the total amount of neural activation. Figure 11.6 illustrates the construction of the stacked ABR from the derived-band ABRs in Figure 11.5. The stacked ABR is constructed by (a) time shifting the derived-band waveforms so the peak latencies of Wave V in each derived band coincide and (b) adding together these shifted derived-band waveforms. The Wave V peaks of the derived-band ABRs are aligned to the Wave V peak latency for the arbitrarily selected 5.7-kHz derived band. The top waveform is the stacked ABR, the sum of the temporally aligned derived-band ABRs shown below it. By temporally aligning the peak activity initiated from each segment of the cochlea, we synchronize the total activity and minimize phase-canceling effects. Thus, compared with standard ABR amplitude measures, the amplitude of the stacked ABR Wave V reflects more directly the total amount of cochlear activity. We refer to this as the s*tacked ABR amplitude.*

Before reviewing some results with this measure, we must discuss some important technical aspects associated with the stacked ABR. ABRs have poor SNRs because responses originate from deep brainstem structures located at a significant distance from the surface-recording electrodes. Even after extensive averaging, ABRs are often still dominated by unaveraged residual background physiological noise. In the stacked ABR approach, it is imperative that the ABR waveforms reflect mostly neural response, not noise. Therefore, techniques for recording and processing

ABRs to ensure consistently low residual noise in the averages (Don and Elberling, 1996; Elberling and Don, 1984) are combined with the derived-band ABR method. In particular, the Fsp measure (Elberling and Don, 1984) is used to help estimate the SNR of an ABR. In addition, we apply a Bayesian weighting approach developed by Elberling and Wahlgreen (1985) to form averages that give more weight to sweeps having less noise and to minimize the destructive effects of large episodic background noise on the ABR. Thus, the stacked ABR method combines the derived-band ABR method with techniques that ensure low levels of residual noise and create weighted averages to minimize the destructive effects of episodic noise. Suggestions for obtaining high-quality derived-band/stacked ABR responses can be found in Don and Kwong (2013).

In an initial study of 25 tumor cases, Don et al. (1997) found that five small (≤1 cm) intracanalicular tumors were missed by standard ABR latency measures (IT5 and I–V delay). However, they demonstrated that all five were detected by this new stacked ABR method. In a larger follow-up study of 54 small tumor cases, Don et al. (2005) demonstrated that the Stacked ABR achieved 95% sensitivity and about 88% specificity with respect to young nontumor, normal-hearing subjects. Figure 11.7 is a plot from Don et al. (2005) showing the cumulative percentile curves for the stacked ABR amplitudes for nontumor, normal-hearing subjects (NTNH) and for the small acoustic tumor subjects (SAT). The stacked ABR amplitudes were normalized to the mean value of adults of the same gender tested under similar testing conditions. Don et al. (2005) established target criteria for excellent sensitivity (95%) and acceptable specificity (50%) and evaluated separately the sensitivity and specificity of these target criteria. It can be seen in Figure 11.7 that a target sensitivity of 95% yields a specificity of 88% relative to the NTNH population. Furthermore, the criterion value for a target specificity of 50% resulted in detection of all the tumor cases in their study (100% sensitivity). Thus, it appears that the stacked ABR measure can significantly reduce the number of nontumor patients sent for imaging, without missing a tumor.*

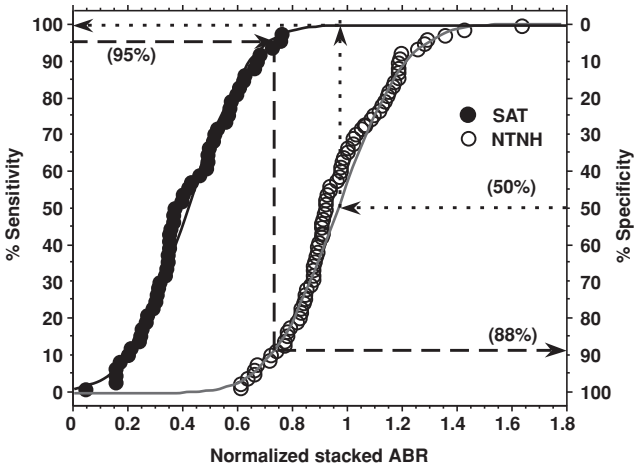


FIGURE 11.7 The cumulative distribution curves for the normalized stacked ABR amplitudes for both the NTNH and the SAT populations. A criterion to achieve 95% sensitivity yields 88% specificity and a criterion to achieve 50% specificity yields 100% sensitivity. [Reprinted from Don M, Kwong B, Tanaka C, Brackmann DE, Nelson RA. [2005] The Stacked ABR: A sensitive and specific screening tool for detecting small acoustic tumors. *Audiol Neurootol*. 10, 274–290, with permission of S. Karger AG, Basel.]

The Chirp

Our understanding of how the cochlea works goes back to the seminal (and Nobel prize-winning) work of Georg von Bekesy. From this work, we know that high frequencies stimulate the base of the cochlea, whereas low frequencies excite the apex of the cochlea. One consequence of this type of "cochleotopic" representation is that the high-frequency (basal) cochlear regions are stimulated first, with the more apical regions stimulated after a bit of a delay (take a peek at the derived-band response latencies shown in Figure 11.5, for a visual representation of this phenomenon). Dau et al. (2000) described an upward frequency sweeping chirp that was used to compensate for the temporal dispersion that occurs in the cochlea. This has come to be called the "Dau" chirp. It has now been reported that a single chirp is not adequate, as the optimal chirp (at least for normal-hearing young adults) gets shorter as the stimulus level increases (Elberling et al. 2010). Figure 11.8 (from Elberling et al., 2010) shows the electrical waveform of a family of chirps, as well as that of a click. Note that with increasing chirp number, its duration increases. Figure 11.9 (also from Elberling et al., 2010) shows grand mean ABR waveforms for the click and chirps, for three stimulus levels. It can be seen that at least for a few of the chirps the grand mean ABR Wave V amplitude appears to be larger than that for the click. Finally, in Figure 11.10 (again, from Elberling et al., 2010), the optimal chirp duration (i.e., that chirp duration which produces the largest Wave V amplitude) varies with stimulus level. Recall that the larger numbered chirps are longer in duration; it can be clearly seen here that the optimal chirp duration increases with decreasing chirp level. Increasing Wave V amplitude by using chirps is one good way of reducing test time for, for example, newborn hearing screening. However, most of the work using chirps has, to date, focused on normal-hearing young adults, and more work is needed to determine the optimal chirp duration across level for newborns and to identify the effects of hearing loss on chirp-evoked ABRs. Some preliminary data

*The stacked ABR was licensed to Bio-logic Corp (now owned by Natus Corp) and was implemented on their ABR system. However, Bio-logic/Natus no longer own the license nor sell or support the systems with the stacked ABR.

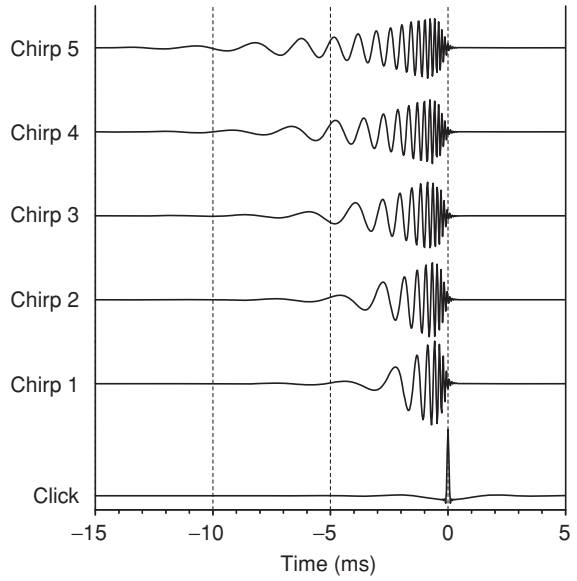


FIGURE 11.8 The electrical waveforms of five different chirp stimuli, as well as the click stimuli, used by Elberling et al. (2010). (Reprinted with permission from Elberling C, Callø J, Don M. (2010) Evaluating auditory brainstem responses to different chirp stimuli at three levels of stimulation. *J Acoust Soc Am*. 128, 215–223, copyright 2010 Acoustical Society of America.)

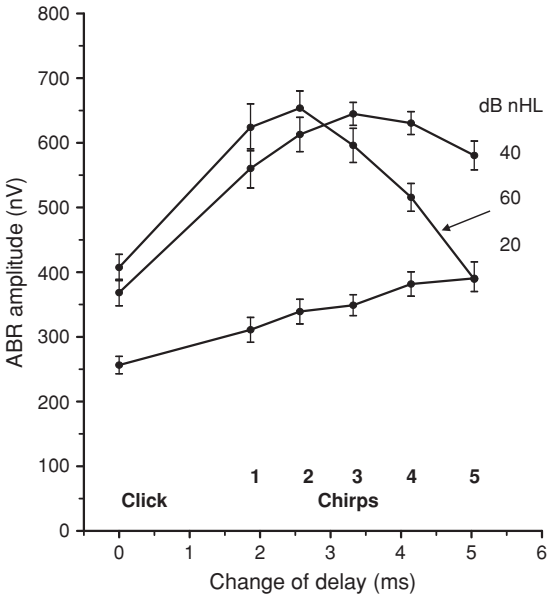


FIGURE 11.10 Mean ABR Wave V amplitude is plotted across stimulus type (five different chirp stimuli; click stimuli). The parameter is stimulus level. (Reprinted with permission from Elberling C, Callø J, Don M. (2010) Evaluating auditory brainstem responses to different chirp stimuli at three levels of stimulation. *J Acoust Soc Am*. 128, 215–223, Copyright 2010. Acoustical Society of America.)

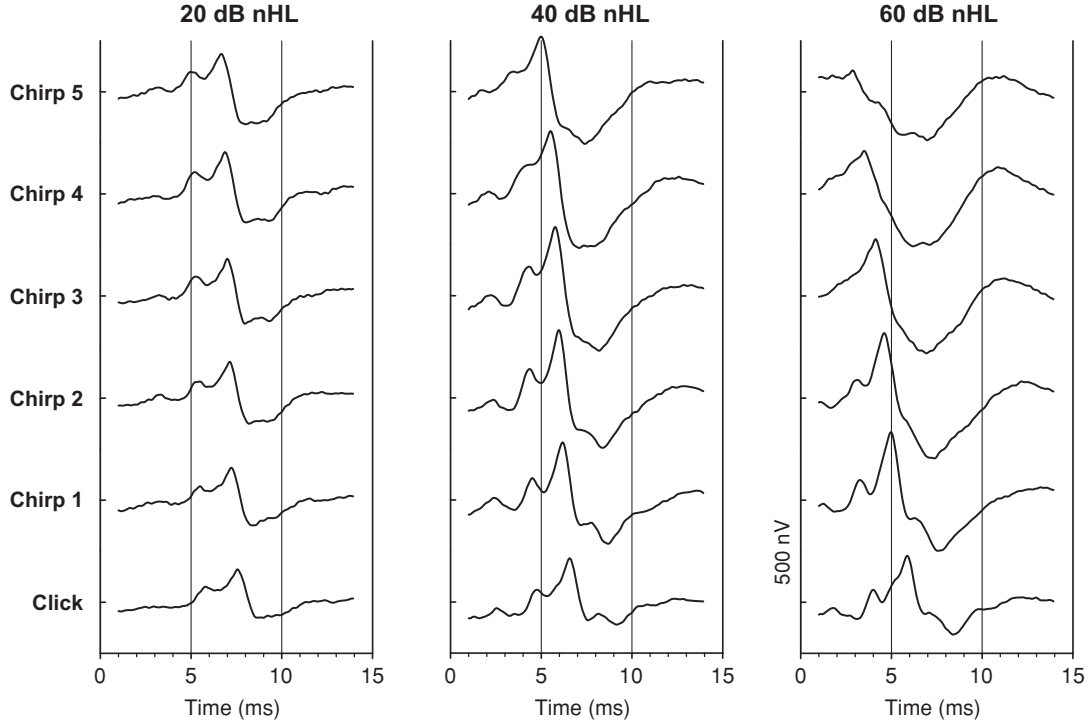


FIGURE 11.9 The grand average ABR waveforms to five different chirp stimuli, as well as the click stimuli, for the three stimulus level used by Elberling et al. (2010). (Reprinted with permission from Elberling C, Callø J, Don M. (2010) Evaluating auditory brainstem responses to different chirp stimuli at three levels of stimulation. *J Acoust Soc Am*. 128, 215–223, Copyright 2010. Acoustical Society of America.)

suggest that chirps elicit larger amplitude ABRs than clicks (at similar sensation levels) in human adults with sensory hearing loss (Maloff and Hood, 2014).

SUMMARY

In this chapter, we have briefly described the anatomy of the auditory nervous system, provided an overview of the various flavors of AEPs, presented a terse overview of the technical aspects of eliciting and recording an AEP, and summarized the effects of various stimulus manipulations on the ABR. We ended by describing some recent work involving high-pass subtractive masking (called the stacked ABR) and upward-frequency–sweeping FM tones (chirps). We hope that you will go out and do something important with your newfound knowledge.

FOOD FOR THOUGHT

1. Do you agree or disagree with the following statement: Students should not be forced to learn the technical details about AEP recording and stimulus generation, as audiologists are not supposed to be technicians.
2. Do you think an audiologist who is trained to do AEPs could expand their clinical practice to include somatosensory-evoked potentials, visual-evoked potentials, vestibular-evoked myogenic potentials, and facial nerve monitoring? In responding to this question, make sure you comment on the adequacy of your professional training for expanding their recording of EPs beyond AEP and describe what additional education and/or training would be required for you to feel comfortable recording (and interpreting) non-AEPs.
3. Do you think it is worth purchasing the equipment and learning about stacked ABRs, or should audiologists not record stacked ABR, as MRIs are quite good at identifying even intracanalicular tumors?

REFERENCES

Borg E, Lofqvist L. (1981) Brainstem response (ABR) to rarefaction and condensation clicks in normal hearing and steep high-frequency hearing loss. *Scand Audiol.* 13 (suppl), 99–101.

Buchwald J, Huang C. (1975) Far-field acoustic response: origins in the cat. *Science.* 189, 382–384.

Burkard R, Hecox K. (1983a) The effect of broadband noise on the human brainstem auditory evoked response. I. Rate and intensity effects. *J Acoust Soc Am.* 74, 1204–1213.

Burkard R, Hecox K. (1983b) The effect of broadband noise on the human brainstem auditory evoked response. II. Frequency specificity. *J Acoust Soc Am.* 74, 1214–1223.

Burkard R, Hecox K. (1987) The effect of broadband noise on the human brainstem auditory evoked response. III. Anatomic locus. *J Acoust Soc Am.* 81, 1050–1063.

Burkard R, Shi Y, Hecox K. (1990) A comparison of maximum length and Legendre sequences to derive BAERs at rapid rates of stimulation. *J Acoust Soc Am.* 87, 1656–1664.

Costa P, Benna P, Bianco C, Ferrero P, Bergamasco B. (1990) Aging effects on brainstem auditory evoked potentials. *Electromyogr Clin Neurophysiol.* 30, 495–500.

Cox L. (1985) Infant assessment: developmental and age-related considerations. In: Jacobson J, ed. *The Auditory Brainstem Response.* San Diego, CA: College-Hill Press; pp 297–316.

Dallos P. (1973) *The Auditory Periphery.* New York: Academic Press.

Dau T, Wegner O, Mellert V, Kollmeier B. (2000) Auditory brainstem responses with optimized chirp signals compensating basilar-membrane dispersion. *J Acoust Soc Am.* 107, 1530–1540.

Davis H. (1976) Principles of electric response audiometry. *Ann Otol Rhinol Laryngol.* 25 (suppl 28), 1–96.

Don M, Eggermont J. (1978) Analysis of the click-evoked brainstem potentials in man using high-pass noise masking. *J Acoust Soc Am.* 63, 1084–1092.

Don M, Elberling C. (1996) Use of quantitative measures of ABR peak amplitude and residual background noise in the decision to stop averaging. *J Acoust Soc Am.* 99, 491–499.

Don M, Kwong B. (2013) The stacked ABR revisited. *ENT Audiol.* 22, 100–103.

Don M, Kwong B, Tanaka C, Brackmann DE, Nelson RA. (2005) The stacked ABR: a sensitive and specific screening tool for detecting small acoustic tumors. *Audiol Neurootol.* 10, 274–290.

Don M, Masuda A, Nelson RA, Brackmann DE. (1997) Successful detection of small acoustic tumors using the stacked derived-band ABR method. *Am J Otol.* 18, 608–621.

Don M, Ponton CW, Eggermont JJ, Masuda A. (1993) Gender differences in cochlear response time: an explanation for gender amplitude differences in the unmasked auditory brain-stem response. *J Acoust Soc Am.* 94, 2135–2148.

Don M, Ponton CW, Eggermont JJ, Masuda A. (1994) Auditory brainstem response (ABR) peak amplitude variability reflects individual differences in cochlear response times. *J Acoust Soc Am.* 96, 3476–3491.

Don M, Vermiglio A, Ponton C, Eggermont J. (1996) Variable effects of click polarity on auditory brain-stem response latencies: analysis of narrow-band ABRs suggest possible explanations. *J Acoust Soc Am.* 100, 458–466.

Eggermont JJ, Don M. (1986) Mechanisms of central conduction time prolongation in brainstem auditory evoked potentials. *Arch Neurol.* 43, 116–120.

Elberling C, Callø J, Don M. (2010) Evaluating auditory brainstem responses to different chirp stimuli at three levels of stimulation. *J Acoust Soc Am.* 128, 215–223.

Elberling C, Don M. (1984) Quality estimation of averaged auditory brainstem responses. *Scand Audiol.* 13, 187–197.

Elberling C, Wahlgreen D. (1985) Estimation of auditory brainstem responses, ABR, by means of Bayesian reference. *Scand Audiol.* 14, 89–96.

Forton G, Offeciers FE, Marquet J. (1990) Het acousticusneurinoma: een histopathologische studie [Acoustic neuroma: a histopathological study]. *Acta Otorhinolaryngol Belg.* 44, 399–402.

Hall J. (1995) *Handbook of Auditory Evoked Responses.* Boston, MA: Allyn and Bacon.

Hall J, Bull J, Cronau L. (1988) The effect of hypo- versus hyperthermia on auditory brainstem response: two case reports. *Ear Hear.* 9, 137–143.

Hecox K, Galambos R. (1974) Brainstem auditory responses in human infants and adults. *Arch Otolaryngol.* 99, 30–33.

Hood L. (1998) Auditory neuropathy: what is it and what can we do about it? *Hear J.* 51 (8), 10–18.

Hyde M. (1994) Signal processing and analysis. In: Jacobson J, ed. *Principles & Applications in Auditory Evoked Potentials.* Boston, MA: Allyn and Bacon; pp 47–83.

Jasper H. (1958) The ten-twenty electrode system of the International Federation. The International Federation of Clinical Neurophysiology. *Electroencephalogr Clin Neurophysiol.* 10, 371–375.

Jewett D, Williston J. (1971) Auditory evoked far fields averaged from the scalp of humans. *Brain.* 4, 681–696.

Johnson EW. (1977) Auditory test results in 500 cases of acoustic neuroma. *Arch Otolaryngol.* 103, 152–158.

Kileny P. (1981) The frequency specificity of tone-pip evoked auditory brainstem responses. *Ear Hear.* 2, 270–275.

Kjaer M. (1979) Differences of latencies and amplitudes of brain stem evoked potentials in subgroups of a normal material. *Acta Neurol Scand.* 59, 72–79.

Kraus N, McGee T. (1994) Auditory event-related potentials. In: Katz J, ed. *The Handbook of Clinical Audiology.* 4th ed. Baltimore, MD: Williams & Wilkins; pp 406–423.

Kraus N, McGee T, Stein L. (1994) The auditory middle latency response. Clinical uses, development, and generating system. In: Jacobson J, ed. *Principles & Applications in Auditory Evoked Potentials.* Boston, MA: Allyn and Bacon; pp 155–178.

Kuk F, Abbas P. (1989) Effects of attention on the auditory evoked potentials recorded from the vertex (ABR) and the promontory (CAP) of human listeners. *Neuropsychologia.* 27, 665–673.

Maloff E, Hood LJ. (2014) Auditory brainstem responses elicited by chirp stimuli in adults with normal hearing and sensorineural hearing loss. *Ear Hear.* 35, 271–282.

Marquet JF, Forton GE, Offeciers FE, Moeneclaey LL. (1990) The solitary schwannoma of the eighth cranial nerve. An immunohistochemical study of the cochlear nerve-tumor interface. *Arch Otolaryngol Head Neck Surg.* 116, 1023–1025.

Marshall N, Donchin E. (1981) Circadian variation in the latency of brainstem responses and its relation to body temperature. *Science.* 212, 356–358.

Moller A. (1994) Neural generators of auditory evoked potentials. In: Jacobson J, ed. *Principles & Applications in Auditory Evoked Potentials.* Boston, MA: Allyn and Bacon; pp 23–46.

Neely JG. (1985) Hearing conservation surgery for acoustic tumors: a clinical-pathologic correlative study. *Am J Otol.* (suppl), 143–146.

Owen G, Burkard R. (1991) The effects of ipsilateral, contralateral and binaural broadband noise on the human BAER to click stimuli. *J Acoust Soc Am.* 89, 1760–1767.

Parker DJ, Thornton ARD. (1978a) Frequency specific components of the cochlear nerve and brainstem evoked responses of the human auditory system. *Scand Audiol.* 7, 53–60.

Parker DJ, Thornton ARD. (1978b) The validity of the derived cochlear nerve and brainstem evoked responses of the human auditory system. *Scand Audiol.* 7, 45–52.

Picton T, Hillyard S. (1974) Human auditory evoked potentials. II. Effects of attention. *Electroencephalogr Clin Neurophysiol.* 36, 191–199.

Picton T, Ouellette J, Hamel G, Smith A. (1979) Brainstem evoked potentials to tonepips in notched noise. *J Otolaryngol.* 8, 289–314.

Popper A, Fay R. (1992) *The Mammalian Auditory Pathway: Neurophysiology.* New York, NY: Springer-Verlag.

Pratt H, Bleich N. (1982) Auditory brain stem potentials evoked by clicks in notched noise. *Electroencephalogr Clin Neurophysiol.* 53, 417–426.

Rowe J. (1978) Normal variability of the brain-stem auditory evoked response in young and old adult subjects. *Electroencephalogr Clin Neurophysiol.* 44, 459–470.

Sato H, Sando I, Takahashi H. (1991) Sexual dimorphism and development of the human cochlea. Computer 3-D measurement. *Acta Otolaryngol. (Stockh.)* 111, 1037–1040.

Schwartz D, Morris M, Jacobson J. (1994) The normal auditory brainstem response and its variants. In: Jacobson J, ed. *Principles & Applications in Auditory Evoked Potentials.* Boston, MA: Allyn and Bacon; pp 123–153.

Speaks C. (1996) *Introduction to Sound.* 2nd ed. San Diego, CA: Singular.

Spoendlin H. (1972) Innervation densities of the cochlea. *Acta Otolaryngol.* 73, 235–248.

Spoendlin H, Schrott A. (1989) Analysis of human auditory nerve. *Hear Res.* 43, 25–38.

Stapells D, Picton T, Durieux-Smith A. (1994) Electrophysiologic measures of frequency-specific auditory function. In: Jacobson J, ed. *Principles & Applications in Auditory Evoked Potentials.* Boston, MA: Allyn and Bacon; pp 251–283.

Starr A, Picton T, Sininiger Y, Hood L, Berlin C. (1996) Auditory neuropathy. *Brain.* 119, 741–753.

Stockard J, Sharbrough F, Tinker J. (1978) Effects of hypothermia on the human brainstem auditory response. *Ann Neurol.* 3, 368–370.

Stockard J, Stockard J, Westmoreland B, Corfits J. (1979) Brainstem auditory-evoked responses: normal variation as a function of stimulus and subject characteristics. *Arch Neurol.* 36, 823–831.

Teas D, Eldredge D, Davis H. (1962) Cochlear responses to acoustic transients: an interpretation of whole-nerve action potentials. *J Acoust Soc Am.* 34, 1438–1459.

Webster D, Popper A, Fay R. (1992) *The Mammalian Auditory Pathway: Neuroanatomy.* New York, NY: Springer-Verlag.

CHAPTER 12

Electrocochleography

Rosamaria Santarelli and Edoardo Arslan*

INTRODUCTION

Electrocochleography (ECochG) is the technique of recording the electrical responses that occur in the cochlear hair cells and auditory nerve in response to acoustic stimulation. ECochG potentials were first obtained by Wever and Bray (1930) who recorded the electrical responses evoked by different acoustic stimuli by using a wire hook electrode placed on the auditory nerve in cats. These potentials closely reproduced the sound waveform and also transmitted speech with great fidelity when electric signals were sent to a loudspeaker, so that the experimenters were able to communicate from one room to another by whispering in the cat's ear. Wever and Bray interpreted these responses as arising from the auditory nerve; however, further research demonstrated that they were generated in the cochlea and they came to be known as cochlear microphonic (CM). Since then, several types of evoked cochlear responses have been recorded from experimental animals as well as from patients showing perforation of the tympanic membrane or who have had ear surgery, via an electrode on the promontory wall or in the window niche. Subsequently, ECochG potentials were recorded from the promontory wall in humans by using a needle electrode which was passed through the tympanic membrane with the aid of an operating microscope (transtympanic approach).

The assessment of the origin of the different types of cochlear potentials in different experimental settings as well as the introduction of several techniques for ECochG recording in humans prompted the proposal that ECochG be used in clinical practice. In the 1970s, academic centers began using the transtympanic approach to assess hearing threshold in uncooperative children (Aran et al., 1971; Arslan et al., 1983). However, soon after its introduction for hearing evaluation, ECochG was set aside because auditory brainstem responses (ABRs) were recognized as a reliable noninvasive tool for hearing threshold estimation in uncooperative children. Nevertheless, over the years the identification of distinctive features of ECochG potentials in some ear disorders such as Ménière disease (Eggermont, 1974) together with the development of noninvasive recording techniques spurred the inclusion of ECochG in the standard protocol for the diagnosis of Ménière disease in many medical centers.

In recent decades, the identification of new disorders such as auditory neuropathy (AN), believed to involve the auditory nerve, inner hair cells (IHCs), and/or the connecting synapses (Starr et al., 1996), prompted the use of a sensitive recording technique for defining the details of potentials arising in both the cochlea and auditory nerve. In addition, it was recognized that the reliability of ABRs in hearing threshold estimation testing was reduced in some categories of children such as those affected by neurologic disorders involving the brainstem (Kraus et al., 1984), thus leading to revision of the role of ECochG in clinical practice and its proposal as a valuable tool for the functional assessment of auditory periphery.

In this chapter, we shall review first the main features of the different components of ECochG potentials and their underlying generators, and then focus on crucial information related to the recording technique. In subsequent sections we will analyze the features of the ECochG potentials recorded from the promontory of normally hearing individuals in response to different types of acoustic stimuli and address the changes induced in these responses by cochlear hearing loss. The main features of ECochG potentials along with their clinical use will then be addressed for specific disorders such as Ménière disease and AN as well as for hearing dysfunction associated with neonatal illnesses.

GENERAL FEATURES OF ECochG POTENTIALS

The ECochG potentials evoked in response to acoustic stimuli result from the superimposition of three components, two originating from receptor elements, the CM and summating potential (SP), and the other, the compound action potential (CAP), arising from auditory nerve fibers (Eggermont, 1976). These components are intermingled in the recordings obtained in response to stimuli of a given polarity and, depending on the type and intensity of acoustic stimulation, cannot easily be distinguished from one another. The most popular method for separating CM from SP and CAP components is

*Deceased

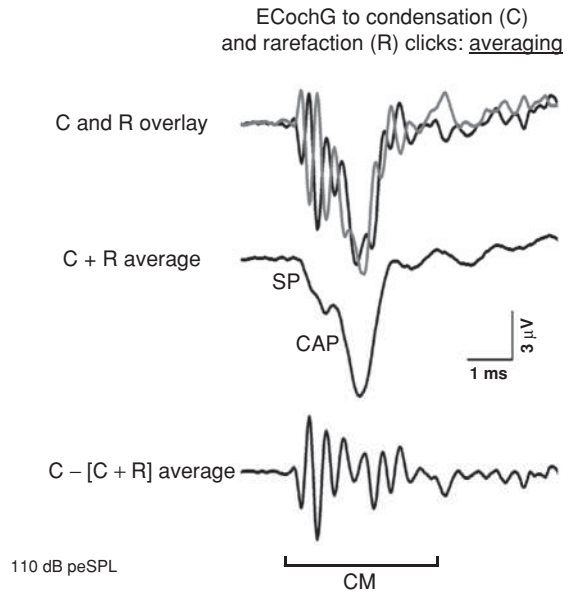


FIGURE 12.1 Procedure utilized to separate the cochlear microphonic (CM) from the compound action potential (CAP) and summating potential (SP). The **top panel** displays the responses to condensation (C, *gray line*) and rarefaction (R, *black line*) clicks recorded from the promontory wall in one normally hearing subject in response to high-level click stimuli (110 dB peSPL). In the **middle panel** the condensation and rarefaction waveforms have been averaged to attenuate CM thus revealing SP and CAP (C + R average). The CM shown in the **lower panel** results from subtracting the (C + R) average from the ECochG response to condensation clicks. (Reprinted with permission from Santarelli R, Starr A, Michalewski H, Arslan E. (2008) Neural and receptor cochlear potentials obtained by transtympanic electrocochleography in auditory neuropathy. *Clin Neurophysiol.* 119, 1028–1041.)

illustrated in Figure 12.1. The top panel displays the ECochG waveforms recorded from the promontory wall in one subject with normal hearing in response to high-level click stimuli (110 dB peak equivalent SPL). Superimposed responses to condensation and rarefaction clicks show the phase-reversed CMs intermixed with negative in-phase SP and CAP. Since CM activity is related to the basilar membrane motion, the procedure of averaging the responses evoked separately by condensation and rarefaction stimuli is applied to extract the CAP together with the superimposed SP. This is shown in the middle panel where the condensation and rarefaction waveforms have been averaged to cancel the CM and reveal the SP and CAP components. The averaged curve is then subtracted from the response evoked by condensation (or rarefaction) stimuli to obtain the CM (lower panel).

CM is believed to originate mainly from the sum of the extracellular components of receptor potentials arising in IHCs and outer hair cells (OHCs), with the latter contributing more to CM generation because of their greater number (Dallos and Cheatham, 1976). Taking into account the estimated length constant (the distance at which the voltage declines to 37% of its value as measured at the place of origin) of the extracellular potential decay along the cochlear partition, the CM recorded at the promontory is thought to arise chiefly in hair cells located in the basal portion of the cochlea. This has been confirmed by experimental data showing that the low-frequency microphonic measured at the round window in guinea pigs is dominated by contributions arising from the basal turn of the cochlea (Patuzzi et al., 1989). Recently, compared the CM and CAP tuning curves obtained for round window recordings in mice through a forward masking paradigm and found that whereas CAP responses are sharply tuned along the whole cochlear partition, CM potentials are tuned only at high frequencies. At these frequencies the rapidly changing phase prevents appreciable summation of extracellular potentials generated by transduction currents in hair cells. Therefore, the CM responses recorded at the round window are thought to result from the passive activation of cochlear receptors with little or no contribution from the active component related to the activity of the cochlear amplifier. These findings suggest that otoacoustic emissions (OAEs) and CM recordings cannot be considered equivalent in assessing the functional integrity of OHCs as they reflect different features of OHC activation, that is, an electric event correlated with the passive motion of the basilar membrane in the case of CM potentials and a mechanical event resulting from active contraction of OHCs for OAE responses.

Compared to CM, the origin of SP is much more controversial. In general, the SP is considered to be a gross reflection of the DC component of receptor potentials, which results from asymmetries in the hair cell transducer function. Because the SP recorded at the round window in chinchillas has been found to decrease in amplitude by over 50% after selective destruction of IHCs (Durrant et al., 1998), the main contribution to SP generation is believed to arise from activation of IHCs located in the basal cochlear turn.

One typical feature of the SP component is that its polarity is dependent upon several factors such as the frequency and intensity of acoustic stimulation, location of the recording electrode, and presence of a cochlear lesion. These polarity changes are believed to depend on the location from which the electrode "sees" the distortion component arising from IHC activation, but they could also reflect the differential contribution of OHCs and IHCs to SP generation. Durrant et al. (1998) have suggested that the residual SP recorded at the round window in chinchillas after selective destruction of IHCs results from the activation of apical OHCs. This is because the receptor potentials arising in OHCs located in the basal turn are symmetrical and cannot contribute to SP generation. In addition, it is generally

acknowledged that electrical activity arising from neural sources such as auditory nerve and cochlear nuclei also contributes to SP generation. Specifically, the abrupt negative potential preceding the CAP evoked by clicks or tone-burst stimuli is believed to arise only from hair cells; however, the baseline shift recorded during sustained tone-burst stimulation is thought to reflect the activation of both receptor and neural elements.

CAP results from the weighted sum of the extracellular components of the action potentials generated by individual auditory nerve fibers in response to acoustic stimulation. In the late 1950s Goldstein and Kiang (1958) hypothesized that CAP could be modeled mathematically as the convolution of the voltage contribution of individual nerve fibers (unit response) with the corresponding probability density function for unit discharges. Subsequently, Kiang et al. (1976) estimated the unit responses by triggering the averaged neural potentials recorded at the round window in cats in response to click stimulation with the spikes arising from individual fibers with different characteristic frequencies (CFs). Then, the contribution of single fibers to the CAP waveform was estimated by convoluting the averaged unit response with the corresponding poststimulus time histogram. The resulting waveforms were summed with appropriate weightings according to the number and distribution of CFs in the auditory nerve. The prediction curve closely resembled the CAP response recorded at the round window in response to click stimulation.

On the basis of these findings, Elberling (1976) modeled the response of the whole auditory nerve evoked by high-level click stimuli. Although click stimulation sets in motion the whole cochlear partition, the model predicts that only fibers with high CF contribute to the CAP. This is because the action potentials arising from fibers with low CF tend to disperse in time as the traveling wave progresses toward the apex of the cochlea thus resulting in a limited probability of summation (Figure 12.2). On the other hand, the number and type of neural fibers contributing to CAP in the basal portion of the cochlea depend on the intensity of click stimulation. Indeed, lowering stimulus level results in a reduced recruitment of high-threshold fibers showing high CF and short latency of activation. As a consequence, the main contribution to CAP generation at low intensity comes from fibers showing lower CF and longer activation latency.

Responses arising from far more restricted areas of the basilar membrane can be obtained by using tone-burst stimuli presented at low intensity (Eggermont, 1974). CAP tuning curves obtained for round window recordings in mice in response to tone-burst stimulation (Cheatham et al., 2011) proved to be very similar to the profiles of tuning curves of single nerve fibers tuned to the frequency of tone-burst stimulation. This means that the CAP recorded in response to tone-bursts appears to be sharply tuned at low intensity and tends to lack frequency selectivity at high stimulus levels. This point is crucial when using CAP evoked by tone-burst stimuli for hearing threshold estimation since frequency selectivity of the CAP response is expected to be preserved only at low-to-moderate stimulus intensities.

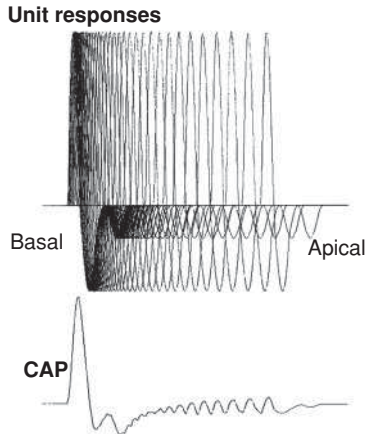


FIGURE 12.2 Schematic diagram of the prediction of the compound action potential (CAP) evoked by click stimulation according to Elberling's model. Unit responses are shown in the **upper panel** spaced with increased latency corresponding to their position along the cochlear partition. The **lower panel** reports the calculated sum-response showing that only fibers with high characteristic frequency contribute to CAP generation. (Reprinted from Elberling C. (1976) Simulation of cochlear action potentials recorded from the ear canal in man. In: Ruben RJ, Elberling C, Salomon G, eds. *Electrocochleography.* Baltimore, MD: University Park Press; pp 151–168.)

RECORDING TECHNIQUES

Recording Site

There are several technical approaches for recording ECochG potentials. In the transtympanic approach, a sterile stainless steel needle electrode, insulated except for the tip, is passed through the tympanic membrane and placed on the promontory wall with the aid of an operating microscope. This procedure requires general anesthesia in children and local anesthesia of the tympanic membrane in adults. It should be performed with the assistance of a physician. Nevertheless, the medical risk of piercing the eardrum is minimal. Another intratympanic technique used at some medical centers consists of placing a "golf-club" electrode in the round window niche after performing posterior myringotomy (Aso and Gibson, 1994). Both intratympanic procedures yield high amplitude potentials because the active electrode is close to the bioelectric generators of cochlear responses. Specifically, the "window-niche" approach is the most sensitive procedure and has the advantage of providing better estimation of residual hearing at low frequencies, thus providing useful information before cochlear implantation in children;

however, it is far more invasive compared to the transtympanic approach.

Several extratympanic techniques have been proposed for ECochG recording. Usually, the active electrode is a ball-tipped electrode placed on the skin of the outer ear canal or on the eardrum. The potentials recorded from the eardrum are larger than those obtained with the electrode resting on the wall of the ear canal, but overall, amplitudes of the ECochG responses recorded from extratympanic sites are far smaller compared to the intratympanic approach because of the greater distance of the active electrode from the cochlea and auditory nerve.

Differences between ECochG recordings obtained from intratympanic and extratympanic sites are illustrated in the example given in Figure 12.3. ECochG waveforms were collected from one child with cochlear hearing loss by simultaneously recording intratympanic and extratympanic potentials. The first were obtained through a transtympanic approach whereas the latter were recorded by means of a ring electrode resting on the skin of the outer ear canal close to the annulus. It can be noticed that CAP amplitude is remarkably greater in the transtympanic derivation. As a consequence, in this particular case, relying on the extratympanic approach for hearing threshold assessment would have led to an overestimation of hearing loss of no less than 10 dB.

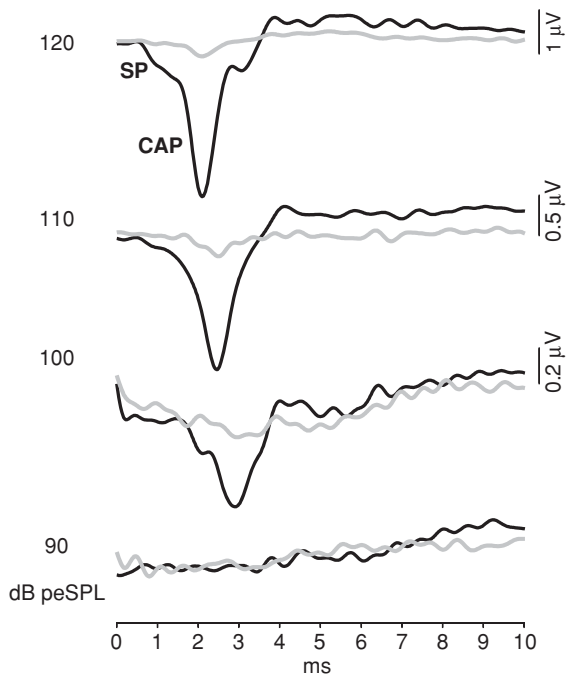


FIGURE 12.3 Simultaneous intratympanic and extratympanic recordings of ECochG responses. ECochG waveforms were collected from one child with cochlear hearing loss at decreasing stimulus levels by simultaneously recording from intratympanic (*black line*) and extratympanic (*gray line*) sites. CAP amplitudes proved to be far smaller in the extratympanic derivation with consequent overestimation of the amount of hearing loss. (Reprinted with permission from Santarelli R, Arslan E. (2013) Electrocochleography. In: Celesia GG, ed. *Disorders of Peripheral and Central Auditory Processing. Handbook of Clinical Neurophysiology.* Vol 10. Amsterdam: Elsevier; pp 83–113.)

In general, the choice of the recording site depends on the purpose of ECochG recordings. A highly sensitive approach is required when recording low-amplitude potentials, requested in hearing threshold assessment, or for evaluation of cochlear activities in patients with AN. In contrast, recording from extratympanic sites appears to be highly reliable for suprathreshold clinical application such as ECochG recordings used for the diagnosis of Ménière disease. The extratympanic approach is probably the procedure of choice in this condition because of the obvious advantage of being noninvasive and independent of the medical setting. Nevertheless, the choice of both electrode site and position in this clinical application also depends on the experience of individual examiners in using a specific technique.

Stimuli

Clicks or tone-bursts are the most commonly used stimuli for evoking ECochG potentials. When using the transtympanic approach, stimulation is usually performed in the free field. In our laboratory we use two high-frequency loudspeakers mounted on a single polyurethane horn (Santarelli et al., 2008). In such conditions the calibration of the stimulus should be very accurate and it is performed by means of a professional sound level meter with the microphone placed at the distance of the patient's ear from the horn. The procedure of comparing the peak-to-peak amplitude of the click to the peak-to-peak amplitude of a 2-kHz tone can be used to calibrate the click level (peak equivalent sound pressure level, peSPL). The maximum stimulus intensity for clicks used in our laboratory is 120 dB peSPL (corresponding to 90 dB nHL, as referred to the psychoacoustical threshold of normally hearing subjects) whereas tone-bursts are presented at the maximum intensity of 100 dB SPL.

In the extratympanic approach and in intratympanic ECochG recordings with the electrode placed in the round window niche, acoustic stimulation is performed by using headphones or insert phones.

As mentioned above, stimulation with clicks is not frequency selective and mainly reflects the activation of the basal portion of cochlear partition, with the contribution of more basal regions varying with the intensity of stimulation. Tone-bursts are considered to be frequency selective, but the degree of frequency selectivity critically depends on the intensity of stimulation.

Another point to be considered when using tone-burst stimulation concerns the rise–fall time of stimuli. Short rise

time, while enhancing synchronization of neural activity by increasing the number of activated fibers, has a detrimental effect on frequency selectivity because of the progressive recruitment of units with high CF. This is a crucial point when using low-frequency tone-burst stimuli for hearing threshold estimation. In practice, preservation of both synchronization and frequency selectivity requires a trade-off between center frequency and rise time of tone-bursts. A rise time equaling two periods of the sine wave is accepted as a good compromise (Eggermont, 1976).

It is important to note that the choice of type and intensity of stimuli depends on the purpose of ECochG recording. Although click presentation remains the most widely used stimulation, we shall see later in this chapter that tone-bursts or more complex stimuli resulting from the combination of different acoustic waveforms have been proposed for specific clinical applications or for evaluation of the electrical activity arising from apical portions of the cochlea.

Recording

Both amplification factor and number of averaged sweeps depend upon the amplitude of the potentials that are recorded. When using the transtympanic approach, an amplification factor of 50,000 is appropriate, whereas higher amplification factors could be required when recording from an extratympanic site.

The number of acquired samples for a single average, when using an intratympanic approach at high stimulation level, can be as low as 100 to 200. However, more samples are usually required at low intensity to reduce the amount of noise and to improve the clarity of the waveform. In our laboratory we use 500 samples for each stimulus polarity (condensation and rarefaction). More trials may be required (at least 1,000) when using an extratympanic approach, the number of averaged samples depending upon the specific electrode placement.

The bandwidth of filter settings should be kept as wide as possible (5 to 8,000 Hz). A high cutoff frequency is desirable to avoid distortion of rapid components as high-frequency noise can effectively be eliminated off-line by using digital filters. A low cutoff frequency is highly recommended for recording "slow" ECochG potentials such as those obtained from patients with AN and for measuring SP amplitude during sustained tone-burst stimulation.

Signal Processing and CAP Extraction

As mentioned above, the procedure most used to cancel the CM and extract both SP and CAP components consists of averaging the responses evoked separately by condensation and rarefaction stimuli (Figure 12.1). This procedure assumes that the CMs evoked by stimuli of opposite polarity are in exact counterphase. This may not always be the case for several reasons. First of all, asymmetry of CM potentials may occur in cochlear lesions. Moreover, acoustic stimulation, particularly when performed at high intensity in the free field, may lead to generation of reflection waves in the outer ear canal which impinge differently on the tympanic membrane when inverting the stimulus polarity. Most importantly, differences in CAP latency have been observed in response to condensation and rarefaction stimuli also in normally hearing ears. These differences have been found to be significantly greater in patients with endolymphatic hydrops. Therefore, a new algorithm has been developed based on the theory of optimal filtering, which estimates CAP and CM components in response to clicks of the same polarity without making any assumption regarding the shape of cochlear potentials (Sparacino et al., 2000). An example is reported in Figure 12.4 for ECochG waveforms obtained from one normally hearing ear in response to 110 dB peSPL clicks. It can be seen that the use of the optimal filtering procedure

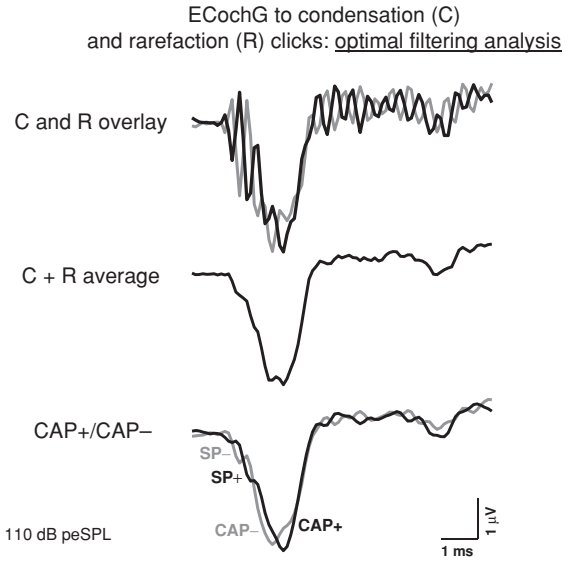


FIGURE 12.4 Analytical procedure based on the theory of optimal filtering to separate the cochlear microphonic (CM) from the compound action potential (CAP) and summating potential (SP). The **top panel** displays the responses to condensation (C, *black line*) and rarefaction (R, *gray line*) clicks recorded from the promontory wall in one normally hearing subject in response to high-level click stimuli (110 dB peSPL). The ECochG waveform obtained by the procedure of averaging the traces to stimuli of opposite polarity is displayed in the **middle panel**. In the **lower panel** CAP and SP have been independently estimated in response to condensation or rarefaction stimuli by using the optimal filtering procedure. The use of this procedure reveals differences in CAP latency and SP amplitudes between stimuli of opposite polarity which would have been overlooked using the classical CM cancellation method.

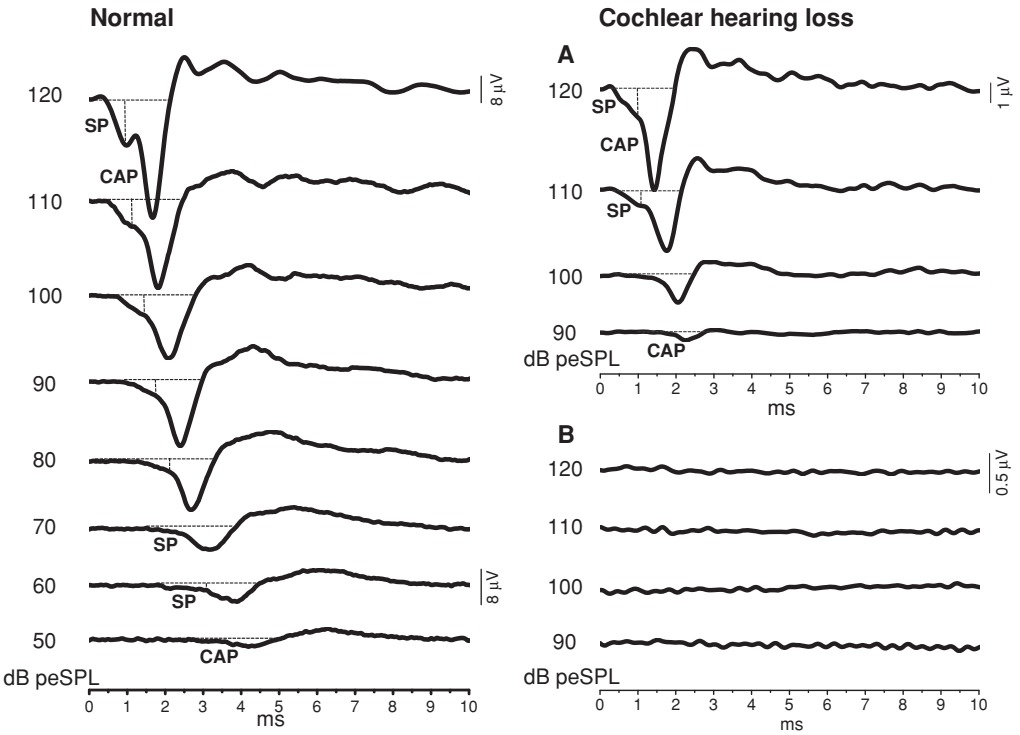


FIGURE 12.5 ECochG potentials in response to clicks at decreasing stimulus intensities recorded from one normally hearing child (**left panel**) and two hearing-impaired children (**right panel**), one with moderate hearing loss (**A**) and the other showing profound deafness (**B**). In the subject with normal hearing, decreasing stimulus level results in gradual latency increase and amplitude reduction of both SP and CAP peaks. SP and CAP were recorded in moderate deafness with increased threshold and reduced amplitude compared to the normal control. In contrast, no ECochG potentials were obtained from the child with profound deafness. SP and CAP labels are reported for the maximum level and for threshold intensity. Time 0 marks CM onset, the *horizontal lines* refer to baseline whereas *dashed vertical lines* mark the SP and CAP peaks in this and in the figures following.

reveals differences in CAP latency in response to clicks of opposite polarity. These differences would have been overlooked using the classical CM cancellation method. In addition, when using the optimal filtering algorithm in this particular example, different SP amplitudes are obtained in responses evoked by condensation and rarefaction clicks. In contrast, SP could not be identified in the waveform obtained through the classical method probably because the amplitude differences between the SP components in response to clicks of opposite polarity result in a smeared SP response in the averaged curve.

THE NORMAL ECochG RESPONSE

After canceling the CM, ECochG potentials consist of SP and CAP components. The shape, amplitude, and peak latency of these responses are dependent on both stimulus type and intensity. An example of ECochG recordings obtained from a normally hearing child in response to 0.1 ms clicks at decreasing stimulation intensities is shown in Figure 12.5 (left panel). At 120 dB peSPL the ECochG waveform begins with the receptor SP, which appears shortly after the onset of CM as an abrupt negative deflection preceding the CAP. Both SP peak amplitude and slope diminish as stimulus level is reduced. In this particular example, SP is still identifiable at an intensity as low as 60 dB peSPL (corresponding to 30 dB nHL).

CAP arises from SP as a negative peak whose latency is progressively delayed as signal intensity decreases. Decreasing stimulus level also results in CAP amplitude reduction whereas the duration is relatively constant at suprathreshold intensities and broadens at low stimulus level. In this particular example, CAP is identified as low as 50 dB peSPL, which corresponds to 20 dB nHL referred to the psychoacoustical threshold of normally hearing subjects.

Mean latency, amplitude, and duration of ECochG potentials are plotted against stimulation intensity in Figure 12.6 for a large sample of normally hearing ears (36 ears from 23 children). Latency was calculated with respect to CM onset, whereas amplitude was measured with respect

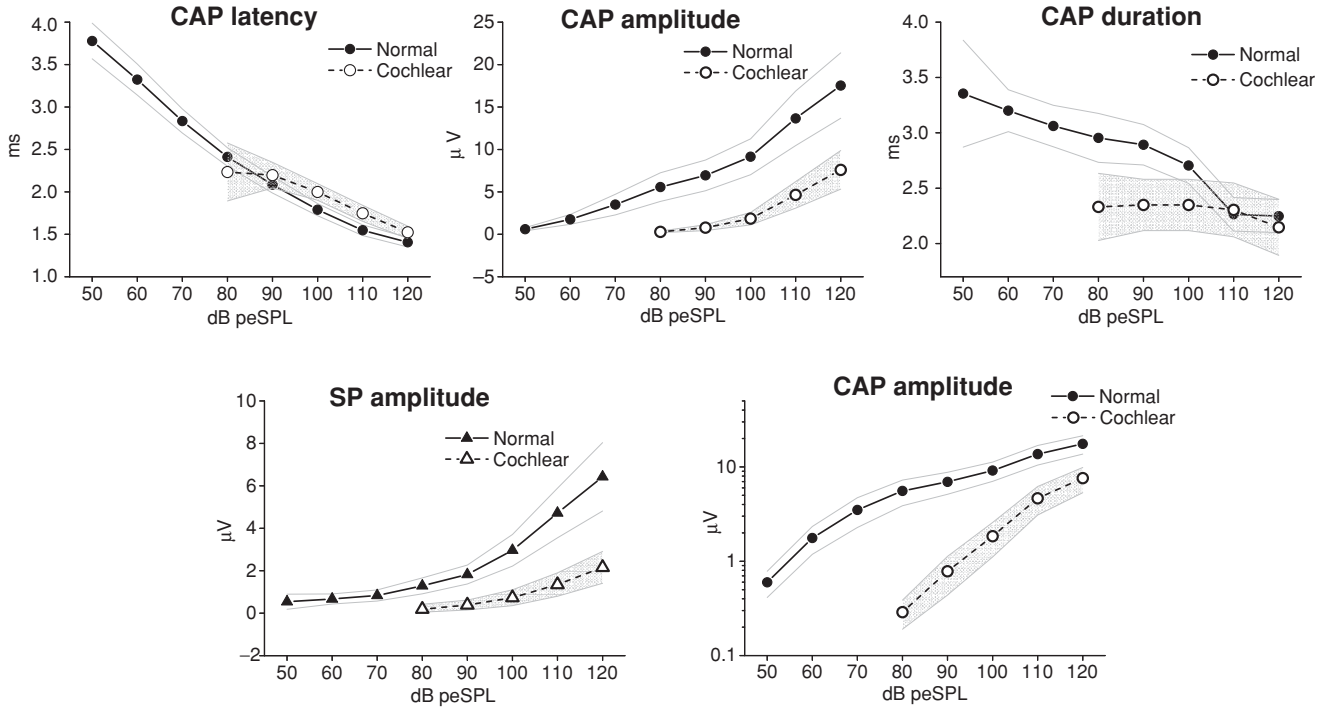


FIGURE 12.6 Intensity functions of CAP and SP potentials in normal hearing and cochlear hearing loss. Mean CAP latency, amplitude, and duration with 95% confidence limits (*shadowed area*) are reported as a function of stimulus intensity for a group of normally hearing ears (36 ears from 23 children) and ears with cochlear hearing loss (19 ears from 23 children, CAP threshold from 80 to 100 dB peSPL). Intensity curves calculated for SP amplitudes are reported in the **lower panel**. Both SP and CAP showed a reduced amplitude in cochlear hearing loss compared to normal hearing without differences in CAP latency. In the **lower panel**, CAP amplitudes have also been plotted on a logarithmic *Y*-scale to show the knee-point marking the activation of the cochlear amplifier at low intensity. In contrast, the amplitude–intensity function calculated for cochlear hearing loss shows an entirely linear behavior and an increased steepness reflecting the lack of cochlear amplifier. (Modified with permission from Santarelli R, Arslan E. (2013). Electrocochleography. In: Celesia GG, ed. *Disorders of Peripheral and Central Auditory Processing. Handbook of Clinical Neurophysiology.* Vol 10. Amsterdam: Elsevier; pp 83–113.)

to prestimulus baseline. As in the example reported in Figure 12.5, decreasing stimulus level results in lengthening of CAP latency and decrease of CAP amplitude. Interestingly, the slope of the amplitude–intensity function is steep at high levels and becomes shallower at low intensities. The duration, which was measured from SP onset to CAP return to baseline, tends to increase at low intensities, but it is not longer than 3 to 4 ms at threshold (50 dB peSPL). The behavior of CAP intensity functions can be explained by taking into account the degree of synchronization of auditory nerve firing during the progression of the traveling wave along the cochlear partition. As mentioned above, at high intensities the most striking contribution to CAP recording during click stimulation arises from auditory fibers innervating the most basal portion of the cochlea. As a consequence, CAP shows a short latency, large amplitude, and short duration because of the synchronous firing of large numbers of high-CF units with high thresholds and short latencies of activation. Decreasing signal intensity induces firing of low-threshold fibers with lower CF and delayed latency of activation because of the progression of the traveling wave along the cochlear partition. The discharge of these units appears to be more dispersed in time and has lower probability of summation compared to high-threshold fibers. As a result, when decreasing the stimulus level, CAP broadens and decreases in amplitude whereas peak latency increases. Nevertheless, the electrical activity arising from auditory fibers innervating the cochlear apex has little or no probability of summation (see Figure 12.2). In practice, the CAP elicited by clicks through a wide range of stimulus intensities mostly results from the activation of auditory fibers with CFs higher than 1 kHz.

CAP recordings obtained in response to tone-burst stimuli at low-to-moderate intensities are sharply tuned on the stimulus frequency. Figure 12.7 illustrates an example of the CAP intensity series obtained from a normally hearing ear (same ear as in Figure 12.5) in response to tone-burst stimuli at different frequencies. Mean CAP peak latency, amplitude, and duration calculated across 10 ears with normal hearing are also reported. Because at low stimulus

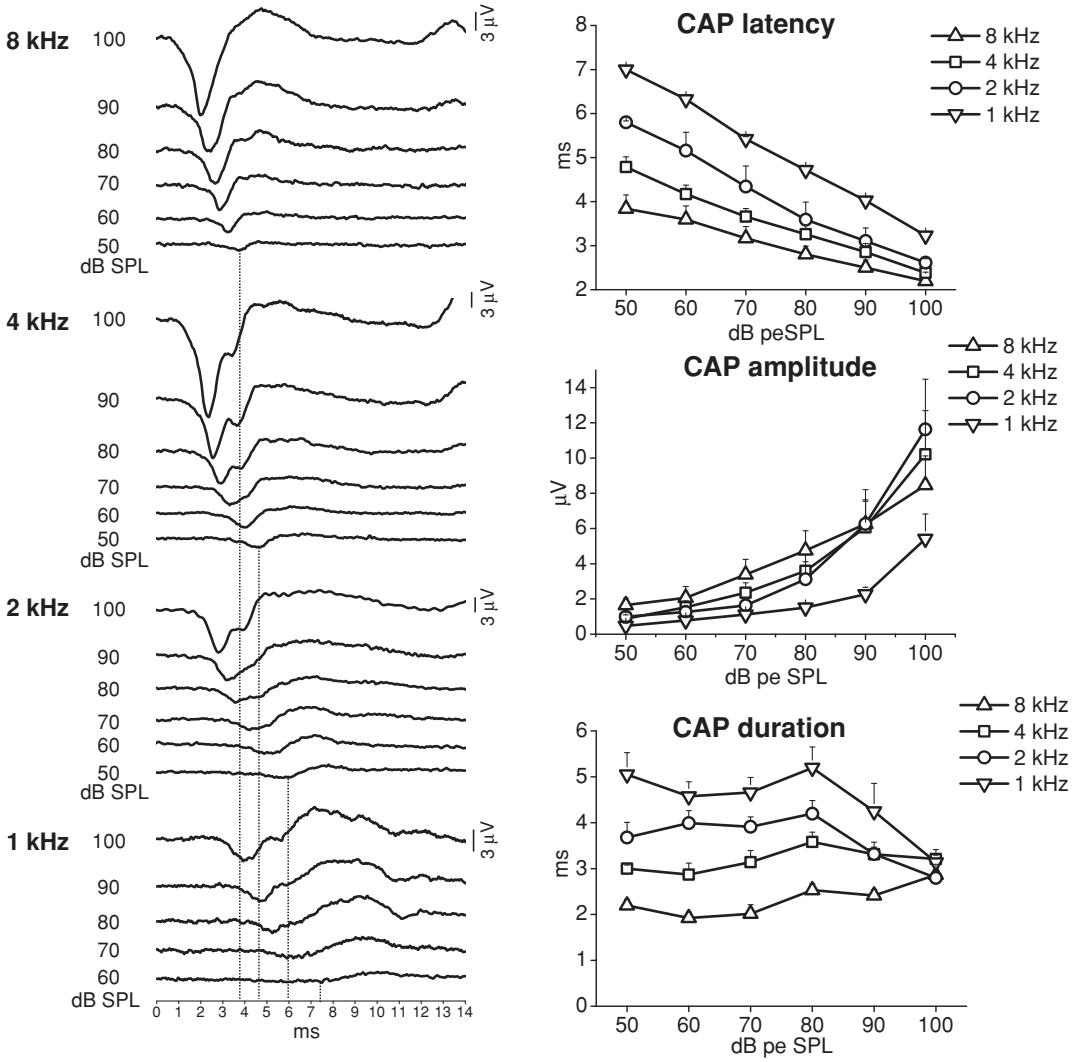


FIGURE 12.7 CAP recordings in response to tone-burst stimuli in ears with normal hearing. The **right panel** shows an example of CAP intensity series obtained from one normally hearing ear (same ear as in Figure 12.5) in response to tone-burst at frequencies of 8, 4, 2, and 1 kHz at decreasing stimulation intensity. Mean CAP peak latency, amplitude, and duration calculated across 10 ears with normal hearing are reported in the **right panel**. At low stimulus intensity, CAP latency and duration increase and amplitude decreases when lowering stimulation frequency reflecting the activation of more apical regions of the cochlear partition. These differences tend to disappear at high stimulus level because of the progressive recruitment of high-CF fibers. One bar indicates one standard error. (Reprinted with permission from Santarelli R, Arslan E. (2013) Electrocochleography. In: Celesia GG, ed. *Disorders of Peripheral and Central Auditory Processing. Handbook of Clinical Neurophysiology.* Vol 10. Amsterdam: Elsevier; pp 83–113.)

levels tone-burst stimulation results in selective activation of restricted regions of the cochlea, both CAP latency and duration tend to increase with decreasing stimulus frequencies. This reflects the activation of increasingly apical regions of the cochlear partition. These latency and duration changes at different frequencies tend to become smaller at high stimulus levels because of the activation of progressively larger cochlear regions biased toward the cochlear base. This behavior is cause for concern when using tone-burst stimuli for the assessment of hearing threshold at specific tone frequencies.

ECochG POTENTIALS IN COCHLEAR HEARING LOSS

Cochlear lesions result in variable degrees of hearing impairment because of hair cell loss. Extensive damage is associated with severe-to-profound hearing loss with absent CAP at high

stimulation intensity, whereas lower degrees of hair cell loss result in variable amounts of CAP threshold elevation.

ECochG recordings obtained in response to click stimulation at decreasing stimulus levels from two children showing respectively moderate and profound hearing loss are reported on the right side in Figure 12.5. No CAP response can be identified at the maximum intensity (120 dB peSPL) in the child with profound deafness (Figure 12.5B). The subject with moderate hearing impairment (Figure 12.5A) shows both SP and CAP components with peak latencies within the normal range; however, CAP threshold appears to be increased and amplitude is markedly reduced compared to the normally hearing control.

Figure 12.6 reports the mean intensity functions obtained from 19 children with bilateral cochlear hearing loss showing CAP response in ECochG recordings (23 ears). These are superimposed on the corresponding functions calculated for ears with normal hearing. To limit heterogeneity, this sample included only children with isolated sensory/neural hearing loss and flat configuration of the audiometric profile as evaluated by behavioral audiometry in the years following ECochG recording. Moreover, etiology of the hearing disorder was genetic in almost all cases. Further criterion for selection was that CAP threshold fell between 80 and 100 dB peSPL to limit the variability of the CAP parameters related to the extension of cochlear damage. Looking at the mean intensity functions calculated for this sample of children, both SP and CAP amplitudes proved to be significantly smaller compared to normally hearing ears. These differences are likely to reflect the decrease in hair cell number, which results in a global reduction of auditory nerve fiber activation. CAP latencies appear to be within normal limits, whereas CAP duration is close to normal values at high levels and deviates from the normal behavior at low intensities as no response prolongation was found when lowering the stimulus level. The absence of remarkable differences in both CAP latency and durations between normal and hearing-impaired ears at high stimulus intensity reflects the preservation of fibers with high threshold and short latency of activation. Shortening of CAP duration at low stimulus levels is likely to result from the loss of low-threshold neural fibers with longer latency of activation. Nevertheless, both CAP latency and duration in response to clicks depend on the audiometric profile as differences in hearing threshold at different frequencies are the major determinant of nerve fiber recruitment along the cochlear partition.

In Figure 12.6 CAP amplitude–intensity curves are also plotted on a logarithmic *Y*-scale. The knee-point in the intensity function calculated for normal ears marks the transition from the steep to the shallow portion of the curve, which refers to the activation of the cochlear amplifier at low stimulus intensities. In contrast, the amplitude–intensity function calculated for cochlear hearing loss shows an entirely linear behavior associated with an increased steepness of the curve compared to normal hearing. Conceivably, the linearity of amplitude–intensity function in cochlear hearing loss results from the lack of cochlear amplifier with consequent reduction of the compressive behavior found in normal hearing.

The use of ECochG recordings in estimating hearing threshold in uncooperative children relies on the correlation between audiometric and CAP thresholds. Although the stimuli used in behavioral and electrophysiological evaluation differ in both duration and frequency, a close correlation between CAP and audiometric thresholds has been found in several studies performed in both adults and children (Aran et al., 1971; Eggermont, 1976). The most reliable measures were obtained in adults and in children who were able to perform conventional audiometry (Parving et al., 1981). Specifically, when using click stimuli, the best correlation between CAP and behavioral thresholds was found at audiometric frequencies of 1, 2, and 4 kHz with correlation coefficients in the order of 0.8 and slopes of the regression line close to 1. The correlation improves when using tone-burst stimuli in the middle frequency range, whereas at 0.5 kHz the slope of the regression lines deviates considerably from 1 (Eggermont, 1976). Recently, to obtain a functional evaluation of the most apical regions of the cochlea, a promising technique has been proposed based on the use of chirps (Chertoff et al., 2010). These are transient stimuli in which the high frequencies have been delayed to compensate for the time-shift between high- and low-frequency regions of the cochlea, thus enhancing the discharge synchronization of the neural fibers innervating the apical regions. Compared to the CAP elicited by classical clicks, the neural responses evoked by chirps show higher amplitudes and reduced slope of the latency–intensity function possibly as a result of a greater contribution to neural activation from the low-frequency portions of the cochlea.

Besides providing a certain degree of frequency selectivity in threshold estimation, the use of tone-bursts in cochlear hearing loss may complement the stimulation with clicks also to provide additional information that proves to be extremely useful when planning the rehabilitative strategy. Figure 12.8 illustrates the ECochG waveforms recorded in response to clicks and tone-burst stimuli from a child with cochlear hearing loss because of biallelic mutation in the *GJB2* gene. CAP was absent in the left ear in response to click stimulation whereas it was recorded from the right ear with 105-dB peSPL threshold. The behavior of intensity functions was suggestive of a cochlear lesion. In the ECochG waveforms in response to tone-burst stimuli presented at the maximum intensity of 100 dB SPL, CAP was identified only at frequencies of 1 and 2 kHz, whereas no response was recorded at higher frequencies. Sound-field audiometry performed 2 years later indicated 70- to 75-dB hearing threshold from 1 to 4 kHz, thus failing to reveal any difference between middle and high frequencies. Interestingly,

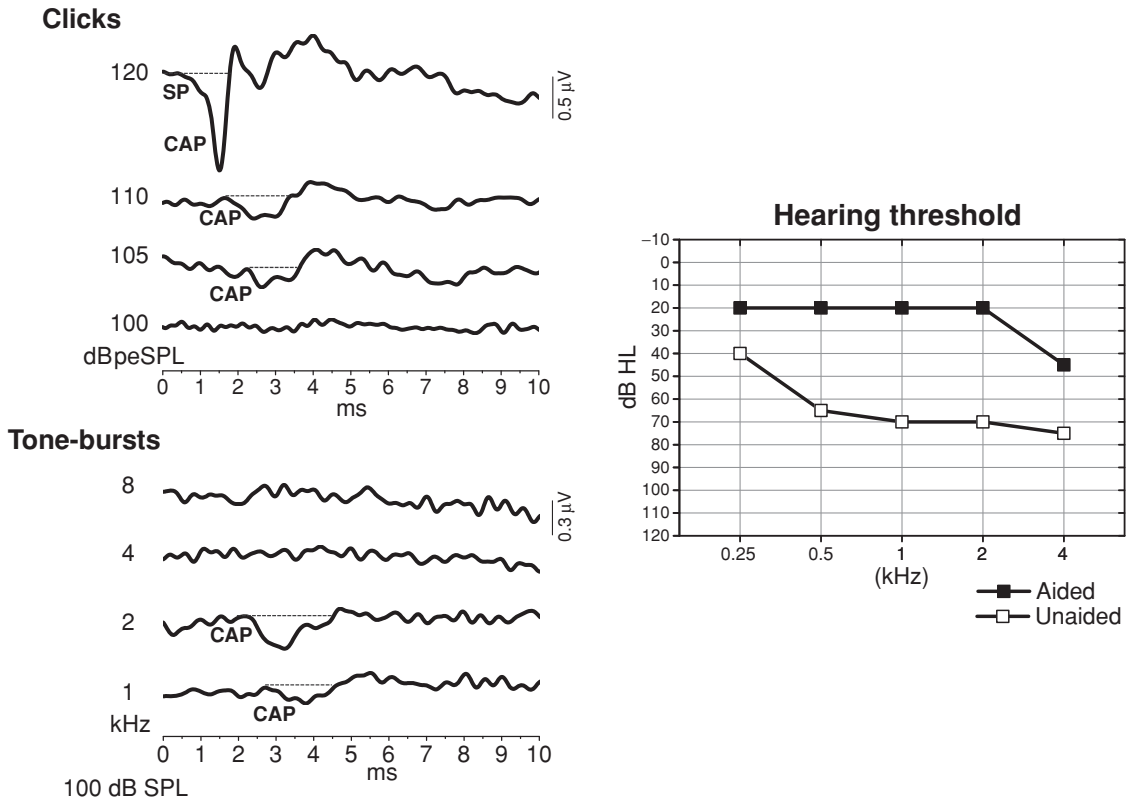


FIGURE 12.8 ECochG responses and audiometric thresholds from one child with cochlear hearing loss because of biallelic mutation in the *GJB2* gene. ECochG potentials to clicks and tone-burst stimuli at decreasing stimulus intensity are displayed in the **left panel**. CAP threshold in response to clicks was 105 dB peSPL (corresponding to 75 dB nHL) whereas CAP responses were elicited only at 0.1 and 2 kHz during tone-burst stimulation with 100-dB SPL threshold. Sound-field audiometry including both unaided and aided thresholds for warble-tones at different frequencies is reported on the **right panel**. The low functional gain found at 4 kHz was in accordance with ECochG responses to tone-burst stimulation thus indicating a predominant involvement of high frequencies by the cochlear damage.

looking at the aided thresholds obtained after achieving the best hearing aid fitting, the functional gain measured at 4 kHz proved to be smaller compared to lower frequencies. This result taken together with the electrophysiological findings suggests cochlear damage primarily involving the high-frequency regions of the cochlea. This information could be valuable for refining hearing aid fitting and could assist in setting the parameters of the frequency transposition algorithm.

Increasing hair cell loss results in extensive cochlear damage with absence of CAP in profound deafness. In contrast, CM potentials are always recorded in ECochG waveforms obtained from hearing-impaired subjects whatever the degree of hearing loss and are believed to result from the activation of residual hair cells (Schoonhoven et al., 1999). Figure 12.9 shows an example of CM recordings obtained in response to click stimuli at decreasing stimulus intensities from two hearing-impaired ears showing elevated CAP threshold (90 dB peSPL) and absence of CAP, respectively. CM waveforms recorded from one ear with normal hearing are also shown. It can be seen that CM is clearly identifiable in both hearing-impaired subjects, although with smaller amplitudes compared to the normal control. Specifically, when comparing CM amplitudes in ears with normal hearing with those obtained from ears with cochlear hearing loss, they proved significantly smaller in hearing-impaired children compared to normal subjects; however, no differences were found between ears with elevated CAP thresholds (same samples as in Figure 12.6) and ears with absence of CAP (32 children, 64 ears). On the basis of these findings it is conceivable that the absence of CMs in surface recordings such as ABRs may be related to the low sensitivity of this technique in detecting low-amplitude CM potentials occurring in ears with extensive cochlear damage. By the same token, the detection of CM in surface recordings does not invariably indicate normal CM and preservation of OHC function. This point is relevant in the diagnosis of AN.

Children showing no CAP in ECochG recordings in response to clicks, who have been followed up at our hospital,

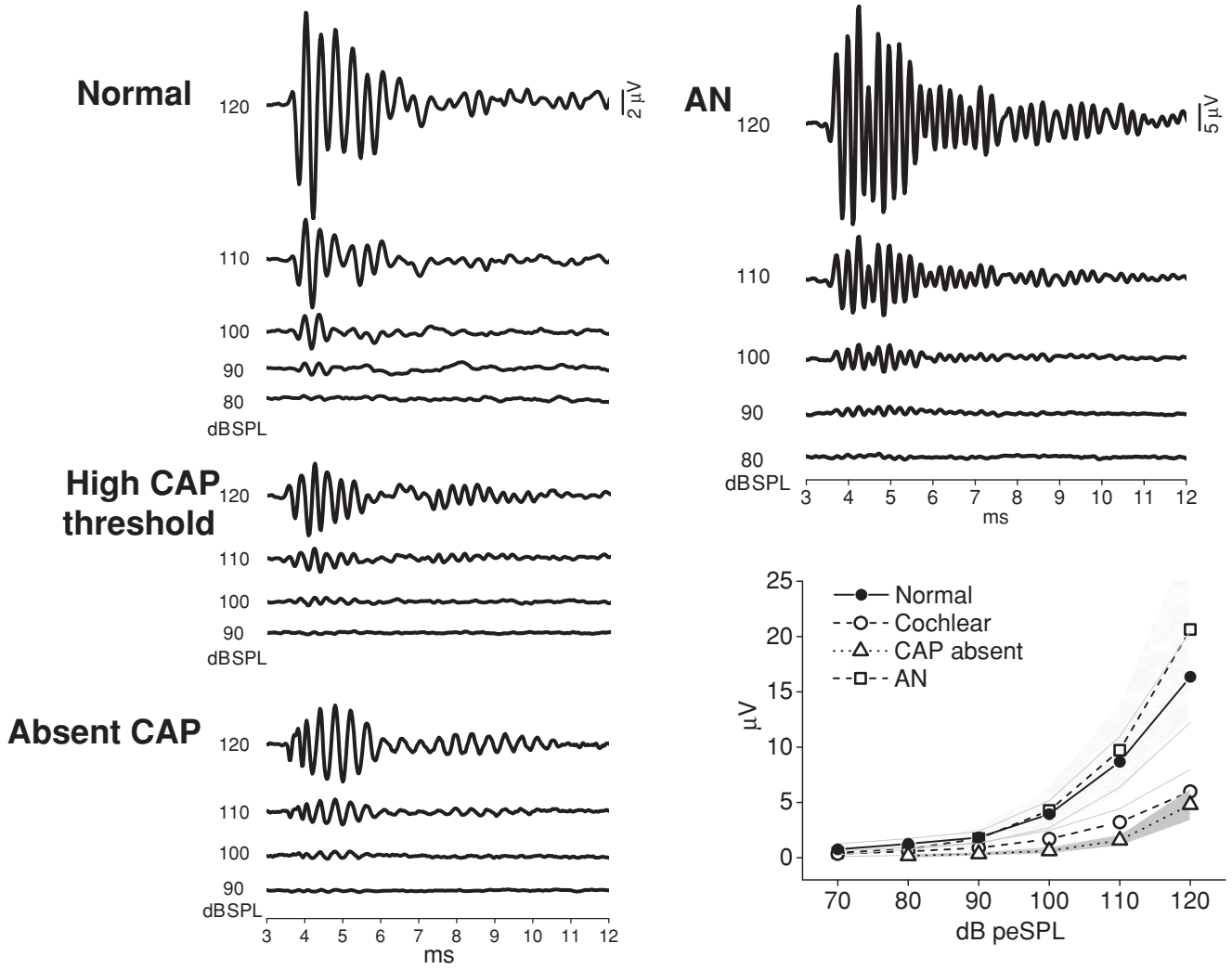


FIGURE 12.9 Cochlear microphonics (CMs) recorded in response to click stimuli at decreasing stimulus intensities from normally hearing individuals, children with cochlear hearing loss, and patients showing the clinical picture of auditory neuropathy (AN). Examples of recordings from one normally hearing and two hearing-impaired ears, one with elevated CAP threshold (90 dB peSPL) and the other with absent CAP, are shown on the **left side** whereas CMs waveforms recorded from one patient with AN are displayed in the **upper right corner**. Mean amplitudes plotted against stimulus intensity for corresponding groups of subjects are reported on the **right side**. CM potentials are clearly identifiable in both hearing-impaired ears with smaller amplitudes compared to the normal control. In contrast, CM amplitudes obtained in the AN subjects were within control values. [Modified with permission from Santarelli R, Arslan E. (2013) Electrocochleography. In: Celesia GG, ed. *Disorders of Peripheral and Central Auditory Processing. Handbook of Clinical Neurophysiology.* Vol 10. Amsterdam: Elsevier; pp 83–113.]

turned out to have profound hearing loss with preservation of some residual hearing at low frequencies as evaluated by behavioral audiometry. This is shown in Figure 12.10 for a group of 68 children. Although indicating a remarkable improvement of puretone sensitivity with hearing aid use, aided thresholds appeared to be beyond the range of conversational speech. On the basis of these findings and taking into account the development of speech perception and language skills, 62 children out of 68 underwent cochlear implantation. The remaining six patients, although included in the candidacy protocol for cochlear implantation, continued to use hearing aids because their parents refused surgery. These findings are relevant in that they suggest that absence of CAP in ECochG potentials in response to high-intensity clicks invariably indicates profound deafness associated with inadequate hearing aid benefit. This information is valuable particularly for rehabilitation planning in uncooperative children for whom the benefits of hearing aid use is difficult to assess because of the reduced ability to perform at behavioral audiometry and speech perception tests. Thus, including ECochG recordings in the assessment protocol for cochlear

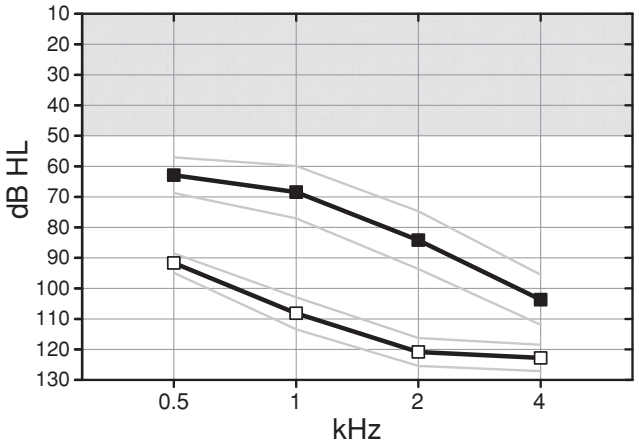


FIGURE 12.10 Means of unaided (*open squares*) and aided (*filled squares*) hearing thresholds with 95% confidence limits (*shadowed area*) from 68 children showing no CAP to click stimulation at high stimulus intensity (120 dB peSPL). All children were fitted with power hearing aids. The *shadowed rectangle* indicates the intensity range calculated for conversational speech. Mean aided thresholds appear to fall beyond the range of conversational speech. (Reprinted with permission from Santarelli R, Arslan E. (2013) Electrocochleography. In: Celesia GG, ed. *Disorders of Peripheral and Central Auditory Processing. Handbook of Clinical Neurophysiology.* Vol 10. Amsterdam: Elsevier; pp 83–113.)

implantation would help in the timely choice of the best rehabilitative strategy.

ECochG RECORDINGS IN MÉNIÈRE DISEASE

The diagnosis of Ménière disease mainly relies on clinical symptoms and thus may be difficult to make, particularly at an early stage. In the 1970s, the finding of increased SP amplitude in ECochG waveforms recorded from patients with Ménière disease was considered as a possible objective hallmark of the disorder (Eggermont, 1976). Since then, several studies have reported that SP amplitude is significantly increased in Ménière disease compared to individuals with normal hearing and hearing-impaired subjects with comparable amount of hearing loss (Mori et al., 1987).

Studies performed on the temporal bones of deceased patients have shown that endolymphatic hydrops is the primary histologic hallmark of the disorder. The increase in endolymph volume is likely to result in an increased pressure in the scala media leading in turn to changes in the electrical properties of hair cells. As a consequence, the receptor potential becomes distorted because of an increase of the DC component, which results in a large SP response. This hypothesis is corroborated by several experimental studies performed in guinea pigs, documenting that changes in SP amplitude could be induced by modifying the resting position of the basilar membrane by means of different techniques such as the association of a continuous low-frequency tone during tone-burst stimulation or the perfusion of perilymphatic spaces with hypotonic solutions.

Unfortunately, in spite of the significant differences in SP amplitude between patients with Ménière disease and subjects with normal hearing or affected by cochlear hearing loss, there is considerable overlap between groups so that the sensitivity of SP amplitude in diagnosing the disorder appears to be no greater than 30% (Mori et al., 1987). Possible underlying reasons are the high variability of SP amplitude in both normal and hearing-impaired subjects and the variable amount of SP attenuation in cochlear hearing impairment resulting from different degrees of hair cell loss. Therefore, the SP enhancement induced by the endolymphatic hydrops may be masked by the amplitude reduction resulting from hair cell loss particularly in the presence of a high degree of hearing impairment. For these reasons, SP amplitude has been replaced by the measure of SP/CAP ratio with improved sensitivity of up to 60% (Coats, 1981; Mori et al., 1987; Orchik et al., 1993; Pou et al., 1996; Sass, 1998). Currently, SP/CAP ratio is the most popular measure performed on ECochG recordings in Ménière disease because it is reliable and easy to obtain. In the majority of studies, ECochG recordings have been obtained in response to high-intensity clicks (90 dB nHL) by using an extratympanic approach. According to the results of these studies, an SP/CAP ratio higher than 0.3 to 0.4 is considered as highly suggestive of endolymphatic hydrops in humans.

Besides changes in SP amplitude, a prolonged duration of ECochG responses has been observed in the ECochG waveforms recorded from patients with Ménière disease (Eggermont, 1976). The increase in duration of ECochG potentials has been attributed to alterations in timing of hair cell activation leading in turn to prolongation of SP potentials. Alternatively, degeneration of terminal dendrites of auditory nerve fibers may lead to abnormal postsynaptic potentials which superimpose on the synchronized neural response (CAP). Examples of ECochG potentials recorded from patients with Ménière disease in response to 120 dB SPL (90 dB nHL) clicks are displayed in Figure 12.11 for two patients showing normal hearing threshold in one ear and moderate hearing loss in the other ear with a puretone average (PTA) (0.5, 1, 2, and 4 kHz) threshold of 30 and 45 dB HL, respectively. These recordings are compared to ECochG waveforms recorded from one normally hearing subject and from one patient with cochlear hearing loss with a PTA of 45 dB HL. In these subjects, SP/CAP ratio was 0.15 to 02 whereas the response duration was around 2 ms. In contrast, patients with Ménière disease showed SP/CAP ratios higher than 0.4 whereas the response duration was considerably prolonged with respect to both normal and hearing-impaired subjects.

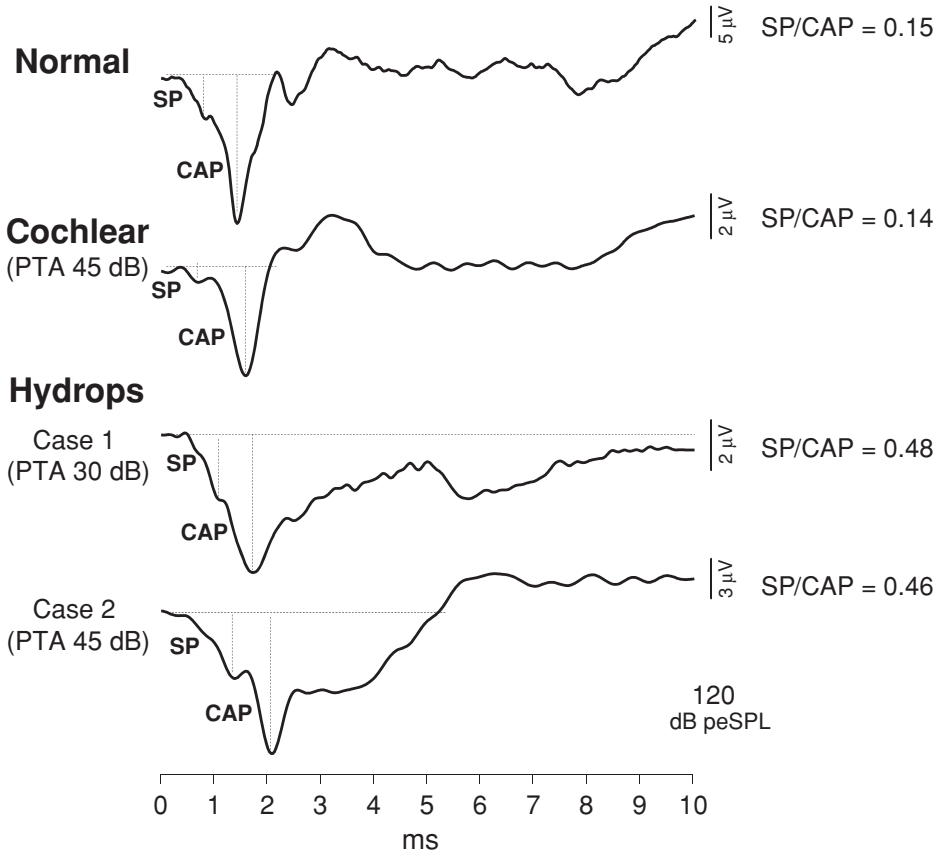


FIGURE 12.11 ECochG recordings in response to 120-dB peSPL clicks from one normally hearing individual, one patient with sensory/neural hearing loss, and two subjects with Ménière disease. Compared to the subjects with normal hearing or with sensory/neural hearing loss, patients with Ménière disease showed increased SP/CAP ratio, delayed CAP peak latencies, and prolonged waveform durations. [Modified with permission from Santarelli R, Arslan E. (2013) Electrocochleography. In: Celesia GG, ed. *Disorders of Peripheral and Central Auditory Processing. Handbook of Clinical Neurophysiology.* Vol 10. Amsterdam: Elsevier; pp 83–113.]

To improve the sensitivity of ECochG recordings in detecting endolymphatic hydrops, further measures have been suggested in addition to the SP/CAP ratio. Ferraro and Tibbils (1999) proposed that when using tone-burst stimulation, response duration could be taken into account by calculating an index resulting from the ratio of the area subtended by the total ECochG waveform to the area subtended by the CAP component below the SP. However, it is unclear whether the inclusion of this measure in the clinical protocol of the diagnosis of Ménière disease provides a substantial advantage compared to the evaluation of the SP/CAP index alone.

Another interesting measure suggested in addition to SP/CAP ratio is the latency difference between the CAP responses evoked by stimuli of opposite polarity (Orchik et al., 1998; Sass et al., 1998). This difference is significantly increased in patients with endolymphatic hydrops possibly as a result of the asymmetry in dynamics of the basilar membrane motion because of increased endolymph volume. It has been estimated that measuring this index in combination with the SP/CAP ratio results in an increase of sensitivity of up to 87%. In this regard, the above-mentioned procedure of optimal filtering could prove very useful for performing the cancellation of CM while preserving the shape of both SP and CAP components for a given stimulus polarity.

A further promising technique relies on reduced modulation of SP amplitude induced by the presentation of a continuous tone in combination with tone-burst stimuli at a frequency of 1 kHz (Iseli and Gibson, 2010). In this stimulation paradigm the increased tension of the basilar membrane because of the endolymphatic hydrops decreases the bias induced by tone presentation which is usually found in ears with normal hearing. This measure has proved to be more sensitive in detecting endolymphatic hydrops compared to the classical SP/CAP index. Sensitivity improves by up to 95% when using this index in combination with the absolute SP amplitude measured on ECochG responses evoked by tone-burst stimulation.

One of the main drawbacks of the measures proposed as alternative to, or in combination with, SP/CAP ratio is that these procedures are not easily performed. Therefore, the measure of SP/CAP index in response to high-intensity clicks remains the most popular index used to support the diagnosis of Ménière disease because of the high level of specificity, simplicity in stimulus generation, and reliability in identification of both SP and CAP.

ECochG POTENTIALS IN AUDITORY NEUROPATHY

AN is a hearing disorder characterized by disruption of temporal coding of acoustic signals in auditory nerve fibers resulting in impairment of auditory perceptions relying on temporal cues (Starr et al., 2008). In the healthy cochlea,

dynamics of activation of both postsynaptic membrane and auditory nerve fibers are well suited for fast and precise signal transmission. At the presynaptic level, temporal precision of acoustic signaling is guaranteed by the fast kinetics of synaptic release, which is triggered by calcium influx through one or two calcium channels, tight coupling of these channels to the vesicle release sites, and parallel release of multiple vesicles through the ribbon synapses. High rates of glutamate release lead in turn to the generation of excitatory postsynaptic potentials (EPSPs) which trigger spike initiation in the auditory nerve fibers through the activation of AMPA receptors. The latter have low affinity for glutamate, which results in quick times of activation and deactivation of postsynaptic membrane thus ensuring preservation of nerve fiber sensitivity in spite of the high amounts of neurotransmitter released in the synaptic cleft. At the level of auditory nerve, both the velocity and precision of spike initiation and propagation are guaranteed by the abundance of Nav1.6 channels and their strategic disposition along the nerve fibers.

The disruption of any one of these mechanisms impairs the precision of temporal coding of acoustic signals. For example, at the presynaptic level, impairment of multivesicular release results in reduction of neurotransmitter availability at the synaptic cleft with generation of small EPSPs with abnormal morphology and dispersed in time. At the postsynaptic level, decrease in the number of auditory nerve fibers and demyelination of spared axons result in a reduction of auditory input directed to the central nervous system and slowed conduction velocity in residual fibers. In both presynaptic and postsynaptic disorders abnormal synchrony of auditory nerve activity underlies the profound alterations of ABRs and impairment of speech perception, both occurring in the presence of normal physiological measures of OHC activities (OAEs and CM). With regard to CM amplitudes, they proved to be within normal limits in patients with AN (Santarelli et al., 2006a). Indeed, mean CM amplitudes calculated for a sample of 11 AN patients with several etiologies (Figure 12.9), albeit showing a high degree of variability, proved to be not significantly different from the corresponding values obtained for normal controls.

Alterations of auditory nerve synchrony may result from genetic disorders or from a wide range of other etiologies (infectious, toxic-metabolic, immunologic) (Santarelli et al., 2006b). However, definite etiologic factors can be identified in about half of patients with AN (Starr et al., 2008). Because of the high variability of etiologic factors and related pathophysiological mechanisms, the term "auditory neuropathies" would be more appropriate (Santarelli et al., 2013). The most well-known forms of AN are the result of genetic diseases and may present as isolated hearing disorders or associated with multisystem involvement (Santarelli, 2010). In general, the majority of isolated AN forms are associated with presynaptic mechanisms, whereas AN disorders with multisystem involvement are invariably subtended by postsynaptic mechanisms and are most commonly associated with peripheral and optic neuropathies.

Among the AN forms unrelated to genetic etiology, the most frequently observed are those possibly affecting children discharged from the neonatal intensive care unit (NICU). It has been estimated that about 5.6% of children failing newborn hearing screening show abnormal ABRs and presence of OAEs consistent with the electrophysiological profile of AN, the majority being discharged from the NICU. Moreover, postmortem examination carried out on temporal bones of deceased neonates has revealed selective IHC loss with high frequency in premature infants compared to full-term babies. On the basis of this finding IHC loss was suggested as a hallmark of AN (Amatuzzi et al., 2011).

In the last decade, the use of ECochG recording has been gaining importance in the diagnosis of AN for it makes it possible to define the details of both receptor and neural responses in the various forms of the disorder (McMahon et al., 2008; Santarelli and Arslan, 2002; Santarelli et al., 2008). Such information may prove extremely valuable in defining objectively the site of auditory neural dysfunction and to shed light on the underlying pathophysiological mechanisms to plan appropriate rehabilitative strategy.

Several ECochG patterns have been identified in response to click stimuli (Santarelli et al., 2008, 2013). In general, IHCs lesions are expected to result primarily in SP abnormalities whereas disorders affecting ribbon synapsis and auditory nerve fibers would induce changes of CAP parameters and morphology with preservation of SP amplitude and latency. Nevertheless, it should be noted that different ECochG patterns can be intermixed because of the possible coexistence of pre- and postsynaptic lesions in some forms of AN particularly at an advanced stage.

In one of the most frequently observed ECochG patterns the response consists of a prolonged negative deflection showing no separation between SP and CAP components. Figure 12.12 (left side) shows an example of ECochG responses recorded in AN at decreasing stimulus levels superimposed on the corresponding traces obtained from one normally hearing control. These recordings were obtained from one adult patient with isolated AN of unknown etiology which had started in adolescence. It can be seen that the SP–CAP complex recorded in the normal control was replaced in the AN patient by a negative potential which appeared to be markedly attenuated in amplitude, increased in duration, and delayed in peak latency.

A second pattern frequently observed in AN consists of the receptor SP potential with normal amplitude and peak latency followed by the sustained negative response. In some subjects a small CAP with delayed peak latency is superimposed on the prolonged potential at high stimulus levels. An example is shown on the right side of Figure 12.12 for an 11-year-old boy presenting with a familiar form of isolated

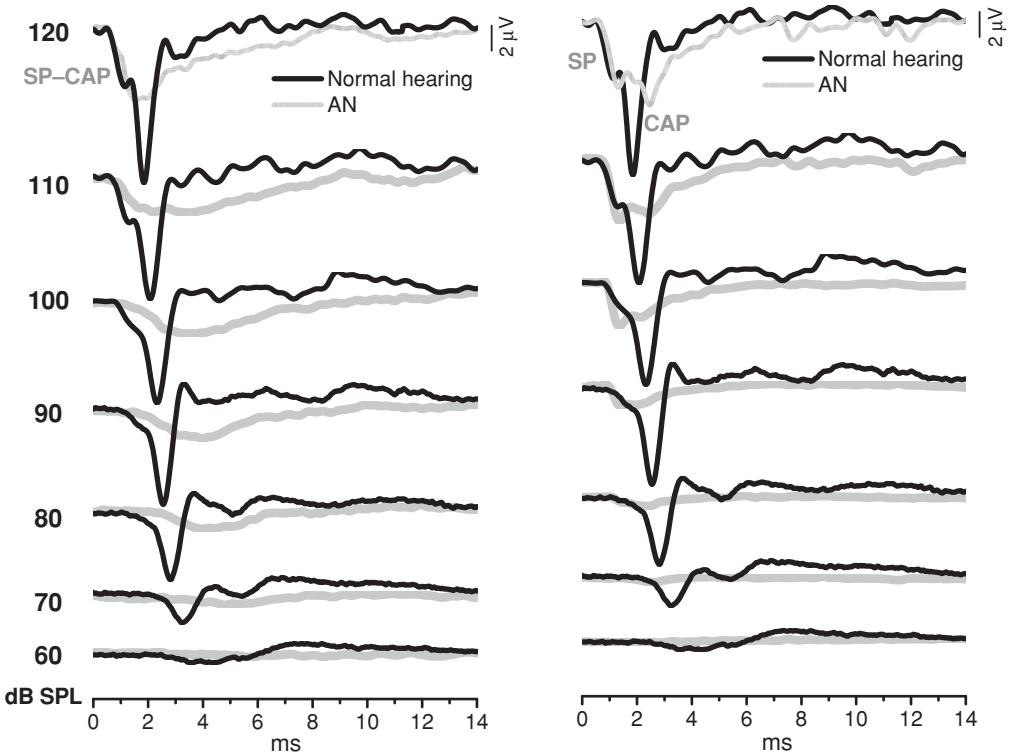


FIGURE 12.12 ECochG responses from two patients with auditory neuropathy (AN). ECochG waveforms recorded from two representative subjects with AN (*gray line*) are reported for decreasing stimulus levels superimposed on the corresponding traces obtained from one normally hearing ear (*black line*). In the example illustrated in the **left panel** ECochG potentials found in the normal control are replaced by an attenuated prolonged negative response showing no separation between SP and CAP components. ECochG recordings from the second patient (**right panel**) showed that the receptor SP is followed by a sustained negative potential with a superimposing small CAP at high stimulus intensity.

AN of unknown etiology. This ECochG pattern showing preservation of SP followed by the prolonged potential is highly suggestive of a peripheral disorder associated with preservation of IHC activation.

A less frequently observed ECochG pattern consists of only the SP component not followed by the prolonged activity. This may indicate preservation of IHC function not followed by nerve fiber activation.

To compare the ECochG responses recorded from AN patients with normal healthy controls, the whole ECochG response was considered as a single event (SP–CAP) because SP and CAP cannot be identified separately in about half of AN patients (Santarelli and Arslan, 2013; Santarelli et al., 2008). Mean measures of SP–CAP peak latency, amplitude, and duration calculated for a sample of 11 AN patients with several etiologies are reported as a function of stimulus intensity in Figure 12.13 and superimposed on the corresponding values calculated for normally hearing subjects. Compared to subjects with normal hearing, SP–CAP potentials recorded from patients with AN appear significantly decreased in amplitude, delayed in peak latency, and increased in duration.

One problem arising with the prolonged potentials recorded in AN patients is whether they originate from neural or receptor activation. With this in mind, we used an adaptation procedure that preferentially attenuates the neural responses with minor changes in SP amplitude (Santarelli and Arslan, 2013; Santarelli et al., 2008, 2013).

What happens in ears with normal hearing is that decreasing the time interval between successive stimuli results in attenuation of CAP amplitude. These changes are likely to result from the complex interaction of several phenomena such as velocity of neurotransmitter release and reuptake, sensitivity of postsynaptic receptors, and refractory properties of auditory nerve fibers, with all of these factors interacting in a complex way and contributing differently to adaptation when changing stimulus level and repetition rate. When using trains of clicks presented at an intensity higher than 60 dB nHL, CAP amplitude progressively decreases for interstimulus intervals lower than 500 ms, the amount of CAP attenuation being independent of signal intensity. One example of ECochG potentials recorded at an intensity of 110 dB peSPL from one normally hearing ear is reported in Figure 12.14. The stimulus sequence

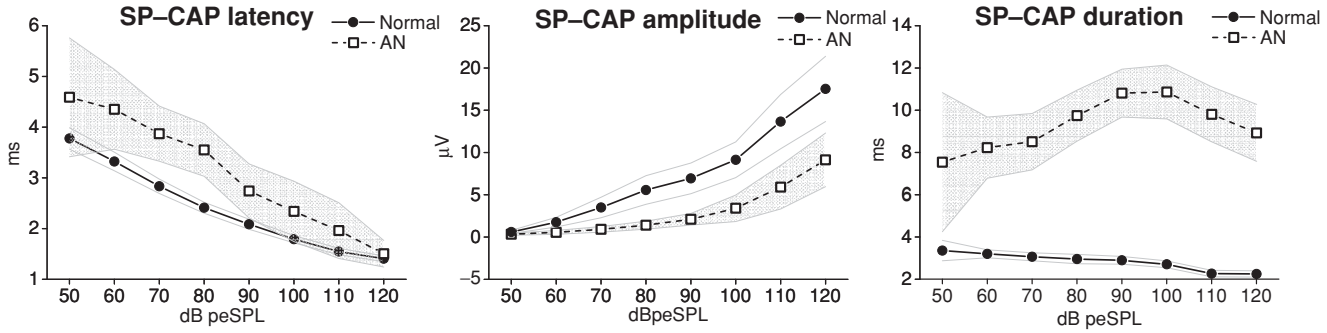


FIGURE 12.13 Intensity functions of CAP and SP potentials in patients with auditory neuropathy (AN). Mean SP–CAP latency, amplitude, and duration with 95% confidence limits (*shadowed area*) are reported as a function of stimulus intensity for a group of patients with AN (11 subjects) superimposed on the corresponding values calculated for normally hearing controls (same sample as in Figure 12.6). Since in the majority of AN patients SP and CAP could not be identified separately, the whole ECochG response was considered as a single event (SP–CAP). Compared to subjects with normal hearing, ECochG potentials recorded from patients with AN were significantly decreased in amplitude, delayed in peak latency, and increased in duration. (Modified with permission from Santarelli R, Arslan E. (2013) Electrocochleography. In: Celesia GG, ed. *Disorders of Peripheral and Central Auditory Processing. Handbook of Clinical Neurophysiology.* Vol 10. Amsterdam: Elsevier; pp 83–113.)

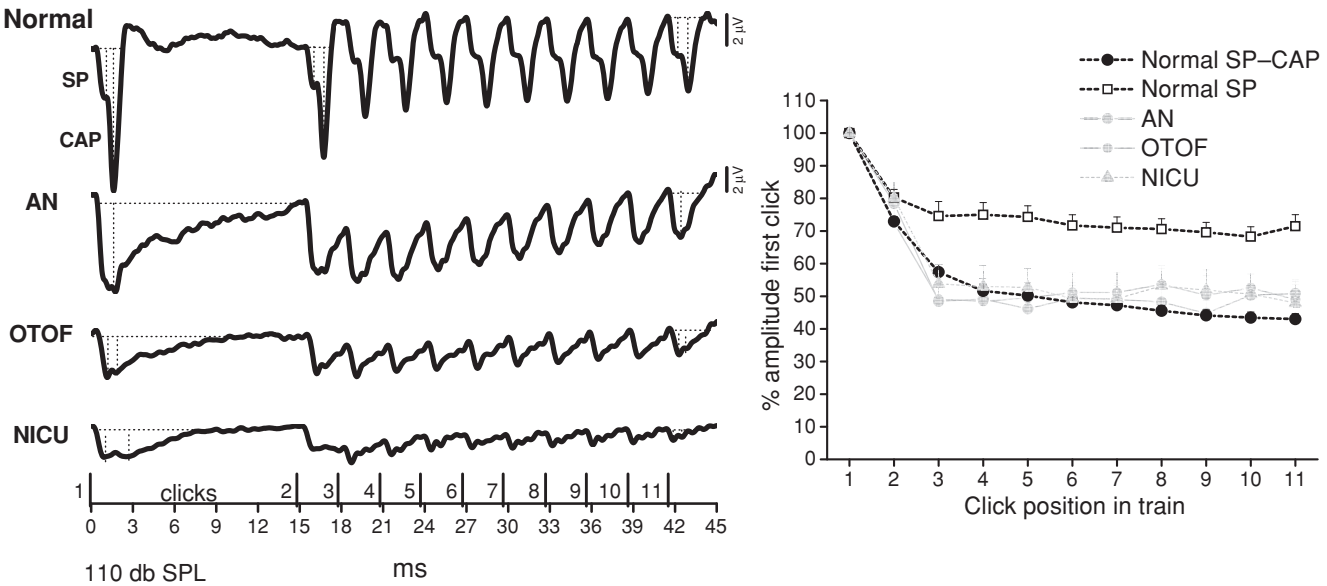


FIGURE 12.14 Adaptation of cochlear potentials. ECochG recordings obtained at 110 dB peSPL in response to the stimulus sequence reported at the *bottom* are displayed in the **left panel** for one normally hearing child and three AN subjects, one boy affected by a familiar form of AN of unknown etiology, and two children with one carrying biallelic mutation in the *OTOF* gene and the other showing hearing impairment related to a rocky neonatal course. In the **right panel** the means and standard errors of normalized SP–CAP amplitudes at 110 dB peSPL are reported as a function of click position in the stimulus sequence for controls and three groups of patients, one group of young adults with AN related to different etiologies (11 patients), children carrying biallelic mutation in the *OTOF* gene (8 patients), and hearing-impaired children discharged from the NICU (10 patients). In the normally hearing controls, CAP amplitude was markedly attenuated after adaptation whereas SP attenuation was much lower. In patients with AN, SP–CAP amplitudes were markedly reduced after adaptation and the amount of response attenuation was comparable with that calculated for CAP in normally hearing controls. (Modified from Santarelli R, Del Castillo I, Starr A. (2013) Auditory neuropathies and electrocochleography. *Hear, Balance Commun.* 11, 130–137.)

consists of an initial click which is followed 15 ms later by a train of 10 clicks separated by an interstimulus interval of 2.9 ms. This sequence was repeated every 191 ms. The duration of the intertrain interval is likely to affect minimally the amplitude of ECochG potentials evoked by the first click of the stimulus sequence. It can be seen that CAP amplitude shows a marked attenuation from the first (1 in Figure 12.14) to the second (2 in Figure 12.14) click of the stimulus sequence, which corresponds to the first click of the high-rate train (ISI 15 ms). Then, further decrease in CAP amplitude is observed during the following 3 to 4 clicks of the train sequence because of further increase of stimulus repetition rate (ISI 2.9 ms). In contrast, SP amplitudes show little or no attenuation from the first to the eleventh click of the stimulus sequence. In this particular example, the amount of attenuation was 50% and 22% for CAP and SP components, respectively. Looking at the means of CAP and SP amplitudes in Figure 12.14, calculated for a group of normally hearing children (same sample as in Figure 12.4), it appears that CAP amplitude drops by about 30% from the first to the second click and shows an additional attenuation within the following 2 to 3 clicks thus attaining a reduction of 60% by the end of the stimulus sequence. The amount of mean SP attenuation is much smaller compared to CAP as the decrease in amplitude from the first to the last click of the sequence reaches 30%.

Examples of ECochG recordings obtained from AN patients in response to high stimulation rate are compared in Figure 12.14 to the responses collected from normal controls. The upper ECochG waveform refers to one boy affected by a familiar form of AN of unknown etiology. ECochG potentials showed no separation between SP and CAP components and were considerably prolonged in duration compared to the normal response. Stimulation at high rate induced a remarkable attenuation of the whole SP–CAP potential with an overall amplitude reduction of 52%. Mean attenuation amplitudes calculated across 11 AN patients (same sample as in Figure 12.13) proved to be comparable to the corresponding values calculated for normal controls. Therefore, the prolonged responses recorded from patients with AN are likely to reflect the activation of auditory nerve fibers. Broadening and attenuation of ECochG potentials reflect impairment of neural fiber synchrony with reduced probability of summation of unitary activities. Thus, the disruption in synchrony may be related to abnormal IHC activation in those AN forms associated with abnormal SP or result from a reduced number of auditory neurons and alteration of conduction velocity in spared auditory fibers when the prolonged potentials follow a normal SP. A lesion selectively involving the terminal unmyelinated portion of auditory nerve fibers has been hypothesized for AN patients affected by optic atrophy because of a mutation in the *OPA1* gene (Huang et al., 2009).

In conclusion, information provided by ECochG recordings are relevant in that they help to identify the lesion site by distinguishing the AN disorders involving IHCs from neural disorders associated with normal SP responses. This information may be of value in predicting the outcome of cochlear implantation since a good outcome is expected in cochlear implant recipients affected by AN disorders involving IHCs, whereas the benefits of cochlear implantation in patients showing neural forms of AN are critically dependent on number and function of spared auditory neurons.

One of the most well-known forms of congenital AN is due to mutations in the *OTOF* gene (DFNB9) with a recessive pattern of inheritance. The *OTOF* gene encodes otoferlin, a transmembrane protein belonging to the ferlin protein family, which plays a crucial role in fast vesicle release at the synapse between IHCs and auditory nerve fibers. It has also been implicated in vesicle replenishment at the synaptic pole of IHCs. To date, more than 50 pathogenic mutations of the *OTOF* gene have been identified, the majority of which are inactivating mutations, which result in a very homogeneous phenotype of profound hearing loss. Over 50% of children carrying biallelic mutations in the *OTOF* gene show preservation of OHC function as indicated by OAE recording, together with absent or high-threshold ABRs.

Examples of ECochG waveforms recorded from five children carrying biallelic mutations in the *OTOF* gene are displayed in Figure 12.15 for 120 dB peSPL intensity superimposed on the grand average of recordings obtained from 26 normally hearing children. The ECochG waveform begins with the SP showing normal amplitude and peak latency. This is followed by a prolonged negative response similar to the broad negative potential recorded in other forms of AN (Santarelli and Arslan, 2013; Santarelli et al., 2009). In some patients, a small CAP is superimposed on this prolonged activity at high stimulus intensity. Interestingly, the sustained potentials are recorded as low as 60 dB peSPL in spite of the profound hearing loss, which was estimated on the basis of visual-reinforced audiometry. The use of the neural adaptation procedure yielded attenuation values similar to those calculated for normal hearing and for other forms of AN (Figure 12.14, means of eight patients), thus indicating that this prolonged activity is generated by auditory nerve fibers (Santarelli and Arslan, 2013; Santarelli et al., 2009).

The normal amplitude and latency of SP potentials recorded in patients carrying mutations in the *OTOF* gene point to preserved activation of IHCs. The sustained response following the SP may result from the dendritic potentials arising in the distal portion of the afferent fibers. It has been hypothesized that a reduction of otoferlin activity leads to abnormal function of the synaptic ribbons with abolition of the fast phase of exocytosis and impairment of multivesicular release. This results in generation of small EPSPs with abnormal morphology and dispersed in time with consequent decrease in synaptic reliability. Indeed,

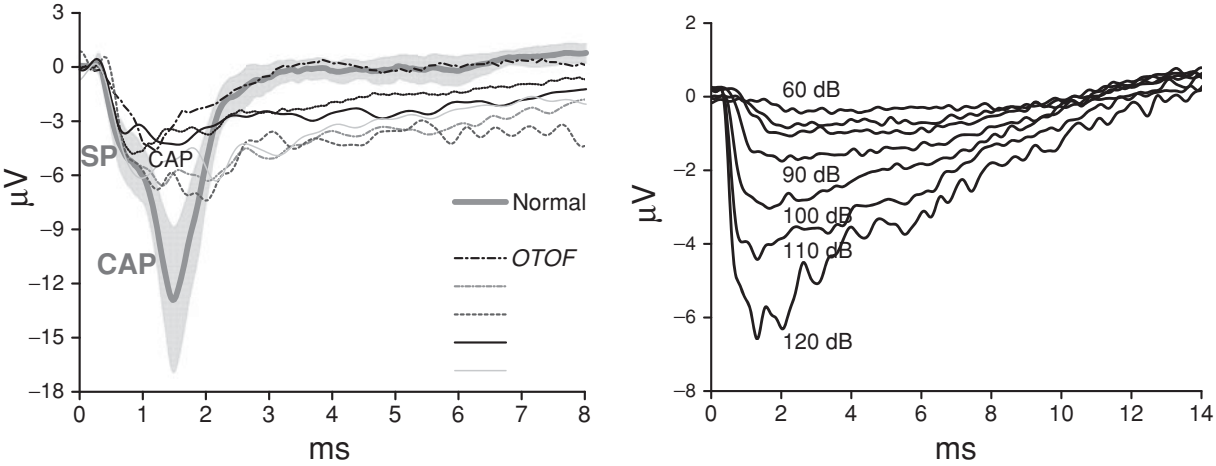


FIGURE 12.15 ECochG potentials recorded from children carrying biallelic mutation in the *OTOF* gene. Cochlear potentials recorded at 120 dB peSPL are reported on the **right side** superimposed on the grand average of the corresponding waveforms obtained from 26 normally hearing ears with 95% confidence limits [*shadowed area*]. In children with mutations in the *OTOF* gene ECochG responses begin with a normal SP followed by a prolonged low-amplitude negative potential. This response is identifiable as low as 60 dB peSPL, which is about 50 dB lower than behavioral threshold [**right panel**]. [Reprinted from Santarelli R, Del Castillo I, Starr A. (2013) Auditory neuropathies and electrocochleography. *Hear, Balance Commun;* 11, 130–137.]

small EPSPs elicited at low stimulus intensity are not followed by spike generation whereas at high intensity, EPSPs may occasionally reach the threshold to trigger action potentials with consequent generation of small CAPs.

These mechanisms are relevant from the point of view of rehabilitation programs for deaf children carrying mutations in the *OTOF* gene. In these patients an excellent outcome of cochlear implantation is expected with restoration of auditory function because auditory nerve fibers are believed not to be primarily involved by the damage related to otoferlin dysfunction.

ECochG POTENTIALS IN PATIENTS AFFECTED BY CNS DISORDERS

The predictive ability of ABRs in estimating hearing thresholds in some categories of children has since 1984 been questioned by Kraus et al. (1984). They showed that hearing thresholds obtained by conventional audiometry in infancy were better than those estimated on the basis of neonatal ABRs in 15% of their sample of children showing ABR abnormalities. Since the clinical history of these patients was suggestive of prematurity, respiratory distress, and hyperbilirubinemia, they hypothesized that these factors depressed brainstem electrical activity with consequent interference in ABR generation. In recent years, several authors have pointed out that the depression of brainstem electrophysiology affects both amplitude and latency of ABR waves, with changes in amplitude being more pronounced than prolongation in latency. Specifically, decreased wave V amplitude, missing waves, and prolonged I–V interval have been reported in children with cerebral palsy (Jiang et al., 2011). Moreover, full-term neonates suffering from hypoxia-ischemia have shown a decrease in amplitude of ABR waves as evaluated by the maximum-length sequence (MLS) technique (Jiang et al., 2013). The reduction was more pronounced for wave V compared to earlier components possibly as a result of predominant involvement of the rostral part of the brainstem. In contrast, neonates with perinatal conditions other than hypoxia-ischemia display a reduction in ABR amplitude which mainly affects earlier components.

In addition to these findings, several studies have found discrepancies between CAP and ABR thresholds in children showing CNS pathology possibly associated with depression of brainstem electrical activity (Arslan et al., 1997; Ryerson and Beagley, 1981). Figure 12.16 displays ABR and ECochG waveforms recorded from three children showing such threshold discrepancies. The first case refers to a 4-year-old child who was born with signs of an as yet unidentified clinical syndrome who had suffered from severe neonatal asphyxia. At the time of our first observation the child presented with tetraparesis, absence of language, and severe mental retardation. He had been diagnosed with profound hearing loss on the basis of ABR recording and was fitted with power hearing aids. Parents requested evaluation at our hospital because the child began crying every time they attempted to force him to wear hearing aids. ABRs evoked by click stimuli showed no components except for wave I which was recorded as low as 70 dB nHL bilaterally. CAP was identified in ECochG recordings with normal amplitude and latency as low as 50 dB peSPL (corresponding to

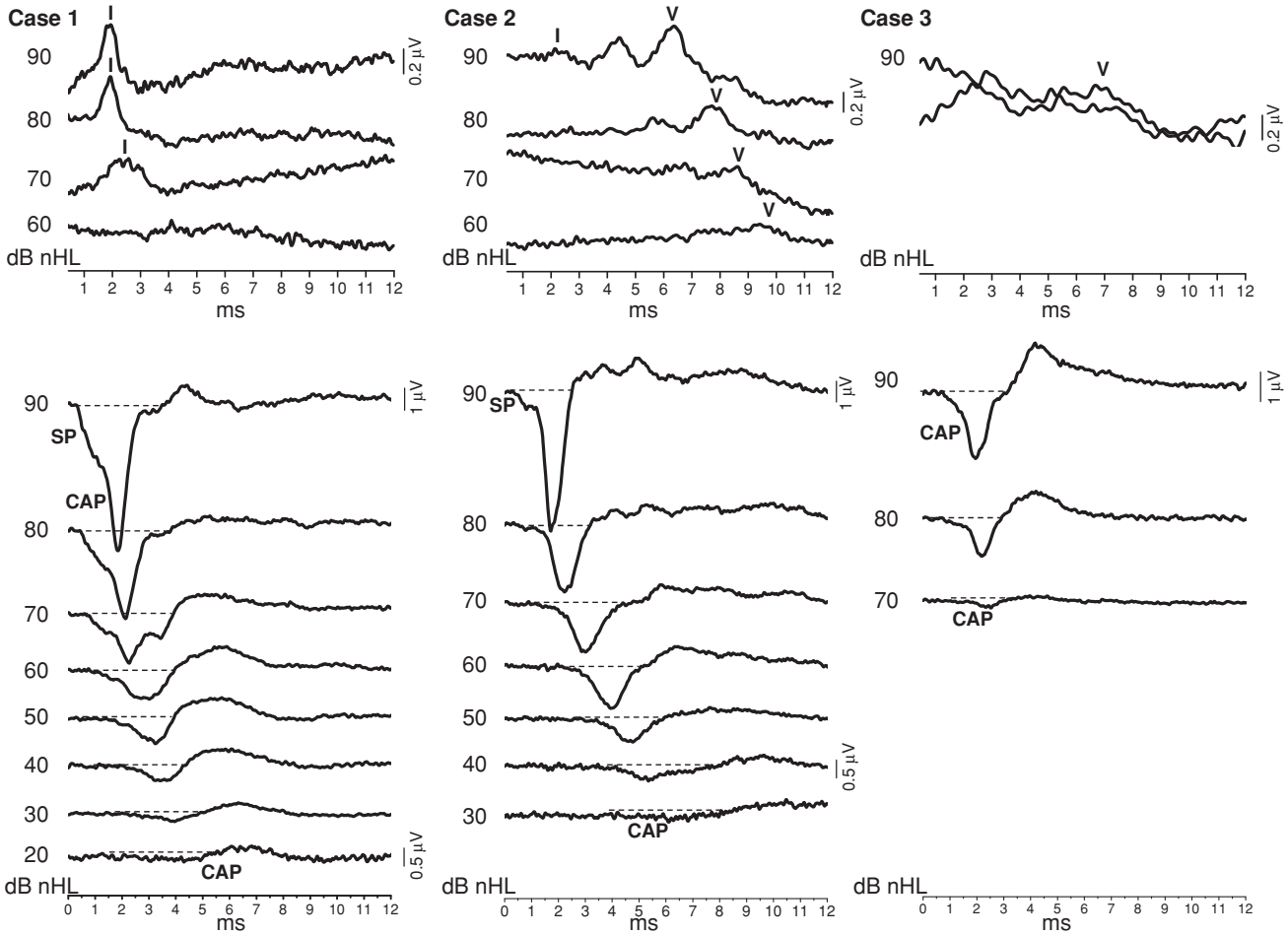


FIGURE 12.16 ABR and ECochG potentials from children with CNS disorders. ABR and ECochG potentials evoked by clicks are shown at decreasing stimulation intensities for three children with CNS disorders. Intensity has been expressed in dB nHL to facilitate the comparison between ABR and ECochG thresholds. Case 1 (**left panel**) refers to a 4-year-old boy with tetraparesis, severe mental retardation, and absence of language. Only wave I was identified in ABR recordings whereas CAP was recorded with normal threshold, latency, and amplitude. ABR and ECochG recordings displayed in the **middle panel** were collected from a 10-year-old girl affected by Wolf–Hirshhorn syndrome who presented with tetraparesis, severe mental retardation, and absence of language. Wave V threshold was 60 dB nHL in the better ear whereas CAP was identified in the ECochG waveforms as low as 30 dB nHL with increased latency because of the coexistence of conductive hearing loss. In the example reported in the **right panel,** ABRs were recorded with 90 dB nHL threshold from an 8-month-old child who was born premature and suffered from sepsis and bowel occlusion. ECochG recordings showed a small CAP with 70 dB nHL threshold.

20 dB nHL) in both ears thus indicating a normal hearing capacity. This example demonstrates that in subjects with signs of CNS pathology the reliability of ABR in hearing threshold assessment may be markedly reduced.

In the second example, ABR and ECochG recordings were collected from a 10-year-old girl affected by Wolf–Hirshhorn syndrome who presented with tetraparesis, severe mental retardation, and absence of language. Wave V threshold in the better ear was 60 dB nHL and thus she was fitted with hearing aids bilaterally. The girl underwent ECochG recording because she refused to use amplification. CAP was identified in the ECochG waveforms recorded from the better ear as low as 30 dB nHL, which indicates that CAP threshold was 30 dB better than that estimated on the basis of ABR recordings. Moreover, CAP latency–intensity function suggested conductive hearing loss, which was in accordance with otoscopic and tympanometric signs of tympanosclerosis. Even in this case, CNS pathology hampered ABR generation with consequent reduction of wave V reliability in hearing threshold estimation. Moreover, the coexistence of conductive hearing loss could have contributed to further decreasing the synchronous firing of ABR generators at low intensity through attenuation of the auditory input to the brainstem.

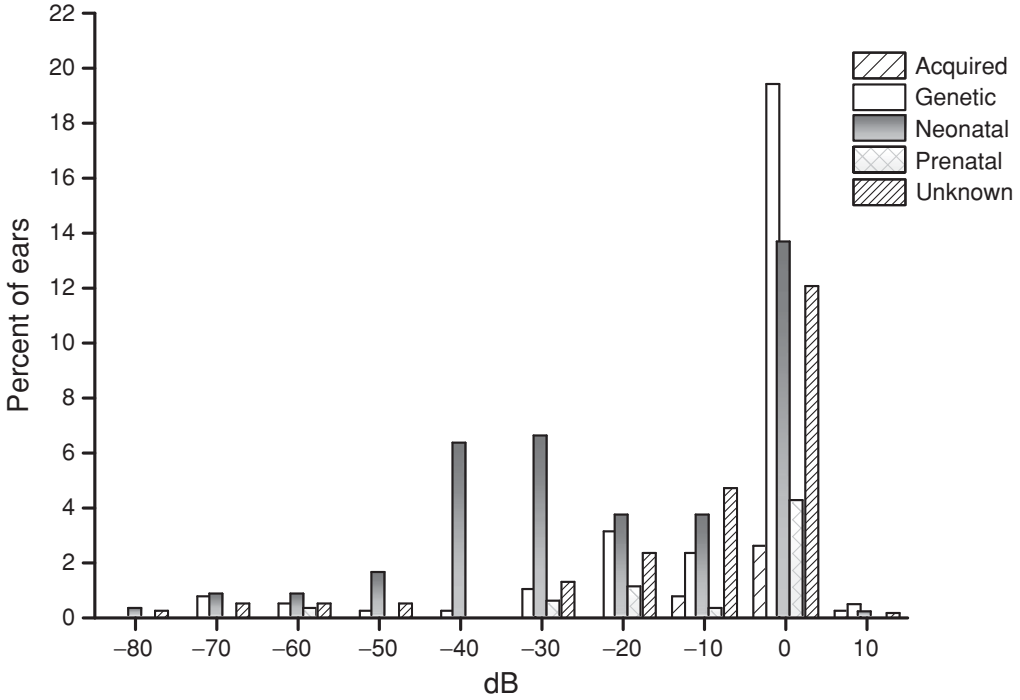


FIGURE 12.17 Comparison between wave V and CAP thresholds. The graph reports the distribution of differences between ECochG and ABR thresholds as evaluated for 597 ears in 302 children submitted to ECochG and ABR recording in the same testing session at the Treviso Regional Hospital from 2000 to 2012. Patients have been grouped according to etiology or risk factors for hearing loss. Negative values indicate that ECochG thresholds are better than ABR thresholds. Differences between ABR and ECochG thresholds higher than 10 dB were found in 34% of all ears. [Modified with permission from Santarelli R, Arslan E. [2013] Electrocochleography. In: Celesia GG, ed. *Disorders of Peripheral and Central Auditory Processing. Handbook of Clinical Neurophysiology.* Vol 10. Amsterdam: Elsevier; pp 83–113.]

Case 3 refers to an 8-month-old child who was born premature and suffered from sepsis and bowel occlusion. OAEs were absent and ABRs in response to clicks were recorded with 90-dB nHL threshold bilaterally. ECochG recordings showed a small CAP, which was recorded at an intensity as low as 70 dB nHL in the right ear and 60 dB nHL in the left ear revealing a 20- to 30-dB difference between ABR and ECochG thresholds. These findings have also been considered as reflecting sustained depression of brainstem auditory function.

Figure 12.17 illustrates the distribution of differences between wave V and CAP thresholds calculated for 302 children (597 ears) who underwent ABR and ECochG testing at our department from 2000 to 2012 (Santarelli and Arslan, 2013). Ears have been grouped into several classes on the basis of etiology or risk factors for hearing loss. The larger group consists of children discharged from NICU. Other etiologies were genetic (isolated or syndromic hearing loss), prenatal (mostly CMV infections), acquired (mostly meningitis), and unknown. Negative values indicate that ECochG thresholds are better than ABR thresholds. Differences in threshold estimation between ABR and ECochG recordings higher than 10 dB were found in 34% of all ears. Interestingly, 21% were from infants discharged from the NICUs. Therefore, relying on the identification of ABR wave V for the assessment of hearing sensitivity in this group of children would have led to an overestimation of hearing loss with inappropriate choice of rehabilitative strategy. Nevertheless, it should be pointed out that this sample also includes children for whom threshold discrepancies could also be related to abnormal firing of auditory nerve fibers (see the paragraph following).

In conclusion, ECochG recordings are mandatory for hearing threshold assessment in uncooperative children presenting with neurologic disorders and abnormal ABRs.

ECochG POTENTIALS IN CHILDREN DISCHARGED FROM THE NEONATAL INTENSIVE CARE UNIT

Infants discharged from the NICU are at increased risk for sensory/neural hearing loss because of the exposure to many risk factors such as prematurity, low birth weight, respiratory distress, anemia, noise, and ototoxic drugs. The combined

effects of all risk factors result in variable amounts of damage involving OHCs, IHCs, or both. As a consequence, hearing-impaired infants discharged from the NICU present with variable degrees of sensory/neural hearing loss possibly related to specific audiologic pictures such as AN. Indeed, about 5.6% of children failing newborn hearing screening show abnormal ABRs and presence of OAEs consistent with the electrophysiological profile of AN.

As mentioned above, a reduced correlation between ABR and hearing thresholds has been well documented in premature babies and in those suffering perinatal asphyxia as a consequence of abnormal firing of brainstem generators of ABRs which retain little or no connection with the dynamics of auditory periphery activation. For this reason, ECochG recordings in children discharged from the NICU showing abnormal ABRs are mandatory if hearing thresholds cannot be ascertained reliably through noninvasive procedures. On the other hand, abnormal ABRs may also result from alterations of auditory nerve firing because of disruption of temporal coding of acoustic signals in auditory nerve fibers. Indeed, postmortem examination carried out on temporal bones of deceased neonates has shown extensive hair cell damage with a higher frequency of selective IHC loss in premature infants compared to full-term babies (Amatuzzi et al., 2011). Since the electrophysiological profile of AN has been found with high frequency in premature infants, the dysfunction of IHCs has been proposed as the primary mechanism underlying AN in children discharged from the NICU. Recently, the use of intratympanic ECochG recording has been proposed for this category of patients to define the details of cochlear potentials and, in general, to evaluate hearing threshold for rehabilitative purposes (Santarelli and Arslan, 2013).

ECochG potentials recorded at decreasing stimulus levels from one representative infant discharged from the NICU who showed the electrophysiological profile of AN are displayed in Figure 12.18 superimposed on the corresponding recordings obtained from one normally hearing child. The clinical history was suggestive of prematurity, hypoxia, and hyperbilirubinemia. At high stimulus intensity ECochG waveforms display the SP potential with reduced amplitude compared to the normally hearing child. This is followed by a negative response showing reduced amplitude, delayed peak latency, and markedly increased duration compared to the normally hearing subject. Stimulation at high rate (Figure 12.14, mean of 10 children) was consistent with the prolonged potentials being generated by neural rather than receptor elements since the size of attenuation after adaptation was similar to that calculated for subjects with normal hearing and other forms of AN. Thus, the ECochG profile obtained from AN children discharged from the NICU is indistinguishable from the most common pattern observed in patients with other forms of AN. Specifically, since SP amplitude calculated for these infants (12 children, mean SP amplitude 4 ± 3.3 μV) is about half that obtained from

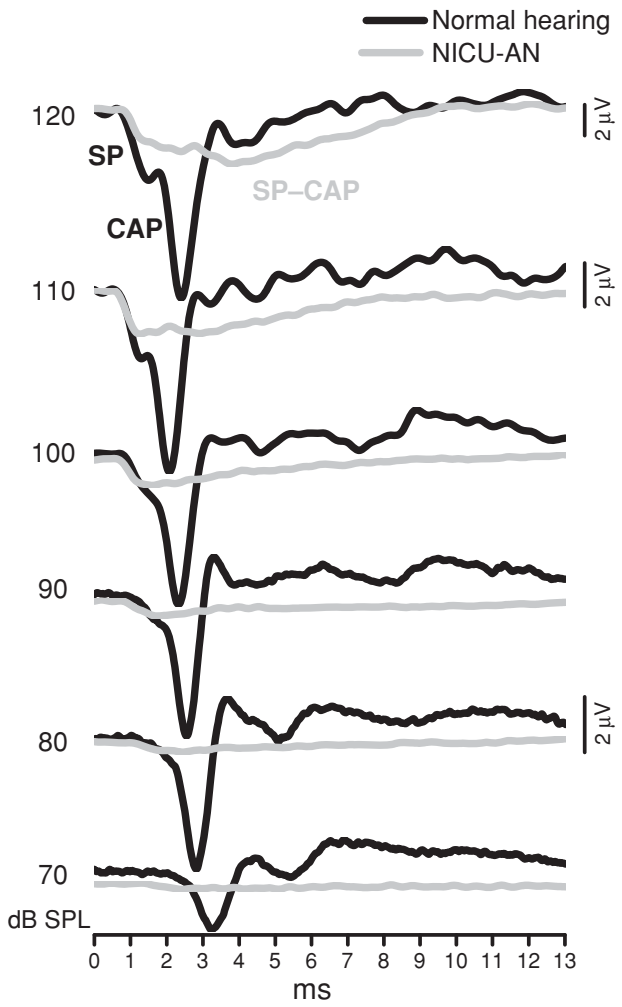


FIGURE 12.18 ECochG responses recorded from one child discharged from the NICU showing the electrophysiological picture of auditory neuropathy (AN). ECochG waveforms (*black line*) are superimposed on the corresponding traces recorded from one normally hearing ear (*gray line*) in response to clicks at decreasing stimulus levels. ECochG responses recorded in the AN child showed the SP with reduced amplitude compared to the normally hearing control, which was followed by a prolonged negative response similar to that obtained in other forms of AN. (Reprinted from Santarelli R, Del Castillo I, Starr A. (2013) Auditory neuropathies and electrocochleography. *Hear, Balance Commun.* 11, 130–137.)

normally hearing controls, alteration of auditory nerve fiber discharge seems to be primarily related to a receptor rather than a neural disorder. This result appears to be in accordance with the finding of a reduced number of IHCs in the cochlea of premature infants.

Low-amplitude prolonged negative potentials were also recorded from infants discharged from the NICU who showed abnormal ABRs and no OAEs. Specifically, of the 59 children from NICU showing abnormal ABRs and absent OAEs who have been submitted to ECochG recordings at our

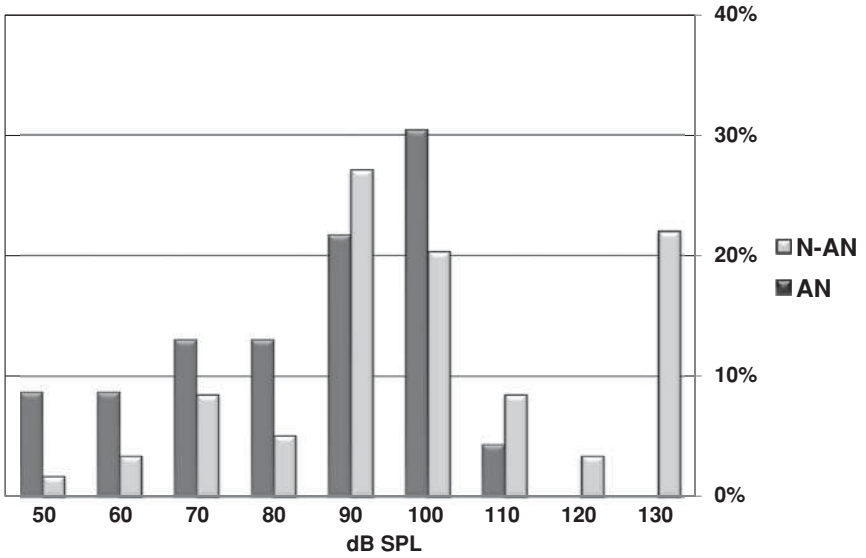


FIGURE 12.19 Distribution of CAP thresholds in two groups of children discharged from the NICU, one showing OAEs and absent ABRs (AN) and the other showing no OAEs (N-AN). Children with the profile of AN had normal or moderately elevated ECochG thresholds, whereas higher CAP thresholds were found in the N-AN group. (Reprinted from Santarelli R, Del Castillo I, Starr A. (2013) Auditory neuropathies and electrocochleography. *Hear, Balance Commun.* 11, 130–137.)

department between 2001 and 2011, 61% showed prolonged responses with no separation between SP and CAP, 13% had distinct SP and CAP associated with an increase of response duration, whereas 25% showed no response at the maximum stimulation intensity (120 dB peSPL). Therefore, the ECochG pattern observed in the majority of these children seems indistinguishable from that obtained from the group of infants with the electrophysiological profile of AN.

Figure 12.19 reports the distribution of ECochG thresholds in the two samples of children discharged from the NICU, one with OAEs (AN, 25 children) and the other without OAEs (N-AN, 59 children). It can be seen that patients with the AN profile show normal or moderately elevated CAP thresholds, whereas higher CAP thresholds were found in children showing no OAEs. These differences may reflect different mechanisms and extension of damage underlying hearing impairment, which is consistent with the high variability of risk factors acting in the NICU. In this view, the distribution of CAP thresholds may reflect a "spectrum" of lesions resulting from different amounts of IHC and OHC loss, synaptic damage, and depression of auditory nerve fiber activity. Independent of OHC function as indicated by OAE detection, abnormal SPs followed by the prolonged potentials are likely to be associated with IHC disorders, whereas an ECochG pattern showing normal SPs followed by the sustained responses may result from synaptic dysfunction or is underlain by damage to the terminal dendrites as suggested for some forms of AN in humans or for noise-induced trauma in guinea pigs.

CONCLUSION

From the point of view of diagnostic evaluation, ECochG recordings proved to be useful for specific applications such as the diagnosis of Ménière disease. More importantly, the use of ECochG potentials could help bring together knowledge from basic research and clinical evaluation as the combination of ECochG recordings with other techniques may contribute to defining the dysfunction in the auditory periphery and to localizing the underlying lesion. This could provide crucial information in the identification of abnormal cochlear responses and may assist in the choice of rehabilitative strategy, particularly in specific categories of children such as those affected by CNS disorders or discharged from the NICU. Moreover, the identification of specific ECochG patterns possibly associated with defined gene mutations may elucidate both site and mechanisms underlying the various forms of AN. Finally, the inclusion of ECochG recordings in the protocol for candidacy for cochlear implantation in children could assist in the assessment of the amount of useful residual hearing for acoustic amplification.

FOOD FOR THOUGHT

1. Do you think that CM detection in surface recording can be considered as a reliable hallmark of AN in subjects with abnormal ABRs?
2. Could intratympanic ECochG recordings be proposed as the procedure of choice for assessing hearing thresholds in uncooperative children showing associated disabilities and abnormal ABRs?
3. Would you include intratympanic ECochG recordings in the standard protocol for diagnosing AN?

DEDICATION

This chapter is dedicated to the memory of Prof. Edoardo Arslan who recently passed away.

He made a substantial contribution to improve knowledge in clinical electrophysiology and had long-standing influence in the field of transtympanic electrocochleography.

He set up most of the electrophysiological apparatus used to record electrocochleography in our laboratory and inspired the interpretation of the findings reported in this chapter with continuous critical discussion.

KEY REFERENCES

thePoint A full list of references for this chapter can be found at http://thePoint.lww.com. Below are the key references for this chapter.

Amatuzzi M, Liberman MC, Northrop C. (2011) Selective inner hair cell loss in prematurity: a temporal bone study of infants from a neonatal intensive care unit. *J Assoc Res Otolaryngol.* 12, 595–604.

Aran JM, Charlet de Sauvage R, Pelerin J. (1971) Comparaison des seuils électrocochléographiques et de l'audiogramme. Etude statistique. *Rev Laryngol Otol Rhinol (Bord).* 92, 477–491.

Arslan E, Prosser S, Conti G, Michelini S. (1983) Electrocochleography and brainstem potentials in the diagnosis of the deaf child. *Int J Pediatr Otorhinolaryngol.* 5, 251–259.

Arslan E, Turrini M, Lupi G, Genovese E, Orzan E. (1997) Hearing threshold assessment with auditory brainstem response (ABR) and ElectroCochleoGraphy (ECochG) in uncooperative children. *Scand Audiol Suppl.* 46, 32–37.

Aso S, Gibson WP. (1994) Electrocochleography in profoundly deaf children: comparison of promontory and round window techniques. *Am J Otol.* 15, 376–379.

Cheatham MA, Naik K, Dallos P. (2011) Using the cochlear microphonic as a tool to evaluate cochlear function in mouse models of hearing. *J Assoc Res Otolaryngol.* 12, 113–125.

Chertoff M, Lichtenhan J, Willis M. (2010) Click- and chirp-evoked human compound action potentials. *J Acoust Soc Am.* 127, 2992–2996.

Durrant JD, Wang J, Ding D, Salvi R. (1998) Are inner or outer hair cells the source of summating potentials recorded from the round window? *J Acoust Soc Am.* 104, 370–377.

Eggermont JJ. (1974) Basic principles for electrocochleography. *Acta Otolaryngol Suppl.* 316, 7–16.

Eggermont JJ. (1976) Electrocochleography. In: Keidel WD, Neff WD, eds. *Handbook of Sensory Physiology. Auditory System. Clinical and Special Topics.* New York: Springer; pp 625–706.

Elberling C. (1976) Simulation of cochlear action potentials recorded from the ear canal in man. In: Ruben RJ, Elberling C, Salomon G, eds. *Electrocochleography.* Baltimore, MD: University Park Press; pp 151–168.

Ferraro JA, Tibbils RP. (1999) SP/AP area ratio in the diagnosis of Ménière's disease. *Am J Audiol.* 8, 21–28.

Goldstein M, Kiang N. (1958) Synchrony of neural activity in electric responses evoked by transient acoustic stimuli. *J Acoust Soc Am.* 30, 107–114.

Huang T, Santarelli R, Starr A. (2009) Mutation of OPA1 gene causes deafness by affecting function of auditory nerve terminals. *Brain Res.* 1300, 97–104.

Iseli C, Gibson W. (2010) A comparison of three methods of using transtympanic electrocochleography for the diagnosis of Meniere's disease: click summating potential measurements, tone burst summating potential amplitude measurements, and biasing of the summating potential using a low frequency tone. *Acta Otolaryngol.* 130, 95–101.

Jiang ZD, Wu YY, Liu XY, Wilkinson AR. (2011) Depressed brainstem auditory function in children with cerebral palsy. *J Child Neurol.* 26, 272–278.

Jiang ZD, Zhou Y, Yin R, Wilkinson AR. (2013) Amplitude reduction in brainstem amplitude response in term infants under neonatal intensive care. *J Child Neurol.* 26, 272–278.

Kiang NYS, Moxon E, Kahn A. (1976) The relationship of gross potentials recorded from the cochlea to single unit activity in the auditory nerve. In: Ruben RJ, Elberling C, Salomon G, eds. *Electrocochleography.* Baltimore, MD: University Park Press; pp 95–115.

Kraus N, Ozdamar O, Stein L, Reed N. (1984) Absent auditory brain stem response: peripheral hearing loss or brain stem dysfunction? *Laryngoscope.* 94, 400–406.

McMahon CM, Patuzzi RB, Gibson WPR, Sanli H. (2008) Frequency-specific electrocochleography indicates that presynaptic and postsynaptic mechanisms of auditory neuropathy exist. *Ear Hear.* 29, 314–325.

Mori N, Asai H, Doi K, Matsunaga T. (1987) Diagnostic value of extratympanic electrocochleography in Menière's disease. *Audiology.* 26, 103–110.

Orchik DJ, Ge NN, Shea JJ Jr. (1998) Action potential latency shift by rarefaction and condensation clicks in Menière's disease. *J Am Acad Audiol.* 9, 121–126.

Orchik DJ, Shea JJ Jr, Ge X. (1993) Transtympanic electrocochleography in Ménière's disease using clicks and tone-bursts. *Am J Otol.* 14, 290–294.

Parving A, Elberling C, Salomon G. (1981) ECochG and psychoacoustic tests compared in identification of hearing loss in young children. *Audiology.* 20, 365–381.

Patuzzi RB, Yates GK, Johnstone BM. (1989) The origin of the low-frequency microphonic in the first cochlear turn of guinea-pig. *Hear Res.* 39, 177–188.

Pou AM, Hirsch BE, Durrant JD, Gold SR, Kamerer DB. (1996) The efficacy of tympanic electrocochleography in the diagnosis of endolymphatic hydrops. *Am J Otol.* 17, 607–611.

Ryerson S, Beagley H. (1981) Brainstem electric responses and electrocochleography. *Br J Audiol.* 15, 41–48.

Santarelli R. (2010) Information from cochlear potentials and genetic mutations helps localize the lesion site in auditory neuropathy. *Genome Med.* 2, 91.

Santarelli R, Arslan E. (2002) Electrocochleography in auditory neuropathy. *Hear Res.* 170, 32–47.

Santarelli R, Arslan E. (2013) Electrocochleography. In: Celesia GG, ed. *Disorders of Peripheral and Central Auditory Processing. Handbook of Clinical Neurophysiology.* Vol 10. Amsterdam: Elsevier; pp 83–113.

Santarelli R, Del Castillo I, Rodríguez-Ballesteros M, Scimemi P, Cama E, Arslan E, et al. (2009) Abnormal cochlear potentials from deaf patients with mutations in the otoferlin gene. *J Assoc Res Otolaryngol.* 10, 545–556.

Santarelli R, Del Castillo I, Starr A. (2013) Auditory neuropathies and electrocochleography. *Hear, Balance Commun.* 11, 130–137.

Santarelli R, Scimemi P, Dal Monte E, Arslan E. (2006a) Cochlear microphonic potential recorded by transtympanic electrocochleography in normally-hearing and hearing-impaired ears. *Acta Otorhinolaryngol Ital.* 26, 78–95.

Santarelli R, Scimemi P, Dal Monte E, Genovese E, Arslan E. (2006b) Auditory neuropathy in systemic sclerosis: a speech perception and evoked potential study before and after cochlear implantation. *Eur Arch Otorhinolaryngol.* 263, 809–815.

Santarelli R, Starr A, Michalewski H, Arslan E.(2008) Neural and receptor cochlear potentials obtained by transtympanic electrocochleography in auditory neuropathy. *Clin Neurophysiol.* 119, 1028–1041.

Sass K. (1998) Sensitivity and specificity of transtympanic electrocochleography in Meniere's disease. *Acta Otolaryngol.* 118, 150–156.

Sass K, Densert B, Magnusson M, Whitaker S. (1998) Electrocochleographic signal analysis: condensation and rarefaction click stimulation contributes to diagnosis in Meniere's disorder. *Audiology.* 37, 198–206.

Sparacino G, Milani S, Magnavita V, Arslan E. (2000) Electrocochleography potentials evoked by condensation and rarefaction clicks independently derived by a new numerical filtering approach. *Audiol Neurootol.* 5, 276–291.

Starr A, Picton TW, Sininger Y, Hood LJ, Berlin CI. (1996) Auditory neuropathy. *Brain.* 119, 741–753.

Starr A, Zeng FG, Michalewski HJ, Moser T. (2008) Perspectives on auditory neuropathy: disorders of inner hair cell, auditory nerve, and their synapse. In: Dallos P, Oertel D, eds. *The Senses: A Comprehensive Reference.* Vol 3. Audition. Amsterdam: Elsevier; pp 397–412.

Wever EG, Bray CW. (1930) The nature of acoustic response: the relation between sound frequency and frequency of impulses in the auditory nerve. *J Exp Psychol.* 13, 373–387.

CHAPTER 13

Auditory Brainstem Response: Differential Diagnosis

Frank E. Musiek, Jennifer E. Gonzalez, and Jane A. Baran

INTRODUCTION

Brief History

Four years after the original article on the auditory brainstem response (ABR) appeared in the literature, Starr and Achor (1975) published their now classic paper on the possible diagnostic use of ABR with neurologic lesions of the auditory brainstem. In their groundbreaking article, they showed abnormal ABR waveforms were relatively common in patients with brainstem involvement of various types. These findings were indeed impressive because the ABR was seldom abnormal in normal control subjects, was noninvasive, and cost relatively little to conduct as a test procedure. This early clinical research relied heavily on the generator sites of the ABR as an anatomical guide to the site of lesion. At about the same time as the Starr and Achor article, Robinson and Rudge (1975) published their clinical research on 30 patients with multiple sclerosis (MS). They reported that 22 of their 30 patients revealed abnormal ABR findings. These results, like those of Starr and Achor, impressed both the audiologic and neurologic communities. Another early study on the use of ABR in patients with neurologic involvement of the brainstem was conducted by Stockard et al. (1977). This was an extensive study on a large cross section of patients (over 100) with a variety of neurologic disorders, which highlighted the potential clinical use of the ABR. These groundbreaking studies, though performed on slightly different populations and using slightly different approaches, all demonstrated the diagnostic value of ABR for brainstem involvement.

In 1977, another classic paper was published by Selters and Brackmann (1977). This report outlined the use of the ABR in the detection of acoustic tumors. Since surgical success with the removal of these tumors was and is often dependent on early detection, interest in this article was extremely high among otologists, audiologists, neurologists, and neurosurgeons. Selters and Brackmann's findings provided a glimpse of studies to come that confirmed their early findings, that is, that the ABR was highly sensitive to acoustic tumors. These researchers also reported a low false-positive rate for the ABR, but cautioned against strict interpretations of the ABR in individuals with severe hearing loss, especially when the high frequencies were affected. Selters and Brackmann championed the practice of interpreting the ABR in light of findings on the audiogram.

These early studies on ABR abnormalities associated with auditory nerve and brainstem pathologies laid the groundwork for continued and expanded clinical applications of the ABR. The clinical research in the 1970s showed the reliability and dependability of the ABR—which remains a key to its continued use. It also demonstrated one of the most important aspects of ABR use, that is, it can be applied across a wide variety of disciplines. At times, this resulted in disagreement and controversy, but it contributed to the remarkable advances and acceptance of the ABR as a diagnostic procedure that followed.

OVERVIEW OF GENERATOR SITES

As alluded to above, the anatomy of the generator sites of the ABR were, and continue to be, critical to diagnostic interpretations. As would be expected, the early research on generator sites of the ABR was done on animals (Buchwald and Huang, 1975). However, as pointed out by Møller and Jannetta (1985), the neural auditory pathways of the auditory nerve and brainstem are different in animals than in man. These differences are probably most obvious for the auditory nerve, which in humans is much longer than it is in small animals. A similar relationship exists for the auditory brainstem pathway, which is also longer in humans when compared to small animals.

Lesion studies from small animals, though helpful, were not as relevant as lesion studies in humans. By the early to mid-1980s anatomical correlates to brainstem lesions in humans were accumulating. These studies were giving rise to the possibilities that the animal data may not be providing the best perspective on the ABR generator sites in humans (Starr and Hamilton, 1976). Although these studies were indeed helpful in attempts to define the ABR generators in humans, the extensive and secondary effects of the lesions often made interpretation difficult. In addition, the ABR is to some degree time locked, meaning that at certain points in time, certain neurons will respond. This is true for the most part, but not totally. For example, some more caudally situated neurons may respond more slowly and

therefore may contribute to an ABR response that is generated largely at a more rostral generator site. This complexity is likely increased by the difference in timing of axonal versus synaptic potentials in the brainstem, both of which can contribute to aspects of the ABR waveform (see Møller and Jannetta, 1985).

Early estimates of the generators for the ABR in humans were somewhat different than what we know now. Perhaps the main differences were differences in Wave II and Wave V. Early interpretations based on animal data suggested that Wave I was generated by the auditory nerve, Wave II by the cochlear nucleus (CN), and Wave V by the inferior colliculus (see Møller and Jannetta, 1985). In the early and mid-1980s, Møller collaborated with Jannetta, a neurosurgeon, to record directly from the auditory nerve and brainstem of humans during neurosurgery. This team effort yielded critical information in regard to the generator sites of the ABR in humans. Accounts of a number of experiments to define generator sites are described by Møller (2000) and the following information is based on this review.

Møller's first major finding in intracranial recordings from humans was that Waves I and II were both generated by the auditory nerve. Wave I appeared to be generated by the more distal aspect and Wave II by the more proximal aspect of the auditory nerve. These waves, which were recorded intracranially, matched well in latency to the latencies of Waves I and II recorded clinically. If Wave II is triggered by the proximal auditory nerve and not the CN in the brainstem then what is generated by the CN? Møller's recordings from near the CN indicated that it matched in latency of Wave III of extra-cranial ABR recordings. More recordings led to the conclusion that Wave III in humans is a CN-generated response. It appears that Wave IV is likely generated primarily by the superior olivary complex based on Møller's observation, whereas Wave V of the ABR is likely generated for the most part by fibers of the lateral lemniscus as they enter into the area of the inferior colliculus. The inferior colliculus, which was originally believed to be the generator site for Wave V, is now believed to be related to a slower, broader wave, likely the negative part of Wave V.

The intracranial recordings of the ABR in humans by Møller and Jannetta showed that the generator sites of the ABR were in fact different from those of earlier interpretations, which were based on animal data. The identification of the generator sites in humans was a major step toward settling the issue of the generators of the ABR and providing a framework for clinical interpretation of this electrophysiological response.

THE IMAGING ISSUE

There has been and continues to be controversy regarding referral patterns for individuals who are suspected to be at risk for vestibular schwannomas (acoustic tumors). There are two competing views. One view is that the patient should be referred directly for imaging, usually magnetic resonance imaging (MRI), to determine if a tumor of the eighth nerve is present. This decision is typically made by the otologist based on the observation of a unilateral sensory/neural hearing loss and/or related auditory symptoms significant enough to support the need for an MRI. This approach has been bolstered by arguments that ABR testing may miss small acoustic tumors (Gordon and Cohen, 1995). The opposing view is that, if the patient is a good candidate for electrophysiological testing, an ABR be conducted before a MRI referral is made. The key argument for this approach is that it will reduce the number of over-referrals for MRI. With healthcare costs being scrutinized more than ever, this approach seems logical and also has garnered support (Musiek et al., 2007; Zappia et al., 1997).

The authors realize that the arguments made for both views have merits, but believe there is, and should be, a role for ABR as a screener for acoustic tumors in present-day medicine. Acoustic tumors are a relatively rare occurrence, reportedly about 1/100,000 in the general population (Kotlarz et al., 1992). However, unilateral sensory/neural hearing loss in adults, a key factor for MRI referral, has a high prevalence with estimates of 21% of all adults undergoing audiologic assessment (Urben et al., 1999). The occurrence of acoustic tumors in individuals with unilateral sensory/neural hearing loss, though higher than in the general population, is still rare with estimates in the 2% to 3% range (Urben et al., 1999; Watson, 1999). Given these statistics, it is easy to recognize how over-referrals primarily based on unilateral sensory/neural hearing loss can occur. Urben et al. (1999) offer a logical decision-making procedure that incorporates the use of ABR test results in the clinical decision as to which patients should be screened and which procedures should be performed for individuals presenting with unilateral sensory/neural hearing loss.

Current concerns over healthcare costs make ABR screening for acoustic tumors a strong consideration. Fair estimates of ABR testing in the United States are in the $300 to $400 range, whereas MRIs with gadolinium contrast can range from $2,400 to $3,000 (Murphy and Selesnick, 2002). Other factors besides the expenses associated with MRI should be considered. Patient access to MRI can be a problem in rural areas, and patients with metal implants or other metal devices cannot be assessed with MRI. Also, some patients cannot tolerate MRI testing because of severe anxiety and/or claustrophobia (Katz et al., 1994).

It is well known that MRI is considered the gold standard for the detection of acoustic tumors; however, it is not well known that MRI is not perfect and false positives do occur. MRI has been regarded as more useful than ABR in the detection of small intracanicular tumors. In one study, however, the false-positive rate for intracanicular tumors was 8 in 25 (32%) using gadolinium enhancement (Arriaga et al., 1995). In their review of the literature, House et al. (2008) reported an MRI false-positive rate ranging from 2%

to 32%. This group also reported a case of a false-positive result of an apparent tumor at the fundus of the internal auditory meatus (IAM) measuring 8 × 3 × 4 mm. At surgery, all four cranial nerves of the IAM were normal. House et al. (2008) offered a note of caution stating that anything resulting in vascular engorgement such as inflammations or infections may look like a neoplasm on contrasted enhancement of an MRI.

It is fair to say that the medical management of small acoustic tumors has changed over the years. There recently has been a greater tolerance for "watching and monitoring" small mass lesions (Bakkouri et al., 2009). This is especially the case for the older patient who has minimal neurologic symptoms. The issue of wait and watch is not without controversy as some surgeons believe that outcomes will be better if smaller tumors are detected early (House et al., 2008). However, it does seem that a greater tolerance for watching and waiting has evolved with evidence for doing so (Bakkouri et al., 2009). Although it is true that many tumors may not grow or may grow only slowly without any serious consequences, it is impossible to predict which tumors will grow slowly and which will grow more rapidly. Therefore, it would seem that once the presence of a small tumor has been confirmed, monitoring with both ABR (and possibly a stacked ABR procedure) and MRI may be advantageous as the combination of these two measures could provide both anatomic and physiological evidence of change or lack of thereof.

Another potential shortcoming of reliance on MRI without including ABR testing is that the ABR provides an index of physiology, which at times certainly differs from anatomical indices (e.g., MRI). There can be abnormal function of the auditory nerve without the problem being a space-occupying lesion such as a vestibular schwannoma or acoustic tumor. Various auditory neuropathies, minor vascular problems, inflammations, and infections may not show up on imaging studies, but may still affect hearing. Moreover, auditory nerve problems in many cases are handled differently than cochlear problems and this differentiation needs to be made. That is, many auditory nerve problems can be of a more serious nature and a possible threat to the patient's overall health, whereas many cochlear problems, though handicapping, are not as much of a health risk.

THE AUDITORY BRAINSTEM RESPONSE: DIAGNOSTIC ASPECTS

In discussing the ABR in its capacity to aid in the diagnosis of specific pathologies, it must first be understood that the procedures used are measures of the function or physiology of the auditory nerve and brainstem and not measures of the structure or anatomy of these areas. By using the pattern of findings obtained for each individual patient through this physiological procedure, it is possible to make inferences about which pathologies may be causing the disruptions in the typical ABR pattern. Although some pathologies of the auditory nerve result in similar patterns on the ABR, due to the relatively small area of the auditory pathway affected, pathologies in more rostral areas of the brainstem tend to result in a wider array of findings, which are dependent on both the size and location of the lesion. Our focus on pathologic ABR patterns will begin with a description of the findings in patients with auditory nerve involvement and will then progress to ABR findings associated with brainstem dysfunction.

ABR AND AUDITORY NERVE INVOLVEMENT

In examining the ABR in the context of auditory nerve involvement, it is useful to break this category into three parts representing the most frequently described pathologies of the eighth nerve: vestibular schwannoma, auditory neuropathy spectrum disorder (ANSD), and other pathologies (including Charcot–Marie–Tooth (CMT) syndrome, vascular loop syndrome, and auditory nerve aplasia and hypoplasia). The "other pathologies" is a somewhat limited list that is far from complete, but it does relate information on some of the more common pathologies affecting the auditory nerve.

Vestibular Schwannoma

Vestibular schwannomas (also commonly termed acoustic neuromas, acoustic tumors, or eighth nerve tumors) are structural anomalies that are so named because of their location and the specific cells that are comprised. These tumors most commonly arise from the proliferation of myelin-producing Schwann cells encasing the vestibular portion of the eighth nerve. Although myelin in the brainstem proper is produced by oligodendrocytes, the myelin surrounding the auditory nerve is produced by Schwann cells, hence the tumors arising from these cells are appropriately referred to as vestibular schwannomas. Making up an estimated 90% of all tumors of the temporal bone, vestibular schwannomas generally are benign and slow growing (Bebin, 1979). These tumors are estimated to grow between 0.1 and 0.2 cm in diameter per year in many cases; however, there are some documented cases in which the tumor does not change in size (Bederson et al., 1991). Unilateral tumors occur in the vast majority of cases (95%), with the most common symptoms being unilateral tinnitus and unilateral progressive hearing loss on the side of the tumor (Jerger and Jerger, 1981). Since the auditory and vestibular portions of the auditory nerve are in such close proximity to one another, their growth in the majority of cases results in high-frequency hearing loss because of the compression of the outer boundary of the auditory portion of cranial nerve VIII. Vestibular symptoms are rarely present because of the

slow rate of growth for these tumors and the tendency of the central nervous system (CNS) to compensate for these gradual changes. If these tumors grow to be larger than about 2 cm in diameter, they may begin to spread into the cerebellopontine angle (CPA), at which point the brainstem may also show signs of compromise.

Patterns of ABR Findings in Vestibular Schwannoma

Absolute Latency Delay

An absolute latency delay for Wave V is likely in cases of vestibular schwannoma and can be used as a diagnostic index. The difficulty is that delayed absolute latency of Wave V can also be a result of cochlear or even conductive hearing loss. The measurement of absolute latency of Wave V is used when the earlier waves are absent, which can create a diagnostic problem in differentiating retrocochlear from cochlear loss (this topic is discussed later in this chapter) (Figure 13.1). In these situations, other measures of the ABR as well as patient history can become important and helpful. Of course, the intensity of the stimulus, recording techniques, and measurement of the waveform can also influence the absolute latency and therefore should be considered (Musiek, 1991; Musiek et al., 1996). When Wave V latency measurements are greater than 6.1 ms in response to a click stimulus presented at a moderately high intensity level, the possibility of an eighth nerve tumor should be considered. It should be remembered that the absolute latency of Wave V becomes more important when other ABR indices cannot be used. Because of the high-frequency weighting (around 3 kHz) that a click stimulus takes on when presented through a normally structured peripheral auditory system, extended Wave V absolute latencies are a cause for concern particularly when audiometric findings indicate relatively good high-frequency hearing. When high-frequency hearing is poor, the interpretation of Wave V latency can become ambiguous. This clinical situation is most difficult and other means to delineate eighth nerve involvement would be required. The absolute latencies of the earlier ABR waves (I and III) are not high in the interpretive scheme because if they are present in a potential case of an eighth nerve tumor other indices can and would be used.

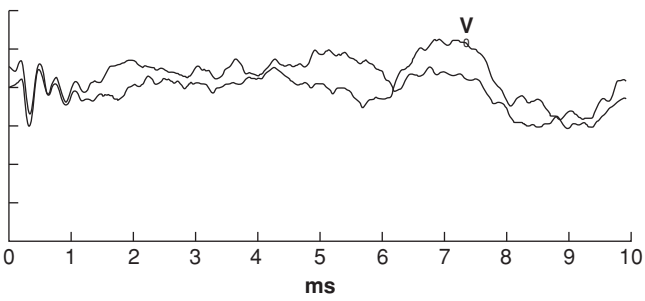


FIGURE 13.1 An auditory brainstem response obtained from a middle-aged patient with a severe high-frequency cochlear hearing loss. The ABR tracings show a delay in the absolute latency of Wave V and the absence of the earlier ABR waves [subtract 0.9 ms per ER-3A inserts*]. Note: For the case studies reported here and in the following figures the ABRs were conducted at 80 to 90 dB nHL using a 100 μs click stimuli at repetition rates of 11 to 17 clicks per second. Filtering was 100 to 3,000 or 1,500 Hz with a 12-dB/octave roll off unless otherwise noted. *Insert phone delay = 0.90 ms.

Interwave Latency Delay

Interwave latency delays are measurements sensitive to the physiological changes that take place with vestibular schwannomas. Because of the fact that most eighth nerve tumors grow between the generator sites of Waves I and III, the I–III interwave interval (IWI) is likely to be extended in these cases (Musiek et al., 1986b). In many clinics and laboratories a I–III IWI extending beyond 2.4 ms is considered abnormal (see Hall, 1992). One of the problems often encountered in applying the I–III IWI is when these two waves (I and III) cannot be obtained, which is a situation that is often encountered when a significant cochlear hearing loss is present (Musiek et al., 1986b). When both Waves I and III are present in patients with eighth nerve tumors, the IWI measurement is extended 85% to 100% of the time. As such, it is a highly sensitive measure (Antonelli et al., 1987; Musiek et al., 1986b; and for review see Musiek et al., 2007). Since the I–III IWI is of significant value in the interpretation of the ABR for diagnostic purposes, clinicians may use a variety of means to obtain these waves. In cases where Wave I is absent or difficult to visualize, electrocochleographic approaches and/or an increased stimulus intensity may be used to enhance Wave I so the I–III IWI index can be derived.

The I–V IWI may also be used in the detection of vestibular schwannoma, though the extension of this interval is often a reflection of an increased I–III IWI measurement (Musiek et al., 1986b). However, in some cases, depending on the precise location and size of the tumor, the III–V IWI may be extended in addition to the I–III IWI making the I–V IWI most helpful to the interpretation as is discussed later in this chapter. Of course, in cases where Wave III is absent, the I–V interval becomes the IWI of choice. Although there is much information on norms for the I–V IWI index, much of the data are quite similar. Therefore, measurements of the I–V interval that exceed 4.4 ms are commonly considered abnormal across many clinics (Hall, 1992). This interval, though, involves a larger area of the auditory brainstem than does the I–III interval and thus cannot be used on its own to precisely determine a site of lesion as the I–V interval can be extended for eighth nerve tumors as well as for brainstem lesions. The I–II IWI could also be used diagnostically; however, since Wave II is not present in all nontumor individuals, its absence in patients with this potential diagnosis is not clinically dependable (see Hall, 1992). However, in

situations where there is suspicion of an eighth nerve tumor and Wave II is present and normal, it is likely that the auditory nerve is not involved (based on the Wave II generator site). The III–V IWI is likely to be normal in cases of vestibular schwanommas, as the generator sites for these waves are more rostral than the auditory nerve. However, in some cases of vestibular schwannomas, the III–V IWI is extended (Møller and Møller, 1983; Musiek et al., 2007). This has been attributed to large tumors in the CPA that have twisted and/or compressed the brainstem.

Interear Latency Comparisons

An interear latency comparison, or interaural latency difference (ILD), using Wave V measurements obtained from the left and right ears can be diagnostically relevant, especially when used alongside interwave latency measurements. First described by Selters and Brackmann (1977) and Brackmann and Selters (1979), ILD measurements are particularly attractive in the process of diagnosing vestibular schwannomas because in some cases, Wave V is the only wave of the ABR that is present in each ear; hence it has greater utility than interwave measures. Also, this is a "within patient" comparison, which has some nice clinical and research advantages. When using this index, one must be keenly aware of the effect that asymmetrical hearing losses have on the ABR. If an asymmetrical hearing loss of cochlear origin is present, it is possible to misinterpret the ABR as abnormal—especially in cases when the hearing sensitivity in the poorer ear is severe enough to result in an ABR with an extended Wave V absolute latency and an absence of the earlier waves. To ensure that the observed effect on the ABR is not because of asymmetrical sensory/neural hearing loss, Selters and Brackmann proposed the use of a correction factor of 0.1 ms for every 10 dB of hearing loss at 4 kHz greater than 50 dB HL; however, the use of correction factors in determining abnormality of ILD measurements can be cumbersome and at times may not be clinically insightful. In some cases, application of the various correction factors to adjust for the potential effects of high-frequency hearing loss can actually hinder correct interpretation (Cashman et al., 1993; Musiek et al., 2007). It must also be noted that asymmetrical hearing loss and vestibular schwannoma do not always occur in isolation, and it is possible to have effects on the ABR resulting from both a cochlear hearing loss and a retrocochlear pathology. Most would agree that when using this measurement, ILDs greater than 0.3 to 0.4 ms should be considered abnormal and would support a diagnosis of retrocochlear involvement as long as the contribution of a cochlear hearing loss can be ruled out (Musiek et al., 1989).

Amplitude Comparison of Waves

The Waves V/I amplitude ratio is another ABR index that has been used to aid in identifying vestibular schwannomas (Chiappa, 1983; Musiek et al., 2007). Before employing this index in the interpretation of the ABR, however, the reliability of amplitude measurements must be examined because of their inherent variability. The ABR should be replicated at least once, and each waveform's Wave I and V amplitude measurements should not vary by more than 20%. When Wave V exists as a portion of a IV–V complex, the greatest amplitude of the complex should be used when calculating the V/I amplitude (peak to trough) ratio. When the Wave V amplitude is less than 0.75 of the amplitude of Wave I, it is considered abnormal and is suggestive of retrocochlear pathology (see Musiek et al., 1996). It should be mentioned that a significantly smaller Wave V than Wave I does not occur very often even in pathology; however, when it does, it should be interpreted with concern (Musiek et al., 1996).

Waveform Morphology and Absence of Waves

Although a waveform's overall appearance or morphology may be difficult to describe and therefore difficult to use in identifying vestibular schwannomas, the absence of waves on an ABR recording is a significant finding. The presence of an eighth nerve tumor should be considered if all waves of the ABR are missing. However, as mentioned in the discussion on asymmetrical hearing losses and their effect on the ABR, the absence of waves must be interpreted along with the audiometric results that were obtained for the patient. The most striking indication of a retrocochlear lesion is the absence of all ABR waves while audiometric results from that ear indicate relatively normal hearing sensitivity. Individual waves may also be absent in cases of vestibular schwannoma, with Wave III most commonly missing (Musiek et al., 1986a). An important interpretation is when Wave I is present and the remaining waves are absent. This is a strong indicator of retrocochlear involvement (see Figure 13.2). Generally, this particular interpretation can be made without an audiogram. However, the absence of early waves must be interpreted alongside the audiogram as individuals with cochlear hearing loss are likely to be missing Wave I, Wave III, or both. There are also some instances of normal hearing individuals (without tumors) missing Waves I and III, but this is relatively rare if appropriate stimuli and recording techniques are used (see Hall, 2007; Musiek et al., 2007). Therefore, the absence of earlier waves is not a reliable indicator of the presence of vestibular schwannoma.

Repetition Rate Shifts

The utilization of high repetition (high rep) rates to help in defining eighth nerve tumors has been controversial. This measure involves the comparison of the latency of Wave V at low and high rates of presentation such as 11 clicks per second to 81 clicks per second. If there is a greater than expected latency increase or a disappearance of Wave V during the high rep rate recording, then the interpretation is for possible eighth nerve pathology. Early reports have shown some value in this procedure (Daly et al., 1977). In many instances, high rep rate measures show abnormalities that have also been revealed by other ABR indices. In these cases,

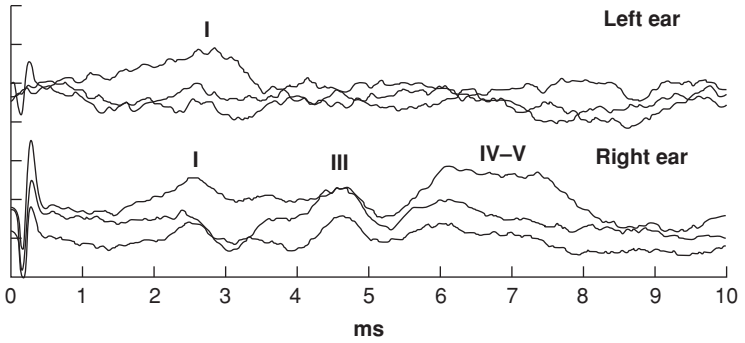


FIGURE 13.2 A middle-aged patient with a left-sided vestibular schwannoma (acoustic tumor) with the waveform showing a poor morphology Wave I and no replicable later waves (subtract 0.9 ms per ER-3A inserts*). *Insert phone delay = 0.90 ms. (Reprinted with permission from Musiek FE, Shinn JB, Jirsa RE. (2007) The auditory brainstem response in auditory nerve and brainstem dysfunction. In: Burkard RF, Don M, Eggermont JJ, eds. *Auditory Evoked Potentials: Basic Principles and Clinical Application.* Baltimore, CA: Lippincott Williams & Wilkins; pp 291–312.)

they may not be essential for the diagnosis (Musiek et al., 1996). However, high rep rate ABRs can be useful in helping to detect eighth nerve tumors and in some cases an abnormal high rep rate measure is the key index (Tanaka et al., 1996). It seems that even after many years of research this measure still remains controversial (see discussion regarding brainstem lesions).

Laterality

Since the site of lesion for vestibular schwannoma tumors is caudal to the location of the first binaural representation in the auditory system (the superior olivary complex), abnormal ABR findings are generally ipsilateral to the ear of stimulus presentation. However, if the tumor grows to a substantial size the contralateral ABR can be affected because of compression and possible twisting and displacement of the brainstem from midline. When this happens, Wave V or the IV–V complex will be missing, delayed, or reduced in amplitude resulting in a III–V IWI extension or a III–V IWI that cannot be measured (Musiek and Kibbe, 1986) (Figure 13.3). Clinically, this presentation of a contralateral ear abnormality could be helpful in a situation where there is considerable hearing loss and no readable ABR on the side of the lesion. If the contralateral ear has reasonable hearing and the ABR shows an effect on the later waves, suspicion of a large lesion may be appropriate and further delineation should be sought (see section on brainstem involvement).

Threshold Measures

Another index which can help with the differentiation of vestibular schwanommas is comparing the behavioral threshold with the ABR threshold. It is well known that the ABR threshold can closely approximate the behavioral threshold of hearing. However, some preliminary data, as well as various case reports, have indicated that in patients with eighth nerve tumors, the difference between the measured ABR threshold and the behavioral threshold is more than would be expected—often, 30 dB or greater (Bush et al., 2008).

In patients with eighth nerve tumors, the most reliable diagnostic measurements to use are the I–III and I–V IWI measurements and the ILD, either alone or in combination. Using these indices in the interpretation of the ABR results in hit rates of 90% or better and false-positive rates of 20% or less (Musiek et al., 2007).

Stacked ABR

Although the ABR has high hit rates and low false-positive rates for medium- and large-sized vestibular schwannomas, smaller sized tumors are often missed because of the small number of nerve fibers affected by the growth. In addition

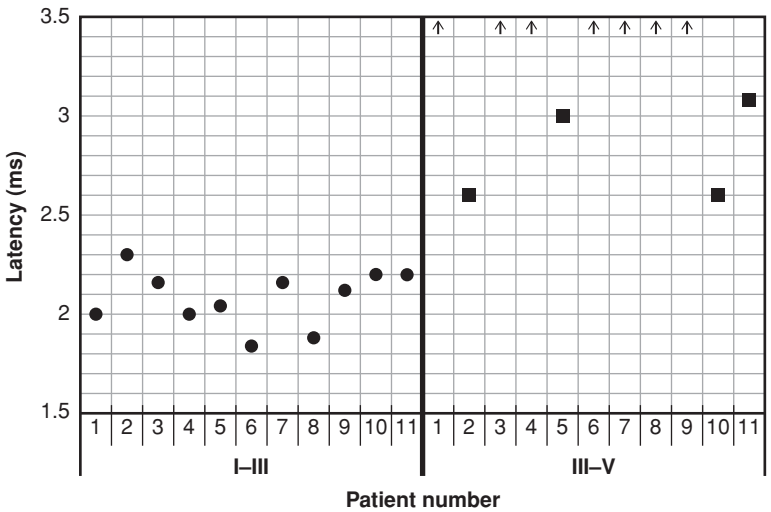


FIGURE 13.3 A scatterplot of I–III and III–V ABR interwave intervals for 11 subjects with large acoustic neuromas. The data displayed are for the contralateral ear (i.e., nontumor ear), all of which had normal or near-normal hearing. Note how the I–III interval is essentially normal for all 11 subjects, whereas the III–V interval is abnormal for 4 ears and Wave V was missing in 7 cases (indicated by *vertical arrow*). (Constructed from data presented in Musiek FE, Kibbe K. (1986) Auditory brainstem response wave IV-V abnormalities from the ear opposite large cerebellopontine lesions. *Am J Otol.* 7(4), 253–257.)

to the standard ABR missing smaller-sized tumors, which by nature of their size affect a smaller number of neurons, tumor location may also lead to false-negative results on the ABR. Given the high-frequency weighting of the click stimulus, a standard click ABR relies on the integrity of the outer boundary of the auditory nerve. Should the location of the tumor not be on this outer boundary but rather in the middle or inner portions of the nerve, the standard ABR is likely to miss these tumors.

Don et al. (1997) developed a method of detecting tumors that are missed by standard ABR testing because of either tumor location or size (i.e., the Stacked ABR). The Stacked ABR aims to measure neural activity not just from a narrow portion of the cochlea (as is the case when the standard ABR is employed), but from the length of the cochlea as a whole. The ABR results would then be a reflection of the neural synchrony lost because of the presence of a tumor regardless of which fibers are affected by that tumor (Don et al., 2005).

A click stimulus is used to evoke this ABR, and the response to the click is separated into five frequency bands via high-pass masking and response subtraction (Don and Eggermont, 1978; Parker and Thornton, 1978a, 1978b). Six stimulus conditions are used to obtain these ABRs and consist of clicks alone without masking and clicks presented along with five variations of simultaneous ipsilateral high-pass pink noise (cutoff frequencies of 0.5, 1, 2, 4, and 8 kHz).

Derived-band ABRs are then obtained by subtracting one high-pass masker condition's ABR from the ABR obtained with high-pass masking one octave above it. For example, the ABR obtained with 0.5-kHz high-pass noise is subtracted from the ABR obtained with 1-kHz high-pass noise. Once derived-band ABRs are obtained for all noise conditions, they are then shifted in time so that each Wave V is in alignment, thus the term "stacked." After the waveforms are aligned, they are added together to form the resulting Stacked ABR. The amplitude of the Stacked ABR is then compared to the ABR obtained using the click stimulus alone (without high-pass pink noise). In normal ears, Wave V from the Stacked ABR should have the same amplitude as the standard click-evoked ABR; however, when a small tumor is present or when the tumor affects more low-frequency–tuned nerve fibers, Wave V from the Stacked ABR will be smaller in amplitude than the amplitude noted in the standard ABR (Don et al., 1997). When tumors are 1 cm in diameter or smaller, the Stacked ABR is highly sensitive (95%) and specific (83%) (Don et al., 1997) (see Chapter 11 for more information on the Stacked ABR procedure).

Auditory Neuropathy Spectrum Disorder

Unlike eighth nerve tumors, which are structural abnormalities that affect the physiology measured with the ABR, ANSD is a purely functional or physiological abnormality that can include effects on the function of the auditory nerve. There is no obvious structural abnormality causing the ABR to be affected; therefore, imaging (i.e., MRI) results in cases of pure ANSD are not remarkable. The strict definition of ANSD is the presence of otoacoustic emissions (a measurement of outer hair cell function) in the complete absence of the ABR. Therefore, the site of origin for ANSD is between the cochlea proper and the brainstem, with possible causes as injury to the synaptic junctions of the inner hair cells and/or the dendrites of the auditory nerve that receive neurotransmitters released by the inner hair cells, injury to the spiral ganglion, and/or damage to axons of cranial nerve VIII. When damage to the auditory nerve axons is present, effects tend to spread to portions of the brainstem. Sites of neuronal injury can occur to the myelin sheath, the axon, or both, as well as to the cell body. Therefore, the list of conditions that can cause ANSD and absent ABRs is widespread and includes low birth weight, prematurity, viral disease, seizure, anoxia, hypoxia, and CMT, Ehlers–Danlos, and Stevens–Johnson syndromes. ANSD may also occur spontaneously with no known pathologic cause. Hyperbilirubinemia (HB), or jaundice, is often linked to ANSD; however, it should be realized that this disorder is primarily a central auditory system disorder as the damage resulting from this pathologic process is first focused on brainstem nuclei (namely, the CN) and not the auditory nerve (Dublin, 1985). This damage may begin to encroach on the auditory nerve's root entry zone into the CN and then to the auditory nerve itself, but the typical understanding of ANSD with primarily auditory nerve damage is not the case with HB. Damage in HB begins in the brainstem and then descends to the auditory nerve.

The underlying physiology of ANSD is seen on the ABR as waveforms that have no measurable aspects (i.e., amplitude and latency of Waves I, III, and V) and are not replicable. Because there are no measurable aspects, none of the usual procedures can be carried out. However, a cochlear microphonic should be present on the ABRs obtained because aspects of cochlear function can be normal or near-normal in ANSD. The cochlear microphonic is a response predominantly generated by the outer hair cells in the cochlea, which occurs before Wave I of the ABR and follows the phase of the presented stimulus. When obtaining ABRs in suspected cases of ANSD, it is important to obtain separate replicated waveforms using single-polarity condensation and rarefaction click stimuli to identify the cochlear microphonic. The cochlear microphonic begins within 1 ms of stimulus presentation and will appear as an initial downward shift from baseline with a rarefaction click stimulus and as an initial upward shift from baseline with a condensation click stimulus. ABR tracings which lack the cochlear microphonic are likely the result of instrumental or clinician error (stimulus not delivered to patient or using alternating polarity clicks). Therefore, equipment and stimulus protocols should be checked to make sure the stimulus is properly delivered to the patient if all waveform aspects of the ABR are absent (see Chapter 14 for more on ANSD).

Charcot–Marie–Tooth Syndrome

As mentioned, CMT disease could be viewed as a specific type of auditory neuropathy. However, it stands as a well-recognized disorder on its own. It was and still is considered an inherited peripheral motor neuron disease, but reports of auditory involvement began to surface in the 1970s and 1980s. Weaknesses of hands and feet are often noticed with some cases clearly having auditory complaints (see Musiek et al., 1982). Patzkó and Shy (2010) relate that CMT is characterized by disruptions in myelin along the nerve axon or damage to the axon itself, which affects the velocity and size of the impulses conducted down the axon and communication among nerves. There are a variety of types of CMT mostly classified by myelin or axon involvement and the types of genes that are involved.

Of interest from an audiologic perspective is that CMT may affect the peripheral nerve (CMT1A) or the CNS (CMTX). Nicholson and Corbett (1996) showed delayed central time on ABR for a CMTX group and only a delayed Wave I for the CMT1A group. However, in another study on CMT1A, the ABRs were essentially normal. This raises the issue of the possibility that some forms of CMT may involve the auditory system and some may not. Clearly, abnormal ABRs have been reported in various groups of CMT patients with delays in the later ABR waves, as well as compromised amplitudes for Wave V (Rance et al., 2012). In apparent long-standing disease, auditory degeneration results in absent ABRs. Some of the variance in ABR findings may be related to the particular type of CMT, but to delineate this is beyond the scope of this chapter. It is worthy to note, however, that the ABR is a key procedure to utilize in the evaluation of CMT, especially when the patient has auditory symptoms.

Auditory Nerve Aplasia/Agenesis and Hypoplasia

In addition to the aforementioned auditory nerve disorders affecting the ABR, it is also possible for the auditory nerve to be missing (aplasia or agenesis) or underdeveloped (hypoplasia). In both cases, the malformation is congenital with the possibility of stenosis of the IAM or cochlear malformations. In a study of 13 ears from nine children with cochlear nerve deficiency, 4 ears exhibited small IAMs on MRI (Buchman et al., 2013). Of the nine children diagnosed with cochlear nerve deficiency, five were affected unilaterally and four were affected bilaterally. In all nine cases, a cochlear microphonic was present with absent ABRs in at least one ear. With a variety of temporal bone malformations that may accompany auditory nerve aplasia or hypoplasia, the audiologic profiles of these cases are wide ranging.

In the case of auditory nerve aplasia, the lack of the nerve prevents the transmission of auditory information to areas of the auditory system beyond the cochlea. It is expected that in an otherwise intact peripheral auditory system, individuals with auditory nerve aplasia will display normal tympanograms, whereas acoustic reflexes will be absent when the aplastic auditory nerve is on the stimulated side. Otoacoustic emissions may or may not be present on the affected side depending on whether or not the cochlea is involved. As the ABR requires neural conduction along the auditory nerve in order for it to be evoked, an absence of the nerve results in the absence of the ABR waves.

In auditory nerve hypoplasia, cochlear function as measured by distortion product otoacoustic emissions (DPOAEs) may be related to the width of an accompanying IAM stenosis. A study of 10 subjects diagnosed with auditory nerve hypoplasia and nonsyndromic IAM anomalies (without other external malformations) found 3 subjects with a very narrow IAM (<30% of average), 3 subjects with intermediate IAM (30% to 70% of average), and 4 subjects with a slightly narrow IAM (>70% of average) (Ito et al., 2007). Of those 10 subjects, 1 from the intermediate group had reduced DPOAEs from 1,000 to 3,000 Hz and 2 from the slightly narrow group had normal DPOAEs in the same mid-frequency range. The remaining seven subjects, including the three in the very narrow group, two in the intermediate group, and two in the slightly narrow group had absent function or severe dysfunction as measured by DPOAEs, demonstrating that although cochlear function may be spared in some patients with slight narrowing of the IAM, findings of absent or severely impaired cochlear function are common among patients with auditory nerve hypoplasia. The ABR was absent in all 10 subjects of this study; however, Taiji et al. (2012) studied six ears with cochlear nerve hypoplasia, all associated with IAM stenosis, and found that whereas DPOAEs were present and normal in one of the six ears, threshold ABRs were present in three of the six ears, excluding the ear with normal DPOAEs. In the cases with demonstrated ABRs, thresholds (dB nHL where Wave V just began to appear) were elevated at 60, 80, and 90 dB nHL, whereas DPOAEs were absent in all three cases. This is not particularly surprising as DPOAEs are a measure of outer hair cell function, whereas a high-intensity ABR relies on inner hair cell integrity. None of the subjects studied had known syndromes or risk factors as a cause of their hearing losses, including HB, hypoxia, or premature birth. Therefore, the presence of an ABR in auditory nerve hypoplasia is difficult to predict from the audiometric profile.

Vascular Loop Syndrome

Vascular loop syndrome is a condition in which the cranial nerves (including the eighth cranial nerve) in the vicinity of the CPA are compressed by a blood vessel, with the anterior inferior cerebellar artery (AICA) being the most commonly affected vessel (Jannetta, 1967). This compression may result in a variety of auditory, vestibular, or facial symptoms among individuals depending on the location and amount of compression.

When compression is present on the auditory portion of the eighth cranial nerve, ABR results are often abnormal.

Extensions of the I–II and/or I–III interwave latencies on the affected side are typical, with these findings likely to improve with surgical intervention. ABR findings from vascular loop syndrome may not differ from those described for vestibular schwannoma; however, the ABR in either case will display abnormalities consistent with eighth nerve involvement. Differentiation between the two disorders after obtaining ABR may be made with imaging techniques such as MRI or computed tomography (CT) (see Møller, 2000; Musiek et al., 2012 for review).

ABR AND BRAINSTEM INVOLVEMENT

General Background

ABR findings in lesions of the auditory brainstem pathway continue to be an important clinical application for audiologists and neurologists. The early studies demonstrated the diagnostic value of the ABR when the auditory brainstem pathway was involved (Starr and Achor, 1975; Stockard et al., 1977). However, as data accumulated over the years clinicians and researchers began to realize that though still valuable, the ABR was not quite as sensitive to a wide variety of brainstem disorders (with the exception of intra-axial lesions) as it was for vestibular schwannomas (Musiek, 1991) for a number of reasons. First, there are a wide variety of known neurologic disorders that may affect the brain, whereas there are relatively few that compromise the auditory nerve (Chiappa, 1983, 1989; Musiek, 1991). However, those lesions that affect the auditory nerve, like vestibular schwannomas, almost always compromise the nerve directly. Second, some neurologic disorders may or may not target the auditory brainstem pathway. For example, MS may arise in the brainstem, but not necessarily in the auditory tracts. In this case the ABR would be normal, but without highly definitive imaging the examiner may not know this and assume that the ABR failed to identify this disease. Also, surveys have shown that the ABR hit rate for MS is in the neighborhood of 60% or slightly better. This is without defining the site of lesion as being in the auditory tracts (Musiek, 1991). However, in cases where the MS lesion is decidedly in the auditory pathway, the ABRs are almost always abnormal (Levine et al., 1993).

ABR testing has become highly useful for those lesions affecting the brainstem for which imaging is of little help. Imaging results are often normal in cases of HB and external toxic exposures and in some cases of head injury. Yet, in many of these cases, auditory and other symptoms abound. Careful ABR analysis in such cases may provide insights as to the nature of the auditory symptoms and the integrity of the auditory brainstem (see Musiek et al., 2007). Effective ABR analysis requires the understanding of the various ABR diagnostic indices and an understanding of various diagnostic patterns, some of which are specific to brainstem involvement.

Patterns of ABR Waves Related to Brainstem Involvement

Absolute Latency Delay

The diagnostic index of absolute latency delay of Waves I, III, or the IV–V complex can reflect conductive, cochlear, or retrocochlear involvement. In regard to brainstem involvement, a delayed Wave I would not be highly useful. A delayed Wave III or IV–V without Wave I as alluded to earlier could be indicative of a number of different sites of lesions. A delayed Wave III with a normal III–V interval or a delayed V with absence of Waves I or III often occurs, but the clinician would have to utilize additional measures to determine if there is brainstem involvement (see Musiek and Lee, 1995; Musiek et al., 2007).

Interwave Latency Delay

The extension of the I–III, III–V, or I–V IWIs is perhaps the most revealing measurement of brainstem involvement. This is because these indices involve the brainstem generators of Waves III, IV, and V. Since Waves I and II are generated by the auditory nerve, the interpretation would be that the auditory nerve is intact and functioning if these waves are present at normal latencies (Møller, 2000). An extension of the I–III IWI could indicate that the auditory nerve is involved, that is, a lesion between the distal segment of the auditory nerve and brainstem or that the caudal brainstem is compromised at or in the area of the CN (Musiek et al., 1986b). Both of these interpretations are plausible and should be entertained. If the I–III IWI is extended but Wave II is present, one might argue for brainstem involvement only; however, there is little, if any, published research on this type of ABR pattern.

Based on generator sites, a III–V interval increase is firmly interpreted as brainstem involvement (Musiek and Lee, 1995; Musiek et al., 2007; Starr and Achor, 1975). It is possible that the III–V interval extension can result from an eighth nerve tumor, but generally these tumors would have to be quite large, affecting the brainstem considerably for this ABR abnormality to be seen (Musiek et al., 1996). This interpretation is dependable for indicating brainstem involvement, but one must be confident in the selection of the peaks of Waves III and V. These waves can be rather distorted and difficult to visualize in patients with brainstem lesions.

The I–V IWI is often extended in brainstem lesions, but it is to a great extent dependent on its subcomponents, the I–III and III–V intervals; that is, often an extended I–III or III–V IWI will drive the extended I–V interval. However, at times one of these subcomponents may be abnormal whereas the other is normal resulting in a normal I–V IWI. This highlights the importance of looking at all IWIs if they are present. For example, if normal IWIs are 2.4 ms or less for the I–III, 2.3 ms or less for the III–V, and 4.4 ms or less for the I–V IWIs, a patient could present with a 2.5 ms III–V and a 1.9 ms I–III and have a normal I–V interval (4.4 ms). Another situation is when the I–III and III–V

IWIs are normal, but when combined, they contribute to an extended I–V IWI (I–III = 2.3 ms, III–V = 2.3 ms), which results in an abnormal I–V interval of 4.6 ms. The main weakness of relying on IWI measures is that often they are not all available; therefore, some of these interpretations cannot be made (see Musiek et al., 1986b). A key advantage in utilizing IWIs is that hearing loss has little, if any, effect.

Interear Latency Comparisons

As discussed earlier, the ILD is a valuable index for the detection of eighth nerve tumors; however, this may not be the case with brainstem lesions (Musiek et al., 1989) because the majority of eighth nerve tumors are unilateral, resulting in unilateral latency delays. This means the ILD will be significant (as Wave V will be delayed more on one side than the other side). Brainstem lesions more often affect both brainstem tracts (ipsilateral and contralateral) either directly or indirectly because of the profuse number of cross-over tracts and commissures in the brainstem. These anatomical factors could lead to Wave V latency delays for both ears, and hence a nonsignificant ILD. As shown in Figure 13.4, the mean ILD for a group of patients with brainstem involvement was significantly smaller than a comparable group of patients with eighth nerve tumors (Musiek et al., 1989).

Amplitude Comparison of Waves

One of the more controversial diagnostic ABR indices is the Wave V to Wave I amplitude ratio. It is well known that Wave V or the IV–V complex is normally larger than Wave I in adults (Musiek et al., 1986a) possibly because more neurons are available as one progresses up the auditory pathway. The more nerve fibers that respond the larger the evoked potential response will be. In patients with pathologic conditions, potential damage to the more rostral brainstem fibers may result in the usually larger response (i.e., Wave V or the IV–V complex) being compromised. Neurologists have generally supported the use of the V–I amplitude ratio as a diagnostic criteria for brainstem involvement (Stockard et al., 1978). On the other hand, audiologists have been less enthusiastic about the diagnostic value of the V–I amplitude ratio. To best use this amplitude ratio certain criteria need to be satisfied. One is that the amplitudes of Wave I and V must be highly replicable on two successive trials (Musiek and Lee, 1995; Stockard et al., 1978). Also, in making the comparison of Wave V to Wave I the largest of the later peaks, either Wave IV or V, should be used (Musiek et al., 2007). When these conditions are met then the V–I amplitude ratio, though not highly sensitive, is highly specific if a ratio of less than 0.75 is used (Musiek and Lee, 1995) (see Figure 13.5).

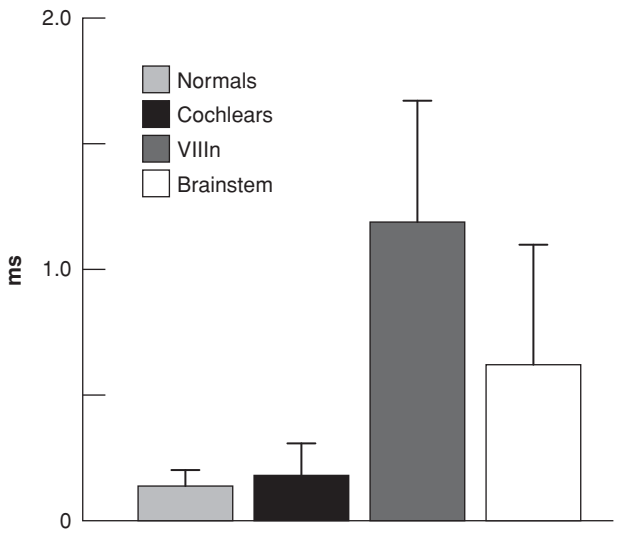


FIGURE 13.4 Plot of the mean and one standard deviation for the interaural latency difference (ILD) for subjects with normal hearing, cochlear involvement, eighth nerve tumors, and brainstem lesions. (Constructed from data presented in Musiek FE, Johnson GD, Gollegly KM, Josey AF, Glasscock ME. (1989) The auditory brainstem response interaural latency difference (ILD) in patients with brainstem lesions. *Ear Hear.* 10(2), 131–134.)

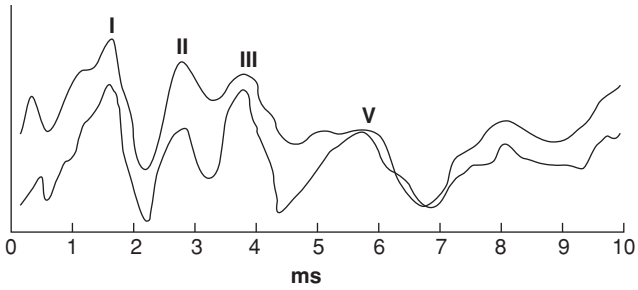


FIGURE 13.5 The ABR from a young adult with an extra-axial brainstem lesion. All latencies were normal; however, Wave I was quite large (~0.7 μV) yielding an abnormal amplitude ratio.

Waveform Morphology and Absence of Waves

Brainstem involvement often results in a highly dependable index for brainstem abnormality when Waves I and III are present and the IV–V complex or Wave V is absent. Brainstem and auditory nerve compromise is also probable when Wave I is present and Waves III and V are absent. When Wave III is absent and Wave V is present, one should look at the I–V IWI. If this interval is normal, then it is likely that the response is normal. An abnormal outcome is when the I–V IWI does not fall within the normal range. Because Wave III absence alone has been reported in normal subjects, this finding must be interpreted with much caution (see Hall, 1992; pp 231–232). At times, Wave IV will be present and Wave V will be absent, which of course is an indication of brainstem involvement. However, in some instances with a high-intensity rarefaction polarity click Wave V can be a very low shoulder on Wave IV and may be mistakenly considered as absent (Gerling, 1989). This can often be clarified by using a contralateral recording, changing polarity, or dropping the intensity level of the click stimulus (Gerling, 1989). The absence of all waves can also occur in individuals with brainstem involvement, but this finding usually occurs

when there is concomitant peripheral hearing loss that prohibits the early waves from evolving.

Contralateral Effects

Large mass lesions on one side, such as an acoustic neuroma, can compress and torque the brainstem sufficiently to cause an ABR brainstem finding on the opposite side when stimulating that ear. This result is not a contralateral recording, but rather a recording from the acoustically stimulated ear (Musiek and Kibbe, 1986). The ear opposite the large lesion, if the hearing is reasonably good, will show normal Waves I and III and an abnormal III–V interval or compromised V or IV–V complex (Musiek et al., 1986a, 1986b). This kind of information can be clinically useful when there may be no ABR response or no hearing on the involved side. Testing the better side, if it yields a "contralateral effect" could be clinically insightful.

Repetition Rate Shifts

It has been thought for many years that increasing the rep rate during ABR testing would place greater physiological strain on the auditory nervous system, and in turn may uncover subtle brainstem abnormalities (see Hall, 1992, pp 138–141). There have been a number of reports that have shown ABR abnormalities at high rep rates, which were not as obvious at lower rates of stimulation (see Hall, 1992 for review). However, there have been questions raised regarding the value of rep rate functions in the diagnosis of auditory system compromise. Some reports indicate that when rep rate functions are abnormal other ABR indices are also abnormal, and therefore the finding of rep rate abnormalities does not distinctly contribute to a reliable diagnosis (Musiek and Lee, 1995; Musiek et al., 1996; see also Hall, 1992). A physiological consideration regarding rep rate functions is that many auditory neurons are capable of responding at much higher rates than most ABR equipment can deliver (usually <100 clicks per second), hence, the auditory system is not challenged as much as was originally thought. One of the most under-researched areas of ABR is the presentation of stimuli at very high rates. Procedures that include maximum length sequences (MLS) and continuous loop averaging deconvolution (CLAD) allow extremely high rates of presentation (up to or even greater than 700 to 800 clicks per second) that would physiologically challenge the brainstem pathways. Some valuable work has been done in this area, but more applied research should be done to recognize the full value of these procedures (see Burkard et al., 1990; Delgado and Özdamar, 2004; Musiek et al., 2007).

Sensitivity and Specificity of ABR to Brainstem Lesions

Overall, it appears that ABR is not as sensitive to brainstem lesions as it is to eighth nerve tumors. A study that investigated the sensitivity for a variety of brainstem lesions found the hit rate was approximately 80%, which was about 10% lower than that of acoustic tumors (Musiek et al., 1996). However, it appears that the ABR hit rate for brainstem involvement is dependent on the type of disorder (Musiek, 1991). For example, ABR abnormalities for intrinsic brainstem lesions are high (>95%), whereas for various degenerative disorders the hit rates are only moderate. Much of the variance is related to the precise location of the lesion (as mentioned earlier, sometimes the auditory pathways are not involved when a brainstem lesion exists), the nature and severity of the disorder, and the particular approach(es) for using ABR in the presence of these disorders. At the risk of oversimplification and brevity, the following is offered as a brief synopsis of ABR findings in some selected brainstem disorders.

Survey of ABR and Specific Brainstem Disorders

Multiple Sclerosis

MS is classified as a demyelinating disease that is often progressive. The onset of the disease is generally in early adulthood and the disease occurs more often in cold climates. The symptoms of the disease often wax and wane, with periods of remission offset by exacerbations of symptoms. Central to the pathophysiology is the damage to the myelin sheaths of the axons of nerve fibers, which can occur in both the peripheral and central nervous systems. The symptoms of the disease depend on the site of involvement. That is, if the auditory nerve fibers are involved, auditory symptoms will often be noted. Even though most of the auditory brainstem pathways are myelinated, only a portion of the auditory nerve tract is myelinated (see Musiek et al., 2012, pp 297–299).

ABR is a useful tool for the evaluation of MS (see Figure 13.6). It can help in the diagnosis and in monitoring

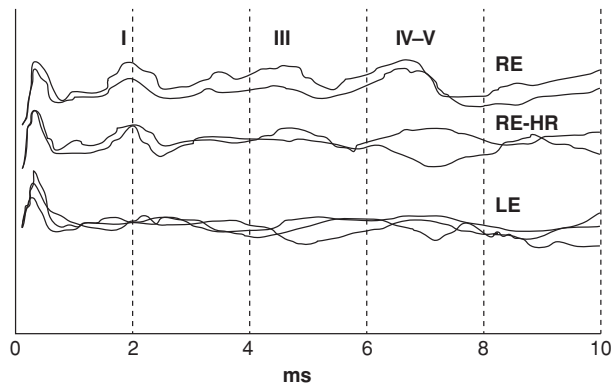


FIGURE 13.6 An ABR from a young adult with multiple sclerosis and a slight high-frequency hearing loss [Key: RE, right ear; LE, left ear; RE-HR, right ear, high rep rate @ 77.9 clicks per second]. [Adapted from Musiek FE, Shinn JB, Jirsa RE. [2007] The auditory brainstem response in auditory nerve and brainstem dysfunction. In: Burkard RF, Don M, Eggermont JJ, eds. *Auditory Evoked Potentials: Basic Principles and Clinical Application.* Baltimore, CA: Lippincott Williams & Wilkins; pp 291–312 with permission.]

the disease's effects on the auditory system. Also, it can be of value in determining if new auditory symptoms in a patient with known MS are a result of MS affecting the auditory system or some other cause. The sensitivity of ABR for MS can be somewhat misleading. Reports indicate the hit rate is quite high, whereas others find it is only mediocre (Levine et al., 1993; Musiek et al., 1994). Much of this variance is related to the fact that in MS the auditory brainstem tract is not always involved (Levine et al., 1993; Musiek et al., 2007). Extended central conduction times, that is, extended Waves I–III, III–V, or I–V intervals, are commonly seen in patients with MS. Further, ABR findings can be influenced by the degree of involvement and the time of evaluation relative to an exacerbation of symptoms. During or close to a period of exacerbation, the ABR could be absent only to recover over a period of days.

Tumors of the Brainstem

Tumors of the brainstem are generally divided into two main groups: extra- and intra-axial. Extra-axial lesions are those that encroach on the brainstem from outside of this structure. For example, tumors of cranial nerves IV through VIII and of the cerebellum as well meningiomas can affect the brainstem and would be considered extra-axial lesions (Figure 13.7). Perhaps one of the more common extra-axial tumors noted by audiologists is the vestibular schwannoma

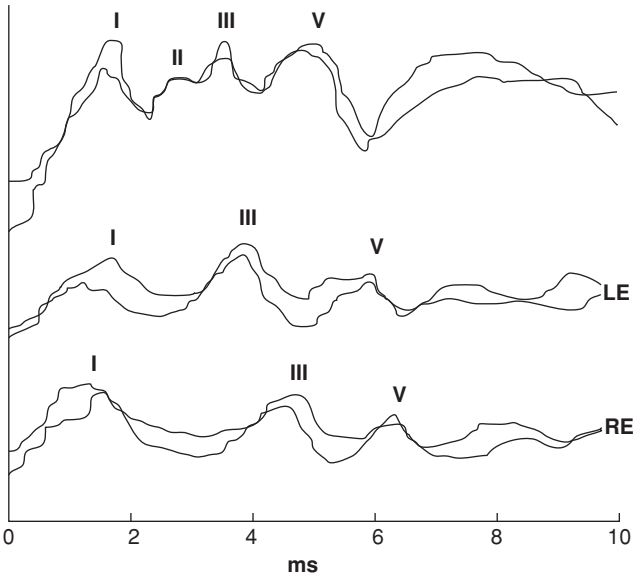


FIGURE 13.7 The top tracing is a normal ABR. The next two tracings are from a patient with a meningioma in the right cerebellopontine angle. Note the extended I–III (3.0 ms) and I–V (4.7 ms) interwave intervals on the involved side. (Reprinted from Musiek FE, Shinn JB, Jirsa RE. (2007) The auditory brainstem response in auditory nerve and brainstem dysfunction. In: Burkard RF, Don M, Eggermont JJ, eds. *Auditory Evoked Potentials: Basic Principles and Clinical Application.* Baltimore, CA: Lippincott Williams & Wilkins; pp 291–312 with permission.)

that grows large enough (usually 2 cm or greater) to affect the brainstem. There remains a paucity of data on extra-axial lesions and ABR sensitivity, but some analyses infer hit rates of around 80% or slightly less for a variety of extra-axial lesions (Musiek et al., 1994, pp 356–358). With the exception of an eighth nerve tumor, most extra-axial lesions, like MS, result in extended central conduction times, though the particular site of involvement does influence which of the specific ABR indices will be affected (see Figure 13.7).

Intra-axial tumors arise from within the brainstem with the most common being gliomas (see Hall, 1992, 2007). Again, there is not a wealth of data, but it appears that ABR is highly sensitive to intra-axial tumors (>90%) (Musiek et al., 1994). Similar to other brainstem lesions, IWIs are often abnormal when intra-axial tumors are present.

Hyperbilirubinemia

High bilirubin levels can be seen in 1 to 3 per 100,000 healthy newborns. If blood serum levels exceed 20 mg/dL it is of clinical concern even though this may not lead to a clinical problem. If bilirubin (unconjugated) is at high levels and crosses the blood–brain barrier, jaundice, HB, and even kernicterus (severe disease state) can occur. Most of the time HB can be easily managed by phototherapy. However, recently HB has become more of a problem because mother and infants are released from the hospital 1 day after giving birth and bilirubin levels generally peak on about the fourth day after birth. Because of this scenario, treatment is often delayed and bilirubin levels increase (see Musiek et al., 2012, pp 309–314).

Audiologists should be aware that high bilirubin levels can damage the CNS and the central auditory nervous system. Specifically, the CN is often damaged resulting in hearing difficulties (Dublin, 1985). Even though newborns with HB can be successfully treated, some manifest auditory problems. Though the incidence of abnormal ABR measures varies considerably (13% to 60%), there is no question that the ABR is a valuable test for detecting the central auditory problem at an early age (Kuriyama et al., 1986; Sharma et al., 2006). Increased absolute latency of Wave V appears to be a relatively common finding in patients with HB and ABR central conduction times (i.e., IWIs) are often abnormal in individuals with this disorder (Sharma et al., 2006). Since the HB lesion site is in the central auditory system, otoacoustic emissions may not be a prudent choice for screening these infants. It is also important to note that with the timely application of therapy, HB resolves and the ABR improves, often to within normal limits (Kuriyama et al., 1986).

Heavy Metal Exposure

Heavy metals such as lead, mercury, cadmium, and arsenic can compromise auditory function and the ABR can be useful in determining the effects from such exposures. Exposure to

heavy metals can slow nerve conduction, generally manifesting as CNS and central auditory system dysfunction. Clearly the greater the level of heavy metal found in the blood, the greater the possibility exists for cognitive and hearing disturbances (see Musiek et al., 2012, pp 311–317). Increased central conduction times during ABR testing are commonly noted in individuals exposed to heavy metals (Araki et al., 2000); however, interesting data from Counter (2002) showed little effect of lead exposure on the ABR in lead glaze workers in South America. These individuals experienced constant exposure to lead in their daily lives, yet normal or near-normal ABRs were found. (Perhaps their central auditory nervous system adapted to the lead exposure.) Nonetheless, in most situations, ABR should be one of the tests administered to evaluate the neurotoxic effect of heavy metals.

Head Injury

It has become a common role for audiologists to be involved in the evaluation of individuals with a head injury. Key information relates that over half of the patients including children and adults with head injury (i.e., traumatic brain injury) may have central auditory deficits (Bergemalm and Lyxel, 2005). The pathophysiology of a head injury is related to the high acceleration and deceleration of the brain within the confines of the cranium. This can result in contusions, hemorrhage, and axon injury (including myelin damage). Also, secondary effects such as ischemia, hypoxia, edema, and increased intracranial pressure are seen (Musiek and Chermak, 2008). The ABR is commonly employed in the evaluation of head injury to check the physiological status of the brainstem (see Figure 13.8). This becomes even more important when the patient involved develops auditory symptoms after the event. Head injuries do not usually result in damage to the brainstem pathways; thus, normal ABRs should be obtained for many patients with head injuries. Therefore, normal ABR findings in head trauma cannot and should not be an indictment against the utility of ABR as an evaluation tool.

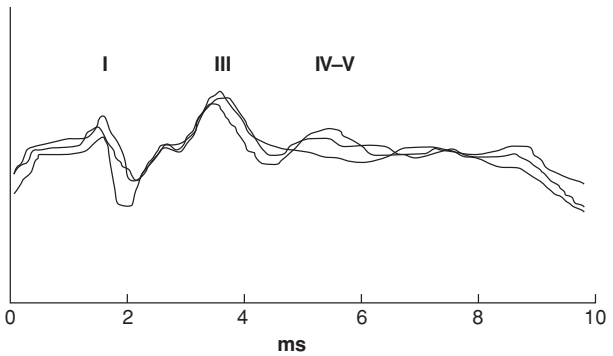


FIGURE 13.8 The ABR from a patient with a head injury (pontine contusion). This patient's symptoms of "difficulty in hearing" on the involved side resulted in a detailed audiologic evaluation including the ABR. (Reprinted from Musiek FE, Shinn JB, Jirsa RE. (2007) The auditory brainstem response in auditory nerve and brainstem dysfunction. In: Burkard RF, Don M, Eggermont JJ, eds. *Auditory Evoked Potentials: Basic Principles and Clinical Application.* Baltimore, CA: Lippincott Williams & Wilkins; pp 291–312 with permission.)

A review of significant studies shows ABR to be abnormal in about 50% of people with head trauma (Musiek et al., 2012), with the I–V interval of central conduction time being perhaps the most sensitive ABR index (Bergemalm and Borg, 2001). Also, increases in the latencies of Wave V and the I–V IWI of the ABR have been shown with increases in the severity of the head injury or traumatic brain injury (Munjal et al., 2010). The use of high rep rate ABRs may provide reliable assessment of patients with traumatic brain injuries (see Musiek et al., 2012, p 319); however, more studies are needed to determine if this is a clinically useful approach to detect auditory abnormalities associated with head injuries. Another ABR measure, the V–I amplitude ratio, as discussed earlier in this chapter, may also be of value in evaluating a head injury, although more research is also needed on this ABR measure.

EFFECTS OF COCHLEAR HEARING LOSS ON THE ABR

Degree and Configuration

The presence of a cochlear hearing loss is often encountered when a patient is being seen for a neurodiagnostic evaluation. If present, a peripheral hearing loss can potentially affect the response recorded, frequently resulting in test results that could be mistakenly attributed to the presence of a neural site of lesion. Unfortunately, the variable effects of a cochlear hearing loss are affected by a number of factors, such as the severity, slope and audiometric configuration of the hearing loss, age and/or gender, and complex interactions among these variables (Hall, 2007; Jerger and Johnson, 1988; Watson, 1999). As a result, many of the ABR diagnostic indices used to identify a retrocochlear hearing loss may not be available or their measurements may lead to a false-positive finding when the audiologist is attempting to render a clinical decision re: the presence of a retrocochlear lesion.

The one consistent observation is that the interwave latency (IWI) measures are largely unaffected by a peripheral hearing loss (both conductive and cochlear). Various studies have shown subtle effects of hearing loss on IWIs, but these effects have been subtle and often decrease the IWIs (see Fowler and Durrant, 1994; Shepard et al., 1992). Therefore, if Wave I of the ABR is present, decisions regarding the presence or absence of eighth nerve and/or brainstem abnormalities are relatively straightforward. Unfortunately, many patients with a moderate to severe

sensory/neural hearing loss lack an identifiable Wave I component, rendering the derivation of one or more of the typical IWI measures (I–III, III–V, I–V) impossible. In the absence of measureable IWIs, the audiologist must rely on other ABR measures, such as the absolute latency of Wave V, an ILD for Wave V, and/or the absence of ABR waves, all of which can be affected by a cochlear hearing loss or retrocochlear involvement.

When unfiltered click stimuli are used to derive an ABR the response is largely being generated by activity in the basal region of the cochlea since the signal reaching the basilar membrane is shaped by the filter effects of the outer and middle ears and the response characteristics of the transducer. Although there are some differences in the resonance peak of different transducers, the resonance peaks for most transducers used in electrophysiological assessments tend to occur in the higher frequency range (usually peaking around 3 to 4 kHz), which gives the stimulus reaching the cochlea a high-frequency emphasis. This transducer response (i.e., the high-frequency emphasis), coupled with the fact that the traveling wave stimulates the base of the cochlea before it stimulates the more apical regions of the basilar membrane, leads to an ABR response that is predominately affected by the status of the high-frequency neurons (Fowler and Durrant, 1994; Musiek et al., 2007). For these reasons, the effects of a cochlear hearing loss on the ABR can be quite variable and are highly dependent on the frequency range (low vs. high frequency), the slope or audiometric configuration (flat, mildly sloping, sharply sloping), and the severity of the hearing loss (see Rosenhamer, 1981).

In cases of low-frequency sensory/neural hearing loss, there tends to be little or no effect on the click-evoked ABR since the neurobiologic response is biased toward the high frequencies as discussed above. As the high-frequency cochlear region is not compromised or only minimally compromised in patients with hearing losses confined primarily to the low frequencies, the ABR waveform indices used for neurodiagnostic purposes typically fall within the normal range.

A high-frequency hearing loss will produce variable results. In a mid- to high-frequency hearing loss, Wave V latency is relatively stable up to 60 dB HL, but it increases as the degree of hearing loss at 4 kHz increases. However, the extent of the latency shift is affected not only by the degree of hearing loss, but also by the slope of the hearing loss (Fowler and Durrant, 1994). In cases of a relatively flat or a mildly sloping hearing loss of mild to moderate severity, the effects of hearing loss on the ABR derived at a high stimulus level (e.g., 80 dB nHL) are minimal or nonexistent. Although some reduction in amplitude may be seen, presumably related to the reduction of neural units, the other ABR indices are typically not affected.

With a sharply sloping severe high-frequency sensory/neural hearing loss, the earlier waves may be reduced in amplitude or absent, and any waves that are present are likely to be delayed in their absolute latencies (Rosenhall, 1981; Rosenhamer, 1981). These changes in the earlier waves are related to (1) the increased travel time for the traveling wave to reach the healthy or active regions of the cochlea and (2) the effective reduction in the stimulus intensity caused by the compromise of the fibers of the basal end of the cochlea, that is, the fibers which respond with the shortest latencies. If Wave I is present and an I–V IWI can be determined, the measured IWI will typically fall within normal limits, contraindicating a retrocochlear lesion. However, if Wave I is not identifiable, the differentiation between a cochlear and retrocochlear lesion becomes more tenuous since both sites of lesion can result in abnormal Wave V latencies and potentially an abnormal ILD especially in cases of unilateral or asymmetrical hearing losses.

Watson (1999) conducted a relatively large-scale investigation designed to examine the effects of a cochlear hearing loss on the specificity and false-positive rates of the various ABR indices. He examined a number of hearing loss variables (including level, slope, and general shape of the hearing loss) in a large number of patients with unilateral or asymmetrical symptoms who were referred for ABR screening for potential retrocochlear involvement. His investigation of 566 ears from 306 participants showed that 85 ears (15%) failed the ABR test. On follow-up, retrocochlear hearing loss was confirmed by the author in seven participants and one additional individual showed an abnormal meatal narrowing (the method of confirmation was unspecified, but assumed to be imaging or surgical confirmation). It was also reported that MRIs were performed on an unspecified number of subjects with normal ABR results and that all ABR "passes" had been under review for at least 3 years (presumably without any additional identifications of retrocochlear lesions). The ABR results for all of the participants with confirmed retrocochlear hearing loss were abnormal, which indicated that the ABR diagnostic measures were highly sensitive for retrocochlear involvement. In patients diagnosed as free of retrocochlear problems, the I–V interval was found to be the most specific measure (>90%) and was relatively independent of degree of high-frequency hearing loss and slope of hearing loss. Although the degree of high-frequency hearing loss did not affect the specificity of the I–V IWI measure itself, there was an effect of the degree of hearing loss (measured at 4 kHz and also as an average threshold at 2 and 4 kHz) and the slope of the hearing loss on the detectability of Wave I, which if not detected precluded the derivation of the I–V IWI. The I–V interval was not measured in 68 of 550 ears (12%) and was found to be significant for the hearing loss at 4 kHz, the average hearing loss at 2 and 4 kHz, and the slope of the hearing loss defined as the difference in audiometric thresholds measured at 4 and 1 kHz.

Wave V specificity was high when hearing loss at 4 kHz was less than 70 dB HL, but was found to decrease with increasing hearing loss and increasing slope with the

specificity of this measure falling below 70% when hearing loss at 4 kHz exceeded 70 dB HL. Abnormal latency measures were found for 48 ears (9%) and Wave V was absent in another 8 ears (2%). Abnormalities of Wave V were found to be relatively low and independent of hearing loss at 4 kHz until this threshold exceeded 60 dB HL. Similar results were associated with increasing average hearing loss, with one in three subjects showing Wave V abnormalities when their 2- and 4-kHz average fell above 60 dB HL. Slope of the hearing loss was also found to affect the specificity of Wave V latency. Nearly half of the cases with Wave V abnormalities (24 of 56 ears) not related to retrocochlear pathology presented with hearing loss slopes measured between 1 and 4 kHz exceeding 30 dB.

A common practice among many professionals is to refer for imaging studies (MRI or CT scans) if asymmetrical or unilateral hearing losses or symptoms are noted during routine audiologic or otolaryngologic assessments, hence bypassing ABR testing in the process. The findings of this study raise questions about this common practice among some professionals. Only a very small number of individuals (~3%) were found to have retrocochlear lesions among the 330 patients who presented with asymmetrical or unilateral hearing symptoms, whereas normal ABR indices were found in the vast majority of the remaining patients. These results would suggest that ABR testing may avoid the need for more costly medical procedures where the incidence of retrocochlear involvement has been shown to be quite low, with cost savings to the healthcare industry.

Formulas to Account for High-Frequency Hearing Loss

The patient who is being evaluated with ABR for neurodiagnostic purposes will typically present with asymmetrical hearing loss. In such cases, an interaural Wave V latency difference may be noted since the absolute latency of Wave V in the affected ear may be delayed. However, a high-frequency sensory/neural hearing loss of cochlear origin may also result in similar delays of Wave V, especially if the hearing loss in the 4-kHz region falls within the moderate to moderately severe range. Under these circumstances, clinicians may be faced with the challenge of determining whether the delayed Wave V latency in the affected ear or poorer hearing loss ear, and the interaural asymmetry that results, is related to cochlear compromise or eighth nerve/auditory brainstem involvement.

A number of procedures have been proposed in an effort to control for latency shifts which may be caused by peripheral hearing loss. Some authors have proposed formulas based on the threshold measure at 4 kHz, whereas others have attempted to account for not only the extent of the hearing loss in the high-frequency range, but also the slope of the hearing loss as the basis for their adjustments Selters and Brackmann (1977) recommended that 0.1 ms be subtracted from the absolute latency measure for Wave V for each 10-dB increase in a hearing loss above 50 dB HL. An ILD exceeding 0.2 ms following the correction for hearing loss above 50 dB HL would be considered abnormal. However, many individuals with a similar hearing loss at 4 kHz have differing Wave V latencies. As a result, the interpretation of an ILD difference following the application of adjustments to the latency of Wave V to account for a high-frequency hearing loss can result in inaccurate clinical decisions.

Prosser and Arslan (1987) proposed a diagnostic index (ΔV) to differentiate between a cochlear and a retrocochlear lesion when a sensory/neural hearing loss was evident. In this approach, a ΔV index is calculated based on the following formula: $\Delta V = L_p\ (90) - L_n\ (90–x)$, where L_p (90) is the latency of Wave V derived at 90 dB nHL, L_n (90) is the Wave V latency derived from a normal intensity function, and x is the average hearing loss at 2 and 4 kHz. These authors suggest that a negative ΔV would be consistent with cochlear hearing loss, whereas a positive ΔV would suggest a retrocochlear lesion. Further, a regression analysis on their cochlear hearing data with a 95% confidence interval for cochlear hearing loss was conducted and indicated that this measure was effective in differentiating cochlear and retrocochlear hearing losses.

Rosenhamer et al. (1981) offered a correction method for high-frequency hearing loss as a guideline for ABR interpretation when an asymmetrical hearing loss was present. These authors recommended that 0.1 ms be subtracted from the Wave V latency for every 10 dB of increased hearing loss above 30 dB HL; loss at 4 kHz greater than 50 dB HL. However, the authors noted the value of using sensation levels as opposed to hearing levels or sound pressure levels when evaluating patients with unilateral or asymmetrical hearing losses, which presumably eliminates the need for the application of a correction factor.

Building on this later concept, Jerger and Johnson (1988) offered the following guidelines for determining click stimulus intensity level for neurodiagnostic evaluations, which attempt to adjust for sensation level by increasing the stimuli intensity level based on the puretone average for high frequencies (1, 2, 4 kHz). Click intensity levels of 70, 80, 90, and 100 dB HL are recommended for male (but not female) patients with high-frequency averages in the following ranges, respectively: 0 to 19, 20 to 39, 40 to 59 and 60 to 79 dB HL. Other researchers (Hyde and Blair, 1981) have additionally suggested that different correction factors are needed for young versus older patients with comparable hearing losses.

Although many researchers support the use of correction factors to account for hearing loss, their application in neurodiagnostic assessments must be used with appropriate levels of caution as a number of factors can affect the latency of Wave V when cochlear hearing loss is present. For example, Wave V latency can vary considerably among patients with the same degree of hearing loss, and interactions between

factors such as age, gender, and hearing impairment have been shown to exist (see Rosenhall, 1981; Watson, 1999). These factors limit the universal application of correction factors.

As the I–V index has been shown to be largely unaffected by peripheral hearing loss, the best approach in addressing the challenges imposed by the presence of a potential cochlear hearing loss would be to take steps to increase the likelihood of deriving a Wave I. In many cases where Wave I is not observed using common procedures (e.g., surface electrodes placed at A1 and A2; stimulus intensity of 70 or 80 dB nHL), an identifiable Wave I can be elicited if the intensity of the eliciting stimulus is increased and/or extratympanic or intracanal electrodes are used. If Wave I can be derived, then an I–V IWI can be determined. As the I–V IWI has been shown to be highly sensitive to retrocochlear hearing loss and is not affected by cochlear and/or conductive hearing loss, the presence of an extended IWI would be a positive finding, which has been shown to be highly sensitive for retrocochlear involvement.

SUMMARY AND CONCLUDING COMMENTS

This chapter has reviewed the perspectives of ABR in reference to auditory nerve and brainstem disorders. The ABR remains a powerful and, in many cases, a necessary tool for shedding light on disorders of the brainstem and auditory nerve—especially from the audiologist's point of view. The early investigators of ABR who pioneered the application of this then new procedure showed how it could be utilized in many ways and across many disciplines. The ABR in its diagnostic infancy triggered a focus on the importance of audiology and audiologists because of its impact on otologic and neurologic diagnosis. These interactions across disciplines benefitted patients as well as research into hearing disorders.

The diagnostic use of ABR is dependent on the anatomy of the generators of its various waves. Though some minor controversies still exist, the generator sites of ABR are known and because of this, the integrity of the auditory nerve and brainstem pathways can be assessed. Aage Møller's work on ABR generators in humans in the 1980s was a key advance for not only auditory science, but also clinical applications. Without firm knowledge of the generator sites, clinical advances could not have been realized.

FOOD FOR THOUGHT

A segment of this chapter discussed a controversial issue related to referring directly for imaging and bypassing ABR for vestibular schwannoma detection. This practice has its advantages, but recently it has become clear that there is an over-referral for expensive imaging testing with a poor ratio of "hits" compared to negative findings. The ABR, if applied appropriately, could help resolve this problem. This issue sorely needs to be revisited again, especially in light of increasing healthcare costs. The model offered by Urben et al. (1999) deserves more attention regarding this issue.

To optimize the use of ABR in retrocochlear diagnosis, an accurate perspective on various ABR measurements or indices needs to be realized. Interwave latency intervals (I–III, III–V, I–V) are solid indicators of auditory nerve and brainstem function. In addition, they are essentially unaffected by hearing loss, a situation that does not exist for other ABR indices. The ILD though influenced by hearing loss still remains highly applicable and very sensitive to eighth nerve involvement, but interestingly not brainstem involvement. Vestibular schwannomas and brainstem disorders that affect the auditory pathways yield various ABR patterns that are helpful in measuring the integrity of the neural auditory tracts. For example, Waves I–III extensions are often indicators of eighth nerve or low brainstem involvement. Absent late waves or extended Waves III–V intervals argue strongly for brainstem compromise. It is also important for the clinician to realize that there are a variety of auditory nerve and brainstem disorders for which ABR testing can be applied. In many instances, imaging may not be helpful in defining a disorder, whereas ABR becomes the best and perhaps the only avenue for effective diagnosis. How can the audiologist participate in the identification of ANSD, head injury with auditory involvement, various neurotoxic effects, and some subtle early auditory degenerative diseases if they do not utilize the ABR?

ACKNOWLEDGMENTS

We would like to acknowledge support from the Royal Arch Research Assistance and the assistance of Julianne Ceruti, Stephanie Waryasz, and Maggi Dunlap in the preparation of this chapter.

REFERENCES

Antonelli AR, Bellotto R, Grandori F. (1987) Audiologic diagnosis of central versus eighth nerve and cochlear auditory impairment. *Audiology.* 26 (4), 209–226.

Araki S, Sato H, Yokoyama K, Murata K. (2000) Subclinical neurophysiological effects of lead: a review on peripheral, central, and autonomic nervous system effects in lead workers. *Am J Ind Med.* 37 (2), 193–204.

Arriaga MA, Carrier D, Houston GD. (1995) False-positive magnetic resonance imaging of small internal auditory canal tumors: a clinical, radiologic, and pathologic correlation study. *Otolaryngol Head Neck Surg.* 113 (1), 61–70.

Bakkouri WE, Kania RE, Guichard J-P, Lot G, Herman P, Huy PT. (2009) Conservative management of 386 cases of unilateral vestibular schwannoma: tumor growth and consequences for treatment. *J Neurosurg.* 110 (4), 662–669.

Bebin J. (1979) Pathophysiology of acoustic tumors. In: House W, Luetje C, eds. *Acoustic Tumors.* Baltimore, MD: University Park; pp 45–83.

Bederson JB, von Ammon K, Wichmann WW, Yasargil MG. (1991) Conservative treatment of patients with acoustic tumors. *Neurosurgery.* 28 (5), 646–650.

Bergemalm PO, Borg E. (2001) Long-term objective and subjective audiologic consequences of closed head injury. *Acta Otolaryngol.* 121 (6), 724–734.

Bergemalm PO, Lyxel B. (2005) Appearances are deceptive? Long-term cognitive and central auditory sequelae from closed head injury. *Int J Audiol.* 44 (1), 39–49.

Brackmann DE, Selters WA. (1979) Brainstem electric audiometry: acoustic neurinoma detection. *Rev Laryngol Otol Rhinol (Bord).* 100 (1–2), 49–51.

Buchman CA, Roush PA, Teagle HFB, Brown CJ, Zdanski CJ, Grose JH. (2013) Auditory neuropathy characteristics in children with cochlear nerve deficiency. *Ear Hear.* 27 (4), 399–408.

Buchwald JS, Huang C. (1975) Far-field acoustic response: origins in the cat. *Science.* 189 (4200), 382–384.

Burkard R, Shi Y, Hecox KE. (1990) A comparison of maximum length and Legendre sequences for the derivation of brain stem auditory-evoked responses at rapid rates of stimulation. *J Acoust Soc Am.* 87 (4), 1656–1664.

Bush ML, Jones RO, Shinn JB. (2008) Auditory brainstem response threshold differences in patients with vestibular schwannoma: a new diagnostic index. *Ear Nose Throat J.* 87 (8), 458–462.

Cashman MZ, Stanton SG, Sagle C, Barber HO. (1993) The effect of hearing loss on ABR interpretation: use of a correction factor. *Scand Audiol.* 22 (3), 153–158.

Chiappa KH. (1983) *Evoked Potentials in Clinical Medicine.* New York, NY: Raven Press.

Chiappa KH. (1989) *Evoked Potentials in Clinical Medicine.* 2nd ed. New York, NY: Raven Press.

Counter SA. (2002) Brainstem neural conduction biomarkers in lead-exposed children of Andean lead-glaze workers. *J Occup Environ Med.* 44 (9), 855–864.

Daly DM, Roeser RJ, Aung MH, Daly DD. (1977) Early evoked potentials in patients with acoustic neuroma. *Electroencephalogr Clin Neurophysiol.* 43 (2), 151–159.

Delgado RE, Özdamar Ö. (2004) Deconvolution of evoked responses obtained at high stimulus rates. *J Acoust Soc Am.* 115 (3), 1242–1251.

Don M, Eggermont JJ. (1978) Analysis of the click-evoked brainstem potentials in man using high-pass noise masking. *J Acoust Soc Am.* 63 (4), 1084–1092.

Don M, Kwong B, Tanaka C, Brackmann D, Nelson R. (2005) The stacked ABR: a sensitive and specific screening tool for detecting small acoustic tumors. *Audiol Neurootol.* 10 (5), 274–290.

Don M, Masuda A, Nelson R, Brackmann D. (1997) Successful detection of small acoustic tumors using the stacked derived-band auditory brain stem response amplitude. *Am J Otol.* 18 (5), 608–621.

Dublin WB. (1985) The cochlear nuclei-pathology. *Otolaryngol Head Neck Surg.* 93 (4), 448–463.

Fowler CG, Durrant JD. (1994) The effects of peripheral hearing loss on the auditory brainstem response. In: Jacobson JT, ed. *Principles and Applications in Auditory Evoked Potentials.* Boston, MA: Allyn & Bacon; pp 237–250.

Gerling IJ. (1989) Interaction of stimulus parameters on the auditory brain stem response: a normal variant. *Ear Hear.* 10 (2), 117–123.

Gordon ML, Cohen NL. (1995) Efficacy of auditory brainstem response as a screening test for small acoustic neuromas. *Am J Otol.* 16 (2), 136–139.

Hall JW. (1992) *Handbook of Auditory Evoked Potentials.* Boston, MA: Allyn & Bacon.

Hall JW. (2007) *New Handbook of Auditory Evoked Responses.* Boston, MA: Allyn & Bacon.

House JW, Bassim MK, Schwartz M. (2008) False-positive magnetic resonance imaging in the diagnosis of vestibular schwannoma. *Otol Neurotol.* 29 (8), 1176–1178.

Hyde ML, Blair RL. (1981) The auditory brainstem response in neuro-otology: perspectives and problems. *J Otolaryngol.* 10 (2), 117–125.

Ito K, Ishimoto SI, Karino S. (2007) Isolated cochlear nerve hypoplasia with various internal auditory meatus deformities in children. *Ann Otol Rhinol Laryngol.* 116 (7), 520–524.

Jannetta PJ. (1967) Arterial compression of the trigeminal nerve at the pons in patients with trigeminal neuralgia. *J Neurosurg.* 26 (suppl 1), 159–162.

Jerger J, Johnson K. (1988) Interaction of age, gender, and sensorineural hearing loss on ABR latency. *Ear Hear.* 9 (4), 168–176.

Jerger S, Jerger J. (1981) *Auditory Disorders.* Boston, MA: Little, Brown.

Katz RC, Wilson L, Frazer N. (1994) Anxiety and its determinants in patients undergoing magnetic resonance imaging. *J Behav Ther Exp Psychiatry.* 25 (2), 131–134.

Kotlarz JP, Eby TL, Borton TE. (1992) Analysis of the efficiency of retrocochlear screening. *Laryngoscope.* 102 (10), 1108–1112.

Kuriyama M, Konishi Y, Mikawa H. (1986) The effect of neonatal hyperbilirubinemia on the auditory brainstem response. *Brain Dev.* 8 (3), 240–245.

Levine RA, Gardener JC, Stufflebeam SM, Carlisle EW, Furst M, Rosen BR, et al. (1993) Effects of multiple sclerosis brainstem lesions on sound lateralization and brainstem auditory evoked potentials. *Hear Res.* 68 (1), 73–88.

Møller AR. (2000) *Hearing: Its Physiology and Pathophysiology.* New York, NY: Academic Press.

Møller AR, Jannetta PJ. (1985) Neural generators of the auditory brainstem response. In: Jacobson JT, ed. *The Auditory Brainstem Response.* San Diego, CA: College-Hill Press; pp 13–32.

Møller MB, Møller AR. (1983) Brainstem auditory evoked potentials in patients with cerebellopontine angle tumors. *Ann Otol Rhinol Laryngol.* 92 (6 Pt 1), 645–650.

Munjal SK, Panda NK, Pathak A. (2010) Relationship between severity of traumatic brain injury (TBI) and extent of auditory dysfunction. *Brain Inj.* 24 (3), 525–532.

Murphy MR, Selesnick SH. (2002) Cost-effective diagnosis of acoustic neuromas: a philosophical, macroeconomic, and technological decision. *Otolaryngol Head Neck Surg.* 127 (4), 253–259.

Musiek FE. (1991) Auditory evoked responses in site-of-lesion assessment. In: Rintelmann WF, ed. *Hearing Assessment.* 2nd ed. Austin, TX: Pro-Ed; pp 383–428.

Musiek FE, Baran JA, Shinn JB, Jones RO. (2012) *Disorders of the Auditory System.* San Diego, CA: Plural Publishing.

Musiek FE, Bornstein SP, Hall JW, Schwaber MK. (1994) Auditory brainstem response: neurodiagnostic and intraoperative applications. In: Katz J, ed. *Handbook of Clinical Audiology.* 4th ed. Baltimore, MD: Williams & Wilkins; pp 351–374.

Musiek FE, Chermak GD. (2008) Testing and treating CAPD in head injury patients. *Hear J.* 61 (6), 36–38.

Musiek FE, Johnson GD, Gollegly KM, Josey AF, Glasscock ME. (1989) The auditory brain stem response interaural latency difference (ILD) in patients with brain stem lesions. *Ear Hear.* 10 (2), 131–134.

Musiek FE, Josey AF, Glasscock ME, 3rd. (1986a) Auditory brainstem response in patients with acoustic neuromas: wave presence and absence. *Arch Otolaryngol Head Neck Surg.* 112 (2), 186–189.

Musiek FE, Josey AF, Glasscock ME. (1986b) Auditory brainstem response: interwave measurements in acoustic neuromas. *Ear Hear.* 7 (2), 100–105.

Musiek FE, Kibbe K. (1986) Auditory brainstem response wave IV-V abnormalities from the ear opposite large cerebellopontine lesions. *Am J Otol.* 7 (4), 253–257.

Musiek FE, Lee WW. (1995) The auditory brain stem response in patients with brain stem or cochlear pathology. *Ear Hear.* 16 (6), 631–636.

Musiek FE, McCormick CA, Hurley RM. (1996) Hit and false-alarm rates of selected ABR indices in differentiating cochlear disorders from acoustic tumors. *Am J Audiol.* 5 (1), 90–96.

Musiek FE, Shinn JB, Jirsa RE. (2007) The auditory brainstem response in auditory nerve and brainstem dysfunction. In: Burkard RF Don M, Eggermont JJ, eds. *Auditory Evoked Potentials: Basic Principles and Clinical Application.* Baltimore, CA: Lippincott Williams & Wilkins; pp 291–312.

Musiek FE, Weider DJ, Mueller RJ. (1982) Audiologic findings in Charcot-Marie-Tooth disease. *Arch Otolaryngol.* 108 (9), 595–599.

Nicholson G, Corbett A. (1996) Slowing of central conduction in X-linked Charcot-Marie-Tooth neuropathy shown by brain stem auditory evoked responses. *J Neurol Neurosurg Psychiatry.* 61 (1), 43–46.

Parker DJ, Thornton AR. (1978a) Frequency specific components of the cochlear nerve and brainstem evoked responses of the human auditory system. *Scand Audiol.* 7 (1), 53–60.

Parker DJ, Thornton AR. (1978b) The validity of the derived cochlear nerve and brainstem evoked responses of the human auditory system. *Scand Audiol.* 7 (1), 45–52.

Patzkó A, Shy ME. (2010) Update on Charcot-Marie-Tooth disease. *Curr Neurol Neurosci Rep.* 11 (1), 78–88.

Prosser S, Arslan E. (1987) Prediction of auditory brainstem wave V latency as a diagnostic tool of sensorineural hearing loss. *Audiology.* 26 (3), 179–187.

Rance G, Ryan MM, Bayliss K, Gill K, O'Sullivan C, Whitechurch M. (2012) Auditory function in children with Charcot-Marie-Tooth disease. *Brain.* 135 (Pt 5), 1412–1422.

Robinson K, Rudge P. (1975) Auditory evoked responses in multiple sclerosis. *Lancet.* 305 (1719), 1164–1166.

Rosenhall U. (1981) Brain stem electrical responses in cerebellopontine angle tumours. *J Laryngol Otol.* 95 (9), 932–940.

Rosenhamer HJ. (1981) The auditory brainstem response (ABR) in cochlear hearing loss. *Scand Audiol Suppl.* 13, 83–93.

Rosenhamer HJ, Lindström B, Lundborg T. (1981) On the use of click-evoked electric brainstem responses in audiological diagnosis. III. Latencies in cochlear hearing loss. *Scand Audiol.* 10 (1), 3–11.

Selters WA, Brackmann DE. (1977) Acoustic tumor detection with brain stem electric response audiometry. *Arch Otolaryngol.* 103 (4), 181–187.

Sharma R, Grover N, Sankhyan N, Sharma ML. (2006) Auditory brainstem responses in neonatal hyperbilirubinemia and effect of therapy. *Indian J Otolaryngol Head Neck Surg.* 58 (4), 340–342.

Shepard NT, Webster JC, Baumen M, Schuck P. (1992) Effect of hearing loss of cochlear origin on the auditory brain stem response. *Ear Hear.* 13 (3), 173–180.

Starr A, Achor J. (1975) Auditory brain stem responses in neurological disease. *Arch Neurol.* 32 (11), 761–768.

Starr A, Hamilton AE. (1976) Correlation between confirmed sites of neurological lesions of far-field auditory brainstem responses. *Electroencephalogr Clin Neurophysiol.* 41 (6), 595–608.

Stockard JJ, Stockard JE, Sharbrough FW. (1977) Detection and localization of occult lesions with brainstem auditory responses. *Mayo Clin Proc.* 52 (12), 761–769.

Stockard JJ, Stockard JE, Sharbrough FW. (1978) Non-pathologic factors influencing brainstem auditory evoked potentials. *Am J EEG Technol.* 18, 177–209.

Taiji H Morimoto N, Matsunaga T. (2012) Unilateral cochlear nerve hypoplasia in children with mild to moderate hearing loss. *Acta Otolaryngol.* 132 (11), 1160–1167.

Tanaka H, Komatsuzaki A, Hentona H. (1996) Usefulness of auditory brainstem responses at high stimulus rates in the diagnosis of acoustic neuroma. *ORL J Otorhinolaryngol Relat Spec.* 58 (4), 224–228.

Urben SL, Benninger MS, Gibbens ND. (1999) Asymmetric sensorineural hearing loss in a community-based population. *Otolaryngol Head Neck Surg.* 120 (6), 809–814.

Watson DR. (1999) A study of the effects of cochlear loss on the auditory brainstem response (ABR) specificity and false positive rate in retrocochlear assessment. *Audiology.* 38 (3), 155–164.

Zappia JJ, O'Connor CA, Wiet RJ, Dinces EA. (1997) Rethinking the use of auditory brainstem response in acoustic neuroma screening. *Laryngoscope.* 107 (10), 1388–1392.

CHAPTER 14

Auditory Brainstem Response: Estimation of Hearing Sensitivity

Linda J. Hood

INTRODUCTION

Early identification of hearing loss is now well established in the evaluation and care of newborn infants. The positive impact of identification of hearing loss in infants on language outcomes has been definitively demonstrated (e.g., Yoshinaga-Itano et al., 1998). Timelines that include identification of hearing loss before 1 month of age, thorough evaluation of an infant by 3 months of age, and implementation of management before 6 months of age are incorporated into widely recognized recommendations (e.g., JCIH, 2007; see Chapter 23). For early identification to be effective, test methods that can accurately quantify auditory threshold sensitivity in infants must be available to provide adequate follow-up of those who do not pass newborn hearing screening.

Objective measures are key components in a test battery for young children in whom, for reasons that might include developmental delays, the ability to obtain reliable responses through behavioral testing is not possible. Auditory-evoked potentials (AEPs), when used and interpreted properly, also provide a powerful method of obtaining reliable estimates of auditory sensitivity in individuals of all ages who either cannot or will not provide reliable results on behavioral hearing tests.

AEPs in general provide objective assessment of auditory function with two broad areas of application: (1) Identification of neurologic abnormalities of the VIII cranial nerve and auditory pathways and (2) estimation of hearing threshold sensitivity. Applications in both of these general areas exist across AEPs obtained from the cochlea to the cortex and in patients of all ages.

The auditory brainstem response (ABR) is an evoked potential used to both assess neural response integrity and obtain estimates of hearing thresholds. Although the ABR is not a test of hearing per se, the information obtained can be useful in estimating or predicting hearing thresholds. To accurately characterize threshold sensitivity, information must be obtained for defined frequency regions, as is standard practice in behavioral testing via puretone audiometry. Presently, there are two approaches that are considered appropriate objective measures for obtaining responses to frequency-specific stimuli in infants and young children. These methods are the ABR and the auditory steady-state response (ASSR). This chapter will focus on the ABR as it can be applied to threshold prediction; the ASSR is thoroughly described in Chapter 15.

A HISTORICAL PERSPECTIVE

AEPs from the cochlea to the cortex have been studied as possible methods to assess hearing. Indeed, Hallowell Davis' pioneering work with cortical potentials first reported in 1939 was, in part, directed toward the development of a technique to assess auditory function without the need for patient participation. Cortical responses and middle latency responses, studied in the 1950s and 1960s, proved useful in acquiring the necessary information about threshold sensitivity (e.g., Davis, 1976). However, these responses generally require patients to be awake, cooperative, and alert during testing to maintain response amplitude. Steady-state responses, recorded with a slower rate of 40 Hz and primarily cortical in origin (Galambos et al., 1981; Kuwada et al., 2002), are also an efficient method of obtaining estimates of hearing sensitivity; however, this response again is affected by sleep and sedation which limit its application in infants and young children. Thus, whereas cortical responses have higher face validity in that they evaluate a greater proportion of the auditory pathway, the applications in infants and young children are limited in the population most in need of an objective method of assessing hearing sensitivity.

If the goal of hearing testing is to determine peripheral hearing sensitivity, then measurement of responses directly from the cochlea would seem ideal. In fact, electrocochleography proved a very useful technique in the 1960s and 1970s. However, the somewhat invasive nature of transtympanic or eardrum recording sites limited its widespread clinical application, particularly in infants and young children. The relative ease of recording the ABR and its resistance to the effects of sleep and sedation have facilitated widespread use of this AEP in prediction of hearing thresholds in infants and children, as well as adults.

WHAT THE ABR TESTS

Although AEPs have proven useful in estimating hearing thresholds, it is important to remember that the ABR is NOT a test of hearing! The ABR and other evoked potentials assess neural synchrony, that is, the ability of the peripheral and central nervous system to respond to external stimulation in a synchronous manner. A synchronous neural response results from simultaneous firing of a group of neurons. Since clinical recording of responses is completed in a far-field manner via electrodes placed on the scalp, away from the source of the response, a sufficient number of neurons must fire together to yield a response of sufficient amplitude to be recorded at this distance.

When the auditory nervous system pathways are functioning normally, we can use evoked potentials to record neural responses to stimuli presented at various intensity levels. Thus, by presenting stimuli at a series of intensity levels above and below threshold, one can infer sensitivity at the periphery based on whether or not sound was able to pass through the ear and cause the sources of the neural response to respond in a synchronous manner. For individuals who do display synchronous neural responses, we can find the lowest stimulus intensity level that yields the neural response and relate that to a threshold for hearing. A limitation, in relation to patients with auditory neuropathy/dys-synchrony, will be discussed later.

ABRs can be obtained at intensities very close to behavioral thresholds if a sufficient number of responses are averaged to adequately reduce the background physiological noise (Elberling and Don, 1987). This requires a greater number of stimuli, on the order of 10,000 sweeps per test level and smaller intensity step sizes, and thus a longer time than is clinically feasible. In routine clinical procedures where fewer responses are averaged, responses can generally be obtained near, but not at, behavioral thresholds in a quiet subject.

WHEN TO USE THE ABR TO ESTIMATE AUDITORY FUNCTION

AEPs are best utilized when the clinician desires a noninvasive, objective approach to assessment of auditory function in infants, children, and adults who cannot participate in voluntary behavioral audiometric procedures. AEPs are especially useful when one wishes to know the sensitivity of each ear separately, to compare responses by air and bone conduction with or without masking, and to estimate auditory function in various frequency regions. Since AEPs are a test of the neural system, insight into the integrity of the neural pathways should also be considered. AEPs should not be used in lieu of a behavioral audiogram in patients who can provide reliable behavioral responses. An ultimate goal in all patients, where possible, should be to obtain behavioral responses and use this information in combination with physiological responses. In infants and young children, behavioral responses may be obtained at a later time, but, in the meantime, appropriate management can and should begin based on AEP results. Comparisons of evoked potential and behavioral responses provide a valuable clinical cross-check and confirmation of results on each measure individually.

WHY ARE FREQUENCY-SPECIFIC STIMULI NECESSARY?

Although broadband stimuli, such as clicks, are useful for evaluation of patients with suspected neurologic disease and for establishing neural synchrony, clicks are not considered appropriate for threshold testing. Because clicks are broad band in nature and also stimulate more basal regions of the cochlea, it is not possible to know the exact frequency range being tested. This is particularly problematic in patients with sloping (either positively or negatively) hearing losses. Furthermore, behavioral puretone audiometry is completed at multiple frequencies, not just one frequency. Figure 14.1 shows two hearing losses where clicks or other broadband stimuli will underestimate or miss a hearing loss. In Figure 14.1A, clicks will **underestimate** or **miss** this high-frequency hearing loss. The latency may be longer since lower frequency regions are stimulated. In Figure 14.1B, a click will **underestimate** or more likely **miss** this low-frequency hearing loss. Responses to clicks will be dominated by cochlear basal responses and will not reflect a hearing loss in more apical regions of the cochlea.

Types of Frequency-Specific Stimuli

A 100-microsecond electrical pulse, impressed on an earphone, generates a broadband signal whose primary

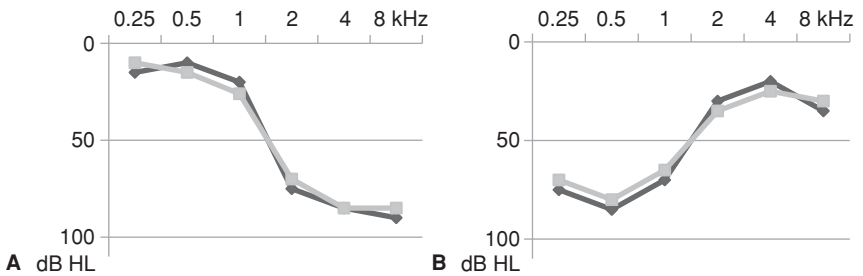


FIGURE 14.1 Example audiograms depicting puretone thresholds for right (*dark gray*) and left (*light gray*) ears that are consistent with a downward sloping hearing loss in (**A**) and an upward sloping hearing loss in (**B**).

frequency emphasis is determined by the resonant frequency of the transducer. With earphones typically used in clinical evaluation, the maximum energy peaks of these clicks are focused in the frequency region between 1,000 and 4,000 Hz (e.g., Don et al., 1979). The greatest agreement with puretone thresholds is in the 2,000- to 4,000-Hz frequency range (Bauch and Olsen, 1986).

Several types of stimuli and recording methods have been proposed to obtain responses from narrower frequency regions. Some alternative stimuli and methods include tone bursts or tone pips, filtered clicks, tone bursts in notched noise, and high-pass masking of clicks or tone bursts. Each type of stimulus appears to have advantages and limitations, and stimulus selection is dependent on frequency specificity, the amount of time available for testing, and the equipment available.

Techniques of masking test stimuli, in an effort to obtain greater control and precision related to the frequency content of the stimulus, have been the focus of several investigations. In a method pioneered by Teas et al. (1962), the cutoff frequency of a high-pass masker is progressively decreased and click ABRs at adjacent cutoff frequencies are subtracted from the filtered ABR and/or from the preceding ABR. This method allows separation of frequency-specific wave components and has been shown to be useful in audiogram reconstruction (Don et al., 1979). A modification of this method uses tone bursts presented in the presence of a high-pass masker (Kileny, 1981). By presenting stimuli simultaneous with a masker, the higher frequency response regions of the cochlea are blocked and the resulting responses reflect activity in frequency areas outside of the masker region.

Presentation of stimuli in notched noise narrows the stimulation to limited regions of the basilar membrane through presentation of a noise masker with components above and below the frequency range of interest (e.g., Picton et al., 1979). The presence of the notched noise masker restricts the cochlear region able to contribute to the ABR to those frequencies within the band of the notch. Although the utility of this method has been clearly demonstrated, additional special equipment or software may be needed to create and present the noise. Further, studies have demonstrated similar results with and without notched noise masking for most cases of hearing loss, with exception being very steeply sloping losses (e.g., Johnson and Brown, 2005).

Presently, tone bursts or tone pips, without high-pass or notched noise masking, are the most widely accepted and preferred stimulus for frequency-specific ABR evaluation. This chapter focuses on the use of tone bursts centered at various frequencies in recording frequency-specific ABRs.

Frequency Specificity versus Neural Synchrony

When using frequency-specific stimuli there is a trade-off between frequency specificity and neural synchrony. Tone bursts with longer rise times will be more frequency specific, but will generate poorer neural synchrony which will affect the quality of the ABR. As already emphasized, the goal of using ABR for threshold prediction is to stimulate isolated regions of the basilar membrane to analyze function in distinct frequency regions. Thus, control of spread of acoustic energy to surrounding frequencies works in opposition to the ability to activate a large number of neural units and obtain a clearly synchronous ABR. The more abrupt the acoustic onset of the stimulus, the more synchronous the neural discharge and the clearer the resulting ABR. However, as noted previously, abrupt onset, broadband stimuli have poor frequency specificity.

Although the ideal stimulus for frequency-specific ABR would be a puretone, this is not possible because stimuli with long rise times (needed to maintain integrity of a puretone) will not yield sufficient neural synchrony to obtain an ABR at the surface of the head. Thus, the stimulus used in ABR testing has a shorter onset (rise time) than a puretone, but longer than a click (which is essentially instantaneous). This results in some spectral spread of the tone burst stimulus, compared to a puretone. However, the spectrum of the tone burst is considerably narrower than a click and thus stimulation along the basilar membrane is restricted and reasonably frequency specific. In interpreting results, it is important to remember that the use of tone bursts results in stimulation of the cochlea at frequency regions surrounding the target frequency as well as at the desired frequency.

Frequency Specificity versus Place Specificity

There is a difference between frequency specificity and place specificity. Frequency specificity refers to the characteristics of the stimulus whereas place specificity reflects a region of the cochlea. Although the frequency regions activated may be relatively narrow at low intensities, there is considerable spectral spread for moderate-to-high–intensity signals. When presented at higher intensities, on the order of 60 to 80 dB HL or higher, even puretones can activate wider frequency ranges on the basilar membrane surrounding the center frequency of the stimulus (Moore, 2004).

Spread of excitation can be particularly problematic in underestimating hearing loss in individuals with steeply sloping hearing losses. A tone burst presented to an ear with a steeply sloping high-frequency hearing loss may yield a response, but that response may be generated from stimulation of a lower frequency responsive region where thresholds are better. Although latencies may be longer because of more apical stimulation, it can remain difficult to determine the source of the response and there is risk of underestimating a hearing loss. Thus, caution must be exercised in interpreting ABRs, particularly as in the case of infants where behavioral thresholds are unknown.

STIMULUS CONSIDERATIONS IN ABR THRESHOLD TESTING

Characteristics of the ABR will change as a result of a number of stimulus, recording, and patient factors. The effects of these factors must be considered when designing test protocols for various clinical populations, determining appropriate stimulus parameters, setting recording parameters, and interpreting ABRs. Stimulus, recording, and subject factors will be discussed in regard to applications in ABR threshold prediction in the following sections. We begin with stimulus factors.

Changes in the settings used in creating test stimuli can affect the latency and the amplitude of the ABR. An understanding of the effects of parametric changes in the stimulus is necessary to correctly interpret test results. And, importantly, understanding the results of adjusting various stimulus settings can be used to the examiner's advantage in obtaining the best possible responses.

Tone Burst Envelope Characteristics and Gating Functions

Whereas puretones have long rise times, in the range of 20 to 200 ms, tone bursts must have shorter rise times to achieve needed synchronous neural responses. As noted earlier, shortening the onset time of a stimulus results in broadened spectral characteristics. The optimal stimulus will maintain as much frequency specificity as possible while allowing activation of a sufficient number of neurons to record a far-field response. Characteristics needed to achieve this goal have been derived from studies that compare various stimulus rise times and durations across frequency and intensity.

Davis et al. (1985) recommended a tone burst with two cycles of rise time, a one-cycle plateau, and two cycles of decay, known as a "2-1-2 envelope." Changes in envelope characteristics affect the spectrum of the stimulus, the intensity (and the loudness because of durational changes and temporal integration), and the latency of the response because a longer rise time results in increased latency. By holding the number of periods in the stimulus constant across different frequencies, the power spectrum is held constant.

To create appropriate stimuli, one needs to recall the duration of a single cycle for various frequencies. For example, one cycle of a 500-Hz tone is 2 ms in duration. Therefore, to create a 2-1-2 envelope, the rise time would be 4 ms (two cycles at 2 ms per cycle), the plateau would be 2 ms (one cycle), and the fall time would be 4 ms (two cycles). This would add up to a total envelope duration of 10 ms. As another example, a 1,000-Hz tone burst would have a total envelope duration of 5 ms (2 ms rise, 1 ms plateau, and 2 ms decay). These stimulus parameters have generally held the test of time with some minor modifications. For example, since the ABR is an onset sensitive response, the plateau contributes little to the utility of the stimulus. Thus, envelopes now used often have no plateau and are referred to as 2-0-2 cycle envelopes.

The envelope of a stimulus is constructed using various types of windowing or gating functions, in other words the way in which stimuli are turned on and off. A linear envelope involves an abrupt change from no signal to the rise (or ramp) of the signal. A nonlinear windowing function, such as a Blackman window, has a curvilinear onset. Although differences in spectra have been observed with linear versus nonlinear functions, studies have shown similar ABR results using either linear or Blackman windowing functions (e.g., Johnson and Brown, 2005; Oates and Stapells, 1997). Thus, although some prefer to use a nonlinear, such as Blackman, function, either can be used. Most current clinical ABR equipment uses the Blackman or nonlinear functions as the default setting.

A caution is needed related to equipment settings for stimulus durations and windowing functions. The way in which these are set varies across ABR systems. Some systems will differentially ask for duration settings in either cycles or milliseconds depending on the type of envelope requested. Further, some systems require information about the total duration of the stimulus whereas others set stimuli according to rise and fall times separately. The key here is to understand the desired and appropriate characteristics and know how to calculate those according to the specific ABR system requirements. Knowing this will avoid errors that could result in stimuli with poor frequency specificity (rise time or envelope too short) or poor neural responses (rise time or envelope too long).

Stimulus Frequency

Higher frequency stimuli elicit shorter ABR latencies than lower frequency stimuli. These latency differences occur because high-frequency stimuli activate more basal portions of the basilar membrane, resulting in earlier neural activation and shorter latencies compared to stimuli centered at lower frequencies. Since lower frequency stimuli have to travel further toward the apex of the cochlea, latencies for these stimuli will be longer. Because all components of the ABR are dependent on cochlear processing, all waves of the ABR (e.g., Waves I, III, V) will display shorter latencies for higher frequency tone bursts and longer latencies for lower frequency tone bursts. (For reference, a sample of a normal ABR, obtained with click stimuli, is shown in Figure 11.1 in Chapter 11.) ABRs obtained to tone bursts centered at 500, 1,000, 2,000, and 4,000 Hz are shown in Figure 14.2. Here it can be seen that latencies for Wave V at comparable intensities are longer for low-frequency tone bursts than for high-frequency tone bursts.

Stimulus Intensity

As stimulus intensity decreases from 70 or 80 dB nHL to the threshold of detectability, all waves of the ABR show a

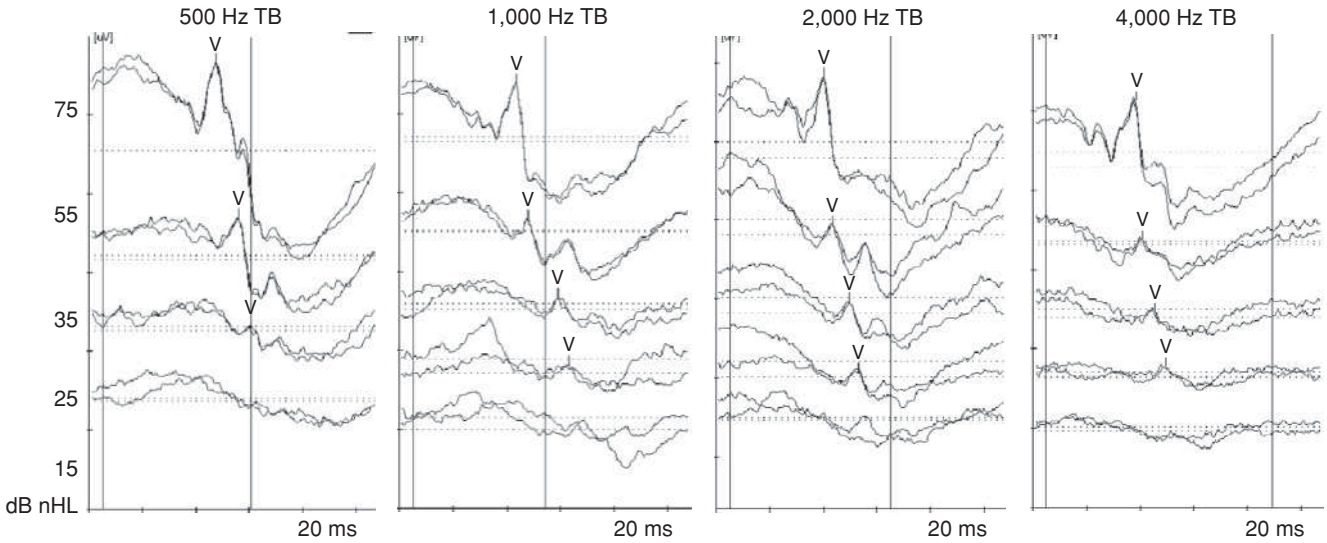


FIGURE 14.2 ABRs to tone bursts centered at 500, 1,000, 2,000, and 4,000 Hz recorded from an individual with normal hearing. The latencies of Wave V at 75 dB nHL are 8.53 ms for a 500-Hz tone burst, 7.70 ms for a 1,000-Hz tone burst, 7.03 ms for a 2,000-Hz tone burst, and 6.53 ms for a 4,000-Hz tone burst, demonstrating the latency decrease as center frequency of the tone burst increases.

systematic increase in latency and decrease in amplitude (Picton et al., 1974; Starr and Achor, 1975). This is illustrated in Figure 14.2. Wave V is most visible at lower intensity levels whereas the earlier components tend to become indistinguishable at lower intensities. The intensity at which earlier waves become less apparent also depends on tone burst frequency. Changes in Wave V latency with intensity are nonlinear with shifts on the order of approximately 0.2 to 0.3 ms per 10 dB through mid-intensity ranges and more rapid changes in latency at lower intensities and near response thresholds.

The amplitude of the ABR is rarely greater than 1 microvolt and no consistent trend in amplitude growth as a function of intensity has been reported (Hecox and Galambos, 1974; Jewett and Williston, 1971). This is most likely related to the considerable variation in amplitude within and among subjects, as amplitude is more highly influenced by noise than latency. The amplitude of Wave V is less affected by intensity decreases than earlier components (Pratt and Sohmer, 1976; Terkildsen et al., 1975).

It is important to note that the actual intensity and frequency information reaching the cochlea is dependent on the acoustic properties of the transducer, the volume of the external ear canal, and middle-ear transmission characteristics. This can be particularly problematic in infants and young children whose ear canals are small. It is possible that technologic advances will facilitate use of a transducer containing a probe microphone to monitor the sound pressure level in the ear canal and a method to account for intensity differences as a function of ear canal volume. This should be especially useful in neonatal screening and testing of infants and young children.

Stimulus Rate

The rate at which test stimuli are presented affects both the latency and the amplitude of the various components of the ABR. In general, at stimulus rates above approximately 30 stimuli per second, the latency of all components of the ABR increases and the amplitude of the earlier components decreases (e.g., Don et al., 1977; Terkildsen et al., 1975). Latency does not increase by the same amount for all components. With increasing stimulus rate, the later components (e.g., Wave V) show a greater latency increase than the earlier components, which results in a prolongation of the Waves I–V interwave interval. Wave V also shows less of an amplitude decrease at high rates, which can facilitate the use of higher stimulus rates in evaluation.

Stimulus rate is an important consideration in testing infants and young children. Faster stimulus rates may help in decreasing test time since more stimuli can be presented in a shorter period of time. Faster stimulus rates may be useful, with some caveats discussed later, in threshold-seeking procedures where only the presence or absence of Wave V is of interest. However, faster stimulus rates may also reduce the clarity and reproducibility of responses, particularly for the earlier components. As noted later, this can be problematic if response amplitude is reduced and thus the signal-to-noise ratio is compromised. Here, results could suggest the presence of a hearing loss when, in fact, hearing thresholds are normal.

Stimulus Polarity

Stimulus polarities for ABR testing can be selected as rarefaction, condensation, or alternating between rarefaction

and condensation stimuli. Because latencies of the various components in the resulting response are dependent on the polarity of the test stimuli, both consistent use of a particular polarity when comparing results to normative data or previous tests and knowledge of the effects of polarity are critical.

A rarefaction stimulus produces an initial outward movement of the earphone diaphragm that generally leads to an outward movement of the footplate of the stapes and an upward motion of the more basal structures of the organ of Corti. Because the upward motion of the basilar membrane is the depolarizing motion for the hair cells, latency is slightly shorter and amplitude is higher for the early components of the ABR for rarefaction pulses in comparison to condensation pulses in the majority of subjects (e.g., Stockard et al., 1979). Condensation stimuli produce an initial inward movement, followed by outward movement and depolarization of the hair cells. Thus the early components of the ABR may be slightly longer in latency than those produced using rarefaction pulses. Wave V amplitude tends to be larger in response to condensation stimuli for normal-hearing subjects. There is no significant latency difference in Wave V latency to rarefaction or condensation stimuli (e.g., Stockard et al., 1979).

At high intensities and for bone-conduction testing, use of alternating polarity reduces stimulus artifact. Alternating polarity stimuli for air-conduction testing, particularly in the lower frequencies, can be a problem as responses in some subjects to condensation versus rarefaction stimuli can be out of phase, as described below. Use of insert earphones, with an inherent delay of 0.9 ms that separates the stimulus generation from the time it reaches the ear, results in reduced interference of stimulus artifact with the response. Therefore, the need for alternating polarity stimuli may be less of an issue for air-conduction testing. Alternating polarity stimuli are recommended when using a bone-conduction transducer where large electrical artifacts from the bone oscillator are problematic.

Polarity Considerations

When the polarity of a stimulus is reversed, latency shifts in the peak of the response may be observed. Typically, higher frequency tone bursts (e.g., 2,000 or 3,000 Hz and above) tend to show little or no latency shift with polarity reversals. However, tone bursts centered at lower frequencies, such as 250 or 500 Hz, can show large latency shifts in some individuals that can degrade the waveform and even be out of phase with the opposite polarity (Gorga et al., 1991; Orlando and Folsom, 1995). Thus, whereas it may seem intuitively desirable to alternate polarity to minimize stimulus artifact, in fact the use of alternating tone bursts for lower frequencies can be detrimental in some cases.

Consistent with the observation of greater latency differences between condensation and rarefaction polarity stimuli for lower frequencies, studies of patients with high-frequency hearing loss show considerable latency changes within individuals as a function of polarity. Simulation of high-frequency hearing loss through high-pass masking indicates that the polarity effects are primarily because of lower frequency contributions to the response that would be particularly apparent in person with high-frequency hearing loss (Schoonhoven, 1992). Large latency differences between polarities are observed in individual subjects whereas there do not seem to be systematic trends when comparisons are made on a group basis (Schoonhoven, 1992; Sininger and Masuda, 1990). Because phase reversals can degrade an ABR sufficiently to interfere with accurate interpretation and reversals appear to have a detrimental effect in some individuals, use of single polarity stimuli is recommended in patients with high-frequency hearing loss and patients, such as infants and young children, where hearing threshold configuration is not known.

There also is a group of patients in which ABRs appear to be present when in fact the acquired waves represent the cochlear microphonic (CM; e.g., Berlin et al., 1998). In these patients, described as having auditory neuropathy/dys-synchrony, the peak latency does *not* increase as the intensity of the stimulus is decreased; the first indication that this is not a neural response. When the polarity of the stimulus is reversed, the waves also invert, consistent with the characteristics of the CM. Present CMs are seen in infants with no ABR but present otoacoustic emissions (OAEs). CM is also seen in infants with present ABRs which is a normal observation, providing evidence of intact cochlear and neural function. Comparing separate averages of both rarefaction and condensation stimuli will aid in identification of patients with auditory neuropathy/dys-synchrony and, as discussed later, this procedure is now a part of our standard ABR protocol.

Chirps

As noted earlier, a click stimulus is theoretically broad in bandwidth because of its rapid onset. Although clicks stimulate broad regions of the cochlear partition, in cases of normal hearing or a flat hearing loss, the resulting ABRs are generally attributed to responses from more basal, or higher frequency, regions of the cochlea. The tonotopic design of the cochlea results in temporal delays because basal portions are activated earlier in time than more apical regions. These cochlear delays can result in phase cancellations based on the accumulation of responses from individual neural units that contribute to the total ABR (Don and Eggermont, 1978). Such phase cancellations can have a detrimental effect on the amplitude of the averaged response of the ABR.

Recently, stimuli that are called "chirps" have been applied to ABR testing (e.g., Fobel and Dau, 2004) and, more recently, frequency band limited chirps have drawn considerable interest as a stimulus for frequency-specific ABR testing (Elberling and Don, 2010). Use of chirp stimuli

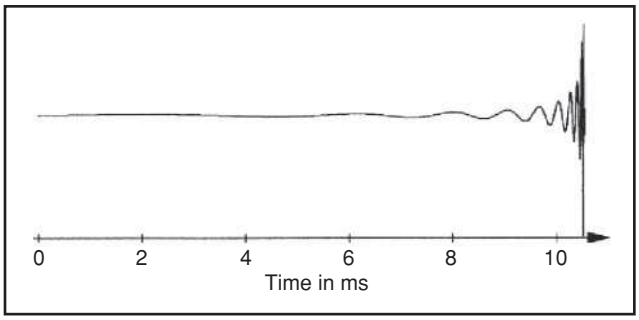


FIGURE 14.3 Example of a broadband chirp, plotted with time on the *x*-axis and amplitude on the *y*-axis. Note that lower frequencies (*toward the left*) precede higher frequencies (*toward the right*) in time. In this way, theoretically, more apical portions of the cochlea are stimulated earlier than more basal regions, resulting in increased synchronous neural firing.

results in ABRs that theoretically allow simultaneous contribution of neural activity from all cochlear frequency regions. In creating a chirp stimulus, higher frequency components contributing to the stimulus are delayed in time relative to the lower frequency components (Dau et al., 2000). Through this stimulus generation, chirps are designed to offset cochlear delays and increase synchronous neural firing, resulting in increased response amplitude (e.g., Fobel and Dau, 2004). An example of a chirp stimulus is shown in Figure 14.3.

Several studies have demonstrated higher ABR amplitude with broadband chirps in comparison to clicks (e.g., Elberling and Don, 2008; Fobel and Dau, 2004; Maloff and Hood, 2014). With the need for frequency-specific stimuli in using ABR to estimate hearing sensitivity in pediatric populations, frequency-specific chirp stimuli have been created. A type of frequency-specific chirps, known as octave-band chirps, may provide a more sensitive frequency-specific metric through generation of higher amplitude responses and thus lower response thresholds (Elberling and Don, 2010). Reports are beginning to appear that systematically compare broadband and frequency-specific stimuli in patients of various ages.

Stangl et al. (2013) compared physiological response amplitudes and thresholds for stimuli currently used in clinical settings (click, tone burst) to broadband and frequency-specific chirp stimuli (CE-Chirp, octave-band chirp). ABR Wave V amplitudes were significantly greater for broadband chirp than click stimuli at 60, 40, and 20 dB nHL, consistent with previous studies (Kristensen and Elberling, 2012; Maloff and Hood, 2014). For frequency-specific stimuli, ABR Wave V amplitudes were generally greater for octave-band chirps than for tone burst stimuli, though the amplitude differences varied with stimulus frequency and level. An example of an intensity series for a 2,000-Hz octave-band chirp is shown in Figure 14.4. Greater differences occurred at higher intensities and no significant differences were found for 500-Hz stimuli. Higher amplitudes for frequency-specific stimuli have also been reported by Ferm et al. (2013) and Wegner and Dau (2002).

Higher amplitude responses can result in improved signal-to-noise ratios. With similar noise levels and higher amplitude responses for chirp stimuli, lower ABR thresholds and better agreement with behavioral thresholds would be predicted. With the desire to utilize the most efficient and sensitive paradigms in pediatric applications where ABR is used to obtain estimates of hearing sensitivity, frequency-specific chirp stimuli may offer advantages for accurately determining hearing sensitivity and hearing loss configuration.

RECORDING CONSIDERATIONS IN ABR THRESHOLD TESTING

Infants differ from adults in many ways and this includes characteristics affecting recording of ABRs. For example, differences in head size and shape should be considered in placing electrodes to obtain optimal responses. Infants have longer ABR wave latencies than adults which translate to lower frequency content of infant ABRs. Both longer latencies and lower frequency characteristics require changes in the recording window and the filter settings, respectively, to be sure to encompass the response of interest.

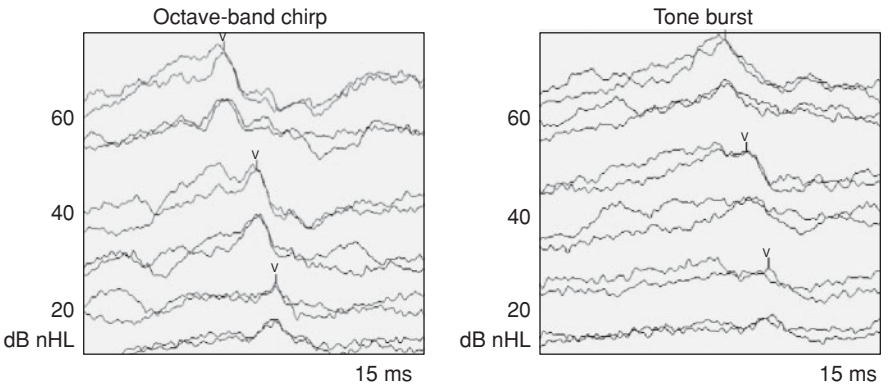


FIGURE 14.4 Examples of ABRs to octave-band chirps in the **left panel** and tone bursts in the **right panel** centered at 2,000 Hz recorded from an individual with normal hearing.

Electrode Montages

Placement of electrodes at the vertex (C_z) and ears (A_1 and A_2) and recording between vertex and ear (C_z–A_1 for the left side and C_z–A_2 for the right side) are optimum for recording the ABR in most conditions. Waves I–III are more prominent in ipsilateral recordings whereas Waves IV and V are better separated in contralateral recordings. Earlobe sites tend to result in less muscle potential than mastoid recording sites and greater Wave I amplitude. Use of a noncephalic site (recording from the vertex to the nape of the neck, C_7) can enhance the amplitude of Wave V.

In infants, the recommended electrode montages differ from adults. A contralateral montage (vertex or high forehead to the ear contralateral to the stimulus ear) is not recommended in infants as the response is poor in newborns in this channel. In a comparison of two electrode montages in infants, thresholds were lower when recorded from the vertex to the nape of the neck (Sininger et al., 2000). Although amplitude was higher at this location, noise levels were also higher than for a vertex to mastoid montage. This may be attributed to proximity of the nape electrode to the torso and additional physiological noise sources. Both electrode montages appear to work well in threshold estimation application.

Filter Settings

When speaking of filtering and filter characteristics related to the recording of the ABR, one is describing the filter band through which the physiological response is recorded from the electrodes. This is distinguished from any filtering of a stimulus transduced through an earphone or bone oscillator. Filtering of the physiological response is used to eliminate as much internal noise (e.g., unrelated muscle potentials, general physiological activity) and external electrical noise (e.g., 60 Hz, other equipment in the environment) as possible. The filters are set to pass the signal of interest, in this case the ABR.

Changes in the frequency band through which the physiological response is filtered affect waveform latency and amplitude. Changing the filter so that there is more high-frequency information in the biologic signal generally decreases the latency of the response. Allowing more low-frequency information into the average typically results in more rounded peaks and longer latencies. Interference from electrical sources and muscle activity increases as more low frequencies are included. Wave V amplitude may increase because it is dependent on the amount of low-frequency energy included because of the low-frequency components of Wave V. Use of very narrow filters and steep filter slopes is discouraged because phase shifting may occur in frequency regions near the cutoff frequencies.

As noted earlier, infant ABRs have longer latencies and contain more lower frequency energy. Therefore, when testing infants, lowering the low-frequency filter setting from 100 to 30 Hz will often enhance the amplitude of infant ABRs (Sininger, 1995; Spivak, 1993).

Time Window

The recording time window, or analysis time, should be set to encompass all components of the response. The duration of the time window will vary with the age of the patient and the intensity and type of stimulus used. For presentation of click stimuli in adult patients, a time window of 10 to 12 ms is usually sufficient to record the ABR since Wave V occurs in normal individuals within 5 to 6 ms of the stimulus at high intensities and within 8 ms for intensities near threshold. Use of insert earphones will delay the response by slightly less than 1 ms which is still within this time frame. In infants, however, ABR waves are longer, on the order of 1 ms or more; therefore, a time window of at least 15 ms is recommended when testing patients below 18 months of age or in patients older than 18 months where delays in neuromaturation are suspected.

Whereas a 10- to 15-ms time window is usually sufficient when presenting click stimuli, ABR testing with tone bursts requires a time window of at least 20 ms. This is particularly true when using 250- or 500-Hz tone bursts where the stimuli have a longer onset time and activate more apical portions of the cochlear partition. In practice, when testing low frequencies at low intensities in an immature system, a window of 25 ms may be desired.

One- versus Two-Channel Recordings

Recording two channels simultaneously allows acquisition of both ipsilateral and contralateral (or midline) recordings simultaneously. In infants, ipsilateral and midline channel recordings are recommended. For diagnostic testing and threshold prediction there are several reasons to obtain two-channel recordings that include:

1. To monitor the ear that was stimulated by the amplitude of the artifact in the response and the position of the trace containing Wave I.
2. To determine the presence and location of the CM and Wave I by comparison to a contralateral recording. The CM and Wave I should not be present, or be diminished, in the contralateral tracing.
3. To obtain better definition of Waves IV and V since they tend to be more separated in contralateral recordings.

Number of Averages and Noise Quantification

The number of averages required varies according to the inherent amplitude of the evoked potential and the amount

of background noise that includes muscle artifact, 60-Hz noise, and EEG activity. For the ABR, usually 1,000 to 2,000 sweeps are used to obtain clear responses in quiet patients when using higher intensity stimuli. At lower intensities where the response is lower in amplitude and in cases where patients are more active and noisier, more averages may be necessary because of the reduced signal-to-noise ratio.

Use of objective estimates of the signal-to-noise ratio, such as F_{sp}, based on the *F*-ratio statistic, is very helpful in determining when a sufficient number of responses have been averaged (Don et al., 1984). Using methods such as F_{sp} can allow, in an objective manner, for averaging of fewer responses at higher stimulus intensity levels where the response has high relative amplitude whereas more responses can be averaged to improve accuracy for low intensity stimuli (i.e., close to response threshold) where the ABR amplitude is low. Point-optimized variance ratio (POVR) is another statistically based signal and noise estimation method, which is implemented in a newborn hearing screening system (Sininger, 1993). In addition to requiring fewer averages, these methods reduce the need to replicate the response and provide objective estimate of response presence.

SUBJECT CONSIDERATIONS IN ABR THRESHOLD TESTING

Age

The ABR changes as a function of age, particularly during the first 12 to 18 months of life, as the auditory neural system continues to mature. These changes have been attributed to continuing myelination of the auditory pathway after birth. Characteristics of ABRs obtained in premature and term infants vary from each other and from those obtained in adults (e.g., Hecox and Galambos, 1974; Salamy, 1984). Reliable ABR components for 65-dB nHL clicks have been reported in newborns of approximately 28 weeks gestational age (Starr et al., 1977). Waves I, III, and V are most visible in infant recordings and the normal Wave V absolute latency for click stimuli in a newborn approximates 7.0 ms at 60 dB nHL. Responses obtained from infants 12 to 18 months and older should resemble those acquired from adults (Hecox and Galambos, 1974).

Wave I may be prolonged in infants, but generally not as much as Wave V, generating longer interwave latencies on the order of 5.0 ms compared to 4.0 ms in adults (Hecox and Galambos, 1974; Starr et al., 1977). This may be related to cochlear maturation, neuronal maturation, reduced efficiency in external and/or middle-ear sound transmission, and occasionally collapsing ear canals. Neural maturation of the auditory system is complex with conduction time adult-like by term birth, pathway lengthening continuing to mature until about age 3 years, and different aspects of myelin development contributing to changes in ABRs in infants (Moore et al., 1996).

Amplitude of the ABR also changes with age. Peak amplitudes (typically measured from the peak to the following negative trough) increase over the first 1 to 2 years of life (e.g., Salamy, 1984). In infants, the amplitude of Waves I and/or III may be greater than Wave V, which is in contrast to higher Wave V amplitude in adults. This changes to more adult-like patterns over the first few years of life.

Gender

Females tend to have shorter latency and higher amplitude ABRs than males. Wave V latency averages about 0.2 ms shorter in females, and amplitude is higher in females, particularly for Waves IV, V, VI, and VII. Females may also show shorter interwave latencies than males. It has been suggested that the source of the differences in latency and amplitude in the ABR between males and females may be related to the observation that cochlear response times are shorter in females than males (Don et al., 1994).

PROTOCOLS AND PROCEDURES FOR PEDIATRIC PATIENTS

A pediatric test protocol includes multiple measures to provide a cross-check among results and to maximize efficiency (e.g., Gravel and Hood, 1999). The combination of middle-ear measures that includes immittance and middle-ear muscle reflexes, OAEs, and ABR provides a comprehensive view of middle-ear, cochlear, and peripheral neural function (Berlin and Hood, 2009). Once it is possible to obtain reliable behavioral information, at around 6 months of age in typically developing children, a combination of physiological and behavioral test results provides important test cross-checks.

Our pediatric protocol utilizes a combination of measures, as described in Table 14.1. We measure either transient (TEOAE) or distortion product (DPOAE) otoacoustic emissions, tympanograms using a 1,000-Hz carrier tone, and a minimum of ipsilateral middle-ear muscle reflexes at 1,000 and 2,000 Hz. We recommend completing these tests prior to the ABR, as pressure may change in the middle ear over the course of a deep sleep or sedation period and this can affect accurate assessment and interpretation of OAEs and middle-ear tests. All air-conduction testing is completed in each ear individually.

Approaches

For ABR testing, one can complete all ABR testing with tone bursts or can use tone bursts in combination with a brief neural integrity screening that uses a click stimulus. It must be emphasized that click stimuli in pediatric testing are *not* used for threshold prediction; clicks are only used to establish the presence of neural synchrony. Thus clicks are only briefly included and are presented at a moderate to high

TABLE 14.1

Suggested Tone Burst ABR Test Parameters

Parameter	Comments
Stimulus	
Type	Tone burst
Polarity	Condensation for AC, alternating for BC
Intensity	Begin at 75 dB nHL, decrease in 20-dB steps, refine to 10-dB steps
Rate	27.7/s; 39.1/s for 500 Hz
Transducer	Earphone, bone oscillator
Recording	
Time window	20–30 ms
Filter band	High pass 30 or 100 Hz; Low pass 1,500 or 3,000 Hz
Number of sweeps	1,500–2,000 at high intensities, more sweeps nearer threshold
Electrode montage	Noninverting at vertex or high forehead; inverting at mastoid or earlobe; second channel recommended with inverting electrode at nape
Subject	
State	Sleeping, resting quietly, sedated for older infants

intensity where responses to rarefaction and condensation polarity stimuli are compared. If responses are present to clicks, then testing proceeds immediately to determine the response thresholds for tone bursts.

When the ABR protocol utilizes tone bursts for all aspects of testing, then testing begins with tone bursts. Click stimuli may be used later in the test sequence if there is any question about the integrity of the tone burst responses that might suggest a neural synchrony problem and possible auditory neuropathy/dys-synchrony.

Neural Integrity

To test neural integrity, we present clicks at a single high intensity (e.g., 75 dB nHL or higher if there is no response at 75 dB nHL) using both condensation and rarefaction polarity, presented or collected separately, so that results can be compared to differentiate the CM from neural response components. The CM (which will reverse in phase as the stimulus does) is distinguished from Wave I of the ABR (which will not show a phase reversal with clicks). It is emphasized that testing with click stimuli is completed *only* at a single high intensity as a method of checking neural response integrity. When it is documented that an ABR is present, then testing proceeds immediately to tone bursts to obtain estimations of frequency-specific thresholds. Figure 14.5 shows examples of an infant with good neural synchrony and an infant with poor neural synchrony. The infant with poor neural synchrony was found to have auditory neuropathy/dys-synchrony.

ABRs to Air-Conducted Tone Burst Stimuli

A key in ABR testing for threshold estimation involves using well-defined frequency-specific stimuli. Typical stimulus durations utilize a minimum of two cycles rise and fall times (Davis et al., 1985; Gorga et al., 1988), which provide both sufficient frequency specificity and neural synchrony. We prefer condensation polarity tone bursts as these provide higher Wave V amplitude in the majority of individuals. We do not recommend using alternating polarity signals (unless each polarity is separately averaged), particularly in the lower frequencies (Orlando and Folsom, 1995). Suggested tone burst ABR parameters are shown in Table 14.2.

Four frequencies are tested in each ear: 500, 1,000, 2,000, and 4,000 Hz. The test order is 2,000, 500, 1,000, and 4,000 Hz *if* OAEs are present at all frequencies. This order was determined based on obtaining key information for management of and monitoring hearing loss along with the

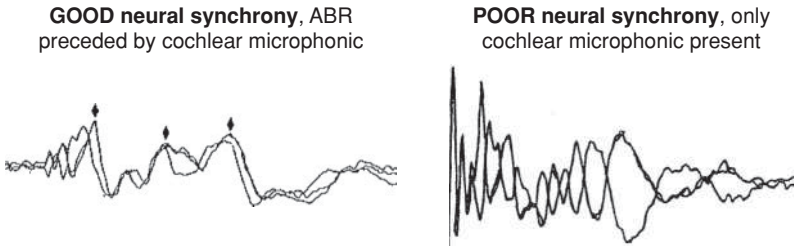


FIGURE 14.5 A patient demonstrating good neural synchrony is shown in the **left panel** where the recording contains a CM at the beginning of the response and Waves I, III, and V of the ABR, with good replication. The patient depicted in the **right panel** has poor neural synchrony and the tracing only shows the reversing CM with no ABR present. Two tracings with condensation clicks and one tracing with rarefaction clicks are shown in each panel.

TABLE 14.2

Suggested Test Protocol for ABR Threshold Testing

Procedure	Comments
Otoacoustic emissions	
TEOAE or DPOAE	Assess aspects of cochlear function, compare to ABR to identify AN/AD For DPOAE, test at 2, 3, 4 kHz with pass criteria of responses >0 dB SPL and at least 6 dB SNR for all three frequencies
Middle-ear measures	
Tympanogram	For infants <7 months, use probe tone >660 Hz
Middle-ear muscle reflex	Minimally screen at 1 and 2 kHz; must be present at normal levels for both frequencies to pass
Checking neural integrity	Condensation and rarefaction clicks at 75 dB nHL Conduct two runs with condensation polarity and at least one run with rarefaction polarity. If ABR obtained, proceed to tone burst testing If no response, increase intensity If no response persists, clamp earphone tube to distinguish stimulus artifact
Threshold prediction	
Tone burst stimuli	Test order: 2,000, 500, 1,000, 4,000 Hz unless OAEs absent at 4,000 Hz; then test 4,000 Hz before 1,000 Hz
Air conduction	Increase or decrease intensity in 20-dB steps, then refining to 10-dB steps
Bone conduction	Alternating polarity; care in placement of bone oscillator

additional consideration that testing at all frequencies may not be possible if a patient wakes up prior to test completion. If no OAE energy is present above 2,000 Hz, the order of the tone burst center frequencies is 2,000, 500, 4,000, and 1,000 Hz to acquire higher frequency information at 4,000 Hz that could be useful in monitoring possible high-frequency progressive hearing loss. The ideal approach is to assess each frequency in each ear and alternate between ears for each frequency (e.g., 2,000 Hz in the right then the left ear). This approach allows the opportunity to obtain at least some information from each ear in the event that the entire test sequence cannot be completed (i.e., infant becomes too active). However, if insertion of an earphone in the opposite ear would result in awakening a sleeping baby, then testing is completed in one ear before testing the opposite ear.

Testing typically begins at a reasonably high intensity, such as 75 dB nHL, or at a level where a response is likely to be present based on history, observation, and so on. Intensity is then increased or decreased by 20-dB steps with replications at each level until no response is obtained. If there is no response at 75 dB nHL, then the stimulus intensity is increased. Each of these responses is obtained twice to judge replicability and assist in determination of threshold. Once the threshold range has been bracketed, the step size is decreased to 10-dB steps. Intensity is lowered until Wave V disappears or a response is observed within the predicted normal range. If the patient is quiet and we predict that sufficient time will be available to complete all parts of the test battery, then the intensity level is increased 10 dB to determine the presence of a response at an intermediary level. For example, if responses are obtained at 75, 55, and 35 dB nHL, but no response is observed at 15 dB nHL, then stimuli are presented at 25 dB nHL. At higher intensities where response amplitudes are higher and thus more easily seen over the noise, fewer sweeps, such as 1,000 or 1,500, may be sufficient. Closer to threshold, averaging of more than 2,000 responses may be necessary.

Lower frequency tone bursts are typically more difficult to discern as they generally do not have the familiar five- to seven-wave complex associated with ABRs to clicks or higher frequency stimuli. For 500-Hz tone burst ABRs, we adjust the filters to 30 to 1,500 Hz to accommodate lower frequency physiological activity. Tone bursts with 4-ms (two cycles) rise and fall times, Blackman envelopes, and either condensation or rarefaction polarity are typically used. The time window is extended to 20 or 25 ms since these responses have longer latencies than responses to clicks. Tone bursts centered at 500 Hz are presented at a rate near 40 per second (we use 39.1 per second) to each ear individually beginning at 75 dB nHL and then decreasing in 20-dB steps until no response is obtained, with the 10-dB step filled in if time allows. If there is no response at 75 dB nHL, then the stimulus intensity is increased.

When interpreting the responses obtained to 500-Hz tone bursts, we look for a single replicable peak that represents Wave V of the ABR. We generally observe a single peak that may have a sinusoidal overlay at high intensities. The latencies obtained range from 8 to 10 ms for high-intensity stimuli to 14 to 16 ms nearer threshold (Gorga et al., 1988). In infants, these latencies may be even longer. In our experience, ABR thresholds to 500-Hz tone bursts obtained from normal-hearing individuals using the test parameters described here are generally between 25 and 35 dB nHL.

ABR to Bone-Conducted Stimuli

When responses to air-conducted auditory stimuli are seen at predicted normal threshold levels, there is no need

to obtain bone-conducted responses, as is true in behavioral audiometry. However, when tone burst responses are not present at predicted normal levels, then ABRs should be completed using bone-conducted stimuli. Criteria for obtaining bone-conduction thresholds are (1) if any of the air-conduction thresholds were not within the normal range; (2) if OAEs, immittance, or reflexes are abnormal; or (3) for subsequent visits, if there is a reason to believe there has been a sensory change.

The test parameters used are the same as those for air-conducted stimuli except that alternating polarity is used to reduce the electrical artifact emitted from the bone oscillator. Tone bursts are presented at progressively decreasing intensities via bone conduction to determine whether there is a discrepancy in intensity levels at which responses are obtained between air- and bone-conduction stimuli. If such a discrepancy does exist, this suggests the presence of an air–bone gap and a conductive or mixed hearing loss.

Care must be taken in the placement of the bone oscillator in infants to assure appropriate response amplitude and test accuracy. Oscillator placement in infants should be at the mastoid since stimuli from the bone oscillator are conducted across the scalp less efficiently in infants than in adults. Stuart et al. (1994) demonstrated that bone oscillator placement in infants has a significant effect on Wave V amplitude. Placement of the oscillator directly behind the ear canal is recommended as placements higher on the mastoid result in lower amplitude. Coupling of the bone oscillator also takes special consideration. The procedure described by Yang and Stuart (1990) provides an excellent method. They recommend coupling the bone oscillator using an elastic headband held in place by a Velcro closure that can be adjusted. The tension of the headband can be adjusted to a recommended coupling force of 400 to 450 g using a spring scale.

The dynamic range for bone-conducted stimuli is different than that for air-conducted stimuli, as is true in audiometric applications. The dynamic range typically does not exceed 50 to 60 dB and the relationship between the output of the oscillator and the "dial reading" on the equipment may vary with different instruments. Stimuli are presented beginning at the highest output level and then decreasing in 20-dB steps as in the other tests. Responses are first obtained without masking and then, if response thresholds are better than those obtained by air conduction and/or there is an asymmetry between ears, masking is used.

Because the output in bone conduction is limited, the responses obtained by bone conduction will rarely show the familiar five-wave complex seen at high intensities in standard air conduction ABRs. Responses will resemble those obtained with air-conducted clicks in the threshold-to-50 or 60 dB nHL range. The latency of waves may vary slightly from those acquired by air conduction based on slight spectral differences among bone-conduction transducers. However, since the primary goal in bone-conduction testing in infants is determination of ABR threshold, this is rarely an issue.

Relation of ABR Thresholds to Behavioral Thresholds

In the final step for predicting behavioral thresholds, the threshold of the ABR (i.e., the lowest level where a physiological response is obtained) is adjusted to relate the physiological (ABR) thresholds to behavioral puretone thresholds. This information is helpful in planning management, fitting amplification, counseling, and reporting results. The term "estimated hearing level" (eHL) is used (e.g., Bagatto et al., 2005). In the case studies presented later in this chapter data are used from the Ontario Infant Hearing Program (OIHP) in Canada where adjustments are 20 dB at 500 Hz, 15 dB at 1,000 Hz, 10 dB at 2,000 Hz, and 5 dB at 4,000 Hz.

The eHL values used in the OIHP as well as grossly similar corrections that are used in other programs were derived from studies that directly compared ABR thresholds and behavioral thresholds in the same subjects. An excellent study by Stapells et al. (1995) recorded ABR thresholds to air-conducted stimuli in infants and children with normal hearing and various degrees of hearing losses. Relationships between ABR and behavioral thresholds were approximately linear across a relatively wide range of intensities (20 to about 90 dB nHL). Across the subject dataset, ABR thresholds averaged (with rounding to the nearest 5 dB) 15, 5, and 0 dB above behavioral thresholds at 500, 2,000 and 4,000 Hz, respectively. In another study, Stapells (2000) reviewed studies meeting test criteria and containing data for ABR and behavioral thresholds. Although some differences existed among studies included in this meta-analysis, based on techniques and calibrations, data were available for infants, children, and adults with normal hearing and with SNHL. Results of this analysis indicated overall approximate differences of 15, 10, 5, and 0 dB for 500, 1,000, 2,000, and 4,000 Hz, respectively, though there was variation between children and adults and between those with normal hearing and with SNHL.

ADDITIONAL CONSIDERATIONS

Maximizing ABR Test Accuracy and Efficiency

There are modifications of ABR procedures that can be used to improve test efficiency and decrease test time. This is particularly important since, when sedation is used, there is a finite time period during which a patient will be asleep and, thus, a limited amount of time to obtain all necessary information.

Picton (1978) recommended the use of faster stimulus presentation rates to reduce test time. He suggested the use of a slow rate at a relatively high intensity for accurate identification of the latencies of Waves I, III, and V. Then, when in the threshold-seeking mode of testing, stimuli can be presented at rates on the order of 50 to 70 clicks per second, or faster. Although Wave V latency is prolonged, the amplitude of Wave V in normal adults is not reduced as much as

earlier components, making acquisition of responses near threshold possible. Slower rates at all levels are indicated if the response disappears when stimulus rate is increased.

Other methods use very high stimulus rates (on the order of 500 to 1,000 stimuli per second) with randomized presentation sequences, known as maximum length sequences (MLS; e.g., Picton et al., 1992) and continuous loop averaging deconvolution (CLAD; Delgado and Özdamar, 2004). Presentation of different sequences to each ear allows testing of both ears simultaneously which, when coupled with fast presentation rates, could decrease overall test time. Limitations in testing immature or compromised systems at very high rates and noise levels need to be considered. Caution should be exercised when testing in infants as responses to faster rates may result in decreased amplitude. In cases where no or poor responses are obtained at high rates, testing with slower rates should follow.

Test Protocol for Older Children and Adults

The test protocol used for older children, over approximately 8 to 10 years of age who are not sedated, and adults may vary from that used for infants and young children. Frequency-specific stimuli remain necessary to adequately complete threshold estimation; however, there are additional paradigms in which tone bursts can be presented. For example, 40-Hz ASSR techniques may be added to tone burst ABR and other ASSR methods (see Chapters 15 and 17). Although the 40-Hz response has not been found useful in young patients, it is quite useful in older patients for estimating auditory function using frequency-specific stimuli (Stapells et al., 1984).

Use of Masking

When using tone bursts or tone pips, as with other stimuli, masking should be used when sensitivity differences between ears could create crossover of sound to the nontest ear. With insert earphones the intensities where crossover occurs during air-conduction testing is higher than with supra-aural earphones. The bandwidth of the masking stimulus should be sufficiently wide to encompass the tone burst stimulus being used. The masking stimulus can be generated via sources other than the ABR equipment, such as an audiometer, as long as effective masking levels for the tone burst stimuli are established.

CAUTIONS AND CONSIDERATIONS

There are a number of factors to keep in mind to assure accurate recording of ABRs and correct interpretation of results. Some of the many factors that we consider when using ABR to predict threshold sensitivity are the following.

Neural Abnormalities

Measuring AEPs requires an intact neural system. Thus, abnormalities that can affect the neural system must be considered in interpreting test results. For example, in the presence of hydrocephalus, the ABR can be obliterated despite normal hearing (Kraus et al., 1984). Patients with auditory neuropathy/auditory dys-synchrony (AN/AD) are characterized by absent or highly abnormal ABRs and present OAEs (Starr et al., 1996). Thus, it is particularly important to obtain OAEs in patients who fail to show an ABR at any intensity. Presence of an ABR at high intensities but not to lower intensity stimuli is consistent with a peripheral hearing loss. Absence of an ABR to high and low intensities may mean either a more severe peripheral hearing loss or a neural disorder. OAEs are useful in distinguishing these two groups and therefore we always obtain OAEs in patients who fail to show an ABR response.

Subject Noise

Improper subject preparation can result in noisy or difficult-to-interpret recordings. Poor electrode impedance or dissimilar impedances among electrodes may yield poorly defined, difficult-to-interpret responses. Subjects who are fussy or tense or placed in an uncomfortable position may produce excessive muscle artifact.

Collapsing Ear Canals

Ear canal collapse or earphone slippage can reduce signal intensity at the ear without the examiner's knowledge. We always use insert earphones to avoid the possibility of collapsing ear canals, a common problem in infants when using supra-aural earphones. Insert earphones are also more comfortable which may enhance the patient's state of relaxation or sleep.

Middle-Ear Function

Part of our test battery always includes middle-ear immittance and middle-ear muscle reflexes as well as OAEs. If a patient requires sedation (as in the case of infants over about 4 to 5 months of age and young children) or sleeps deeply, then it is important to obtain these measures *before* doing the ABR. During sedated or natural deep sleep, positive pressure may build up in the middle ears that will compromise the results of middle-ear measures and OAEs if completed at the end of testing.

The ABR Is Not a Hearing Test

Finally, it is always important to remember that the ABR and other AEPs are not hearing tests. In those patients who do not have good neural synchrony, other means of estimating auditory function must be sought. In addition, passing an ABR as an infant does not preclude the possibility

of acquired or later onset hereditary hearing loss. Thus, in reporting test results, we always inform parents and referral sources of the importance of monitoring a child's speech and language development and observing a child's responses to his/her auditory environment. If a child fails to develop speech and language, parents are advised to seek appropriate evaluation and management.

AUDITORY NEUROPATHY/ AUDITORY DYS-SYNCHRONY

The term auditory neuropathy/auditory dys-synchrony (AN/AD), also referred to as auditory neuropathy spectrum disorder (ANSD), describes patients who demonstrate intact outer hair cell function/active cochlear processes shown by OAEs and/or CMs and poor VIII nerve/brainstem responses as a consequence of disturbed input from the inner hair cells, abnormal synaptic function, or peripheral neural pathology (Berlin et al., 1993; Starr et al., 1991, 1996). Further evidence of effects on neural function are demonstrated by generally absent or sometimes elevated middle-ear muscle reflexes (Berlin et al., 2005) and abnormal medial olivocochlear reflexes, measured via efferent stimulation effects on OAEs (Hood et al., 2003). Most AN/AD patients show bilateral symptoms, though function may be asymmetric between ears, and cases of unilateral AN/AD have been documented.

Despite fairly similar findings from current physiological measures, there is considerable variation in characteristics and functional communication abilities across patients (e.g., Berlin et al., 2010; Starr et al., 2000). Clinical presentation typically, but not always, includes difficulty listening in noise, may include fluctuation in hearing ability, and, in the case of infants and children, most often involves delayed or impaired development of speech and language. AN/AD may or may not be accompanied by neural problems in other systems. Patients with AN/AD typically demonstrate timing problems (Zeng et al., 1999), which suggest a disturbance in neural synchrony. This variation impacts both evaluation and management of AN/AD.

Estimates of the incidence of AN/AD suggest that it occurs at a rate of about 10% in those individuals who have a dys-synchronous ABR (see Figure 14.5 for an example of poor synchrony) or an ABR result consistent with a severe or profound estimate of hearing sensitivity. This rate is based on evidence from studies of school-aged children with severe–profound hearing losses (e.g., Berlin et al., 2000) and infant populations (e.g., Rance et al., 1999; Sininger, 2002). Some studies and populations report higher incidence of AN/AD. For example, in the NICU, Berg et al. (2005) observed that about 24% of 477 infants failed their ABR in one or both ears, while passing OAEs bilaterally. To detect AN/AD in newborns, hearing screening must include ABR. If only OAEs are tested, then AN/AD will be overlooked.

Clinical findings in patients with an AN/AD are most accurately described with physiological measures that assess cochlear hair cell and peripheral neural function. On behavioral measures, patients with AN/AD show puretone thresholds ranging from normal sensitivity to the severe or profound hearing loss range (e.g., Berlin et al., 1993; Starr et al., 1996). Speech recognition is variable across individual patients in quiet and generally poorer than expected with ipsilateral competing stimuli (e.g., babble, noise) in all patients with AN/AD.

In infants and young children, ABR is used as the definitive measure in determining AN/AD. ABRs are typically absent in patients with AN/AD, although some patients demonstrate small responses for high-level stimuli. In the germinal paper defining auditory neuropathy, Starr et al. (1996) reported absent ABRs in 9 of 10 patients (aged 4 to 49 years) and the 10th patient had an abnormal ABR characterized by Wave V responses only to high-intensity stimuli. The Berlin et al. (2010) review of ABR data for 186 patients with AN/AD ranging in age from infants through adults indicated that 138 (74%) patients had absent ABRs whereas 48 (26%) showed abnormal responses characterized by presence of low-amplitude Wave V only at high stimulus levels of 75 to 90 dB nHL. This distribution of responses is very similar to that reported by Starr et al. (2000) for 52 patients where 73% had no ABR and 27% had abnormal responses.

The absence or abnormality of all components of the ABR including Wave I suggests that the most distal portions of the VIII nerve are affected, either directly or indirectly, in AN/AD. This characteristic distinguishes AN/AD from space-occupying lesions affecting the VIII nerve, where Wave I of the ABR may be seen in recordings obtained with surface electrodes. Results of radiologic (MRI and CT) evaluation are characteristically normal in AN/AD patients.

Several distinct differences exist between cochlear responses, such as the CM, and neural responses, such as the ABR. These responses can be distinguished by using appropriate recording methods. The most direct method of separating the CM and ABR is to compare responses obtained with rarefaction polarity stimuli to those obtained with condensation stimuli. CM follows the characteristics of the external stimulus; thus, the direction of the CM reverses with a change in polarity of the stimulus. For higher frequency stimuli and clicks, neural responses such as the ABR in normal individuals may show slight latency shifts with polarity changes but do not invert. Therefore, cochlear and neural components can be distinguished based on whether or not the peaks invert with reversing stimulus polarity. Refer to Figure 14.5 that shows ABRs obtained to both condensation and rarefaction polarity stimuli. In the left panel, the CM inverts at the beginning of the tracing whereas the ABR (neural response) does not invert. In AN/AD, the entire response inverts with polarity changes confirming that it is completely CM and not ABR activity.

An important consideration in evaluating ABRs in newborns and infants is neuromaturation of the ABR after birth that continues through 12 to 18 months of age. Although the ABR is typically present at birth, it is possible that factors

such as premature birth, risk factors, or other trauma surrounding birth may delay development of synchronous neural responses. More information is needed to adequately understand the reasons for poor synchrony and the factors that may contribute to later development of the ABR in some infants who initially present with the signs of AN/AD. At present, estimates of the number of newborn infants who present with dys-synchronous ABRs and later develop a normal ABR are unclear. In the meantime, it is important to closely monitor infants over the first year of life both with ABR and with other indices of auditory development, continually modifying management plans as needed.

CASE STUDIES

Two case studies are shown here to exemplify the use of tone bursts in ABR testing. Responses to tone bursts centered at frequencies ranging from 500 to 4,000 Hz, as described in this chapter, provide the information needed to make predictions about the degree and configuration of hearing thresholds. When predicted thresholds are not normal, sufficient information is obtained to proceed directly with appropriate management.

Case Study 1: Normal Hearing

Case study 1 is an 18-month-old female who was referred for testing based on delayed speech and language development. Development was otherwise normal. There was history of hyperbilirubinemia and the possibility of AN/AD was considered, prompting the recommendation of an ABR.

Middle-ear tests and OAEs were within the normal range for each ear. Following a check for neural synchrony, as described above, in each ear, tone burst ABRs were completed. Testing began with tone bursts centered at 2,000 Hz, followed by 500 Hz and 4,000 Hz. Testing of tone bursts centered at 1,000 Hz was not completed because of patient restlessness. However, with information in both ears at three frequencies, sufficient information was available to make threshold predictions.

Replicated responses were obtained at several intensity levels for each tone burst stimulus. Not all responses are shown; rather those used to determine response threshold are displayed. As shown in Figure 14.6 and in Table 14.3, ABR threshold was 40 and 30 dB nHL for the left and right ears, respectively, for tone bursts centered at 500 Hz, 30 dB nHL in each ear for tone bursts centered at 2,000 Hz, and 20 dB nHL in each ear for tone bursts centered at 4,000 Hz. Correction factors, based on the Ontario program guidelines, were used to obtain estimated hearing levels. Predicted thresholds for all frequencies were within normal limits for both ears. Therefore, test results ruled out AN/AD and also suggested that peripheral hearing loss was not a factor in this child's speech and language delay. This child and her family were referred for speech/language intervention.

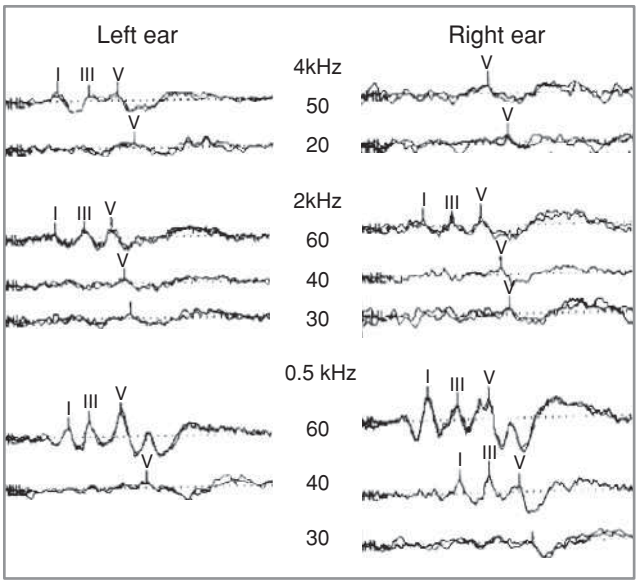


FIGURE 14.6 ABRs obtained for tone bursts centered at 500, 2,000, and 4,000 Hz for the pediatric patient described in case study 1. The time window for all responses is 20 ms.

Case Study 2: Moderate Hearing Loss

Case study 2 is a 2-year-old male who was referred for testing based on a history of middle-ear problems and a family history of hearing loss. Parents were concerned with speech and language development and lack of responses to sound in some situations. Development was otherwise normal.

Middle-ear tests were within the normal range for each ear at the time of ABR testing. OAEs were absent in both ears. Following a check for neural synchrony, as described above, in each ear, tone burst ABRs were completed. Testing

TABLE 14.3

Case Study 1: Normal Hearing

	L-500	L-2000	L-4000	R-500	R-2000	R-4000
dB nHL	40	30	20	30	30	20
Correction	20	10	5	20	10	5
dB eHL	20	20	15	10	20	15

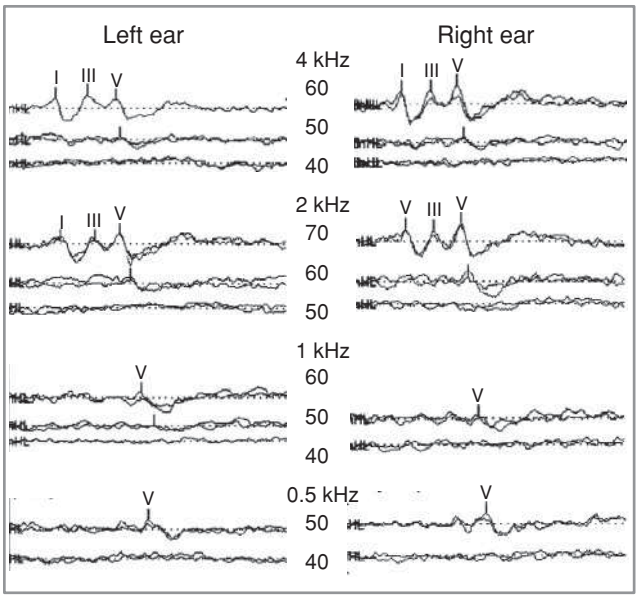


FIGURE 14.7 ABRs obtained for tone bursts centered at 500, 1,000, 2,000, and 4,000 Hz for the pediatric patient described in case study 2. The time window for all responses is 20 ms.

began with tone bursts centered at 2,000 Hz, followed by 500, 1,000, and 4,000 Hz.

Replicated responses were obtained at several intensity levels for each tone burst stimulus. As with the previous case presented, not all responses are shown; rather those used to determine response threshold are displayed. As shown in Figure 14.7 and in Table 14.4, ABR threshold was 50 dB nHL in each ear for tone bursts centered at 500 Hz, 50 dB nHL in each ear for tone bursts centered at 1,000 Hz, 60 dB nHL for each ear for tone bursts centered at 2,000 Hz, and 50 dB nHL in each ear for tone bursts centered at 4,000 Hz. Correction factors, based on the Ontario program guidelines, were used to obtain estimated hearing levels. Predicted air-conduction thresholds were consistent with a mild-to-moderate hearing loss bilaterally. Because air-conduction thresholds were not normal, bone-conduction ABRs were completed using alternating polarity tone bursts. Bone-conduction ABR thresholds were similar to air-conduction thresholds, consistent with a sensory/neural hearing loss. Based on the air- and bone-conduction test results, this child and his family were referred for speech/language intervention and management with amplification.

SUMMARY

A number of considerations to increase accuracy and efficiency of the ABR in pediatric threshold prediction have been presented through discussion of normal characteristics, considerations in improving test sensitivity and accuracy, and in the context of a clinical test protocol. Use of a test battery and cross-check principles is important in all assessments. History, physiological, and behavioral test results need to make sense (agree). Efficiency is needed in minimizing time and prioritizing information. It is important to use strict criteria and technically correct methods in both physiological and behavioral testing. Finally, whereas the ABR is *not* a hearing test per se, the information related to auditory function and hearing threshold sensitivity obtained from physiological measures *can* and *should* be used in implementing management programs.

There is no dispute that the ABR is an objective, indirect method of estimating hearing sensitivity based on the presence of responses at various intensity levels. This is particularly valuable in infants and young children. Ear-specific and frequency-specific information can be obtained and directly applied to management of identified hearing losses. Using ABR in estimating hearing sensitivity can be challenging in assuring accuracy of responses while also obtaining the needed information in a timely manner, a necessity in pediatric patients. Key goals include maximizing the signal, in this case the amplitude of the ABR, while controlling and minimizing noise as much as possible. Although the ABR is well established as a method of predicting hearing thresholds, areas remain for investigation and refinement. Such areas include refinement of stimuli and stimulus paradigms, accurate calibration of signals in infants (e.g., Lightfoot et al., 2007), methods of accurately assessing and controlling noise levels, and removing subjectivity from determination of response presence.

FOOD FOR THOUGHT

1. What do you think is the "most ideal" stimulus or stimulus set for predicting hearing thresholds in infants and young children? Consider the current state of the art, what you want to know when testing an infant, the characteristics of an auditory system that is still maturing, and what might yield the most accurate information.
2. Noise is an issue in evoked potential testing, as electrodes cannot select the response from the noise. Several methods

TABLE 14.4

Case Study 2: Moderate SN Hearing Loss

	L-500	L-1000	L-2000	L-4000	R-500	R-1000	R-2000	R-4000
dB nHL	50	50	60	50	50	50	60	50
Correction	20	15	10	5	20	15	10	5
dB eHL	30	35	50	45	30	35	50	45

hold promise for providing specific information about noise levels that can help in response determination. Some methods have been touched on in this chapter. Think about the noise problems in recording ABRs in infants and discuss (1) what you would do to try to control the noise and (2), more importantly, what approach you might use to assess noise levels in an objective manner.

3. There is subjectivity in ABR related to marking latencies of waveforms and, in the context of this chapter, in deciding the threshold of a response, that is, when a response is present and when there is no response. Discuss the criteria that you would use to decide if a response is present and how you would try to make your decisions as objective as possible.

REFERENCES

Bagatto MP, Moodie ST, Scollie SD, Seewald RC, Moodie KS, Pumford J, et al. (2005) Clinical protocols for hearing instrument fitting in the desired sensation level method. *Trends Amplif.* 9, 199–226.

Bauch CD, Olsen W. (1986) The effect of 2000–4000 Hz hearing sensitivity on ABR results. *Ear Hear.* 7, 314–317.

Berg AL, Spitzer SB, Towers HM, Bartosiewicz C, Diamond BE. (2005) Newborn hearing screening in the NICU: profile of failed auditory brainstem response/passed otoacoustic emission. *Pediatrics.* 116, 933–938.

Berlin CI, Bordelon J, St. John P, Wilensky D, Hurley A, Kluka E, Hood LJ. (1998) Reversing click polarity may uncover auditory neuropathy in infants. *Ear Hear*. 19, 37–47.

Berlin CI, Hood LJ. (2009) Current physiologic bases of audiologic interpretation and management. In: Katz J, Medwetsky L, Burkard R, Hood L, eds. *Handbook of Clinical Audiology.* 6th ed. Philadelphia, PA: Lippincott, Williams and Wilkins; Chapter 22.

Berlin CI, Hood LJ, Cecola RP, Jackson DF, Szabo P. (1993) Does type I afferent neuron dysfunction reveal itself through lack of efferent suppression? *Hear Res.* 65, 40–50.

Berlin CI, Hood LJ, Morlet T, Den Z, Goforth L, Tedesco S, et al. (2000) The search for auditory neuropathy patients and connexin 26 patients in schools for the deaf. *ARO Abstr.* 23, 23.

Berlin CI, Hood LJ, Morlet T, Wilensky D, Li L, Rose Mattingly K, et al. (2010) Multi-site diagnosis and management of 260 patients with auditory neuropathy/dys-synchrony (auditory neuropathy spectrum disorder). *Int J Audiol.* 49, 30–43.

Berlin CI, Hood LJ, Morlet T, Wilensky D, St. John P, Montgomery E, et al. (2005) Absent or elevated middle ear muscle reflexes in the presence of normal otoacoustic emissions: a universal finding in 136 cases of auditory neuropathy/dys-synchrony. *J Am Acad Audiol.* 16, 546–553.

Dau T, Wegner O, Mellert V, Kollmeier B. (2000) Auditory brainstem responses with optimized chirp signals compensating basilar-membrane dispersion. *J Acoust Soc Am*. 107, 1530–1540.

Davis H. (1976) Principles of electric response audiometry. *Ann Otol Rhinol Laryngol.* 85(suppl 28) (3 Pt3), 1–96.

Davis H, Hirsh SK, Turpin LL, Peacock ME. (1985) Threshold sensitivity and frequency specificity in ABR audiometry. *Audiology.* 24, 54–70.

Delgado RE, Özdamar Ö. (2004) Deconvolution of evoked responses obtained at high stimulus rates. *J Acoust Soc Am.* 115, 1242–1251.

Don M, Allen AR, Starr A. (1977) Effect of click rate on the latency of auditory brain stem responses in humans. *Ann Otolaryngol.* 86, 186–195.

Don M, Eggermont JJ. (1978) Analysis of the click-evoked brainstem potentials in man using high-pass noise masking. *J Acoust Soc Am*. 63, 1084–1092.

Don M, Eggermont JJ, Brackmann DE. (1979) Reconstruction of the audiogram using brain stem responses and high-pass masking noise. *Ann Otol Rhinol Laryngol Suppl.* 88(suppl. 57), 1–20.

Don M, Elberling C, Waring M. (1984) Objective detection of averaged auditory brainstem responses. *Scand Audiol.* 13, 219–228.

Don M, Ponton CW, Eggermont JJ, Masuda A. (1994) Auditory brainstem response (ABR) peak amplitude variability reflects individual differences in cochlear response times. *J Acoust Soc Am.* 96, 3476–3491.

Elberling C, Don M. (1987) Threshold characteristics of the human auditory brain stem response. *J Acoust Soc Am.* 81, 115–121.

Elberling C, Don M. (2008) Auditory brainstem responses to a chirp stimulus designed from derived-band latencies in normal-hearing subjects. *J Acoust Soc Am*. 124, 3022–3037.

Elberling C, Don M. (2010) A direct approach for the design of chirp stimuli used for the recording of auditory brainstem responses. *J Acoust Soc Am.* 128, 2955–2964.

Ferm I, Lightfoot G, Stevens J. (2013) Comparison of ABR response amplitude, test time, and estimation of hearing threshold using frequency specific chirp and tone pip stimuli in newborns. *Int J Audiol.* 52, 419–423.

Fobel O, Dau T. (2004) Searching for the optimal stimulus eliciting auditory brainstem response in humans. *J Acoust Soc Am.* 116, 2213–2222.

Galambos R, Makeig S, Talmachoff PJ. (1981) A 40-Hz auditory potential recorded from the human scalp. *Proc Natl Acad Sci.* 78, 2643–2647.

Gorga MP, Kaminski JR, Beauchaine KA. (1991) Effects of stimulus phase on the latency of the auditory brainstem response. *J Am Acad Audiol.* 2, 1–6.

Gorga MP, Kaminski JR, Beauchaine KA, Jesteadt W. (1988) Auditory brain-stem responses to tone bursts in normally hearing subjects. *J Speech Hear Res.* 31, 87–97.

Gravel JS, Hood LJ. (1999) Pediatric audiologic assessment. In: Musiek FE, Rintelmann WF, eds. *Contemporary Perspectives in Hearing Assessment.* Boston, MA: Allyn and Bacon.

Hecox KE, Galambos R. (1974) Brain stem auditory evoked responses in human infants and adults. *Arch Otolaryngol.* 99, 30–33.

Hood LJ, Berlin CI, Bordelon J, Rose K. (2003) Patients with auditory neuropathy/dys-synchrony lack efferent suppression of transient evoked otoacoustic emissions. *J Am Acad Audiol.* 14, 302–313.

Jewett D, Williston J. (1971) Auditory evoked far fields averaged from the scalp of humans. *Brain.* 94, 681–696.

Johnson TA, Brown CJ. (2005) Threshold prediction using the auditory steady-state response and the tone burst auditory brain stem response: a within-subject comparison. *Ear Hear.* 26, 559–576.

Joint Committee on Infant Hearing. (2007) Year 2007 Position Statement: principles and guidelines for early hearing detection and intervention programs. *Pediatrics.* 120(4), 898–921.

Kileny P. (1981) The frequency specificity of tone-pip evoked auditory brain stem responses. *Ear Hear.* 2, 270–275.

Kraus N, Özdamar Ö, Heydemann PT, Stein L, Reed N. (1984) Auditory brainstem responses in hydrocephalic patients. *Electroencephalogr Clin Neurophysiol.* 59, 310–317.

Kristensen SGB, Elberling C. (2012) Auditory brainstem responses to level-specific chirps in normal hearing adults. *J Am Acad Audiol.* 23, 712–721.

Kuwada S, Anderson JS, Batra R, Fitzpatrick DC, Teissier N, D'Angelo WR. (2002) Sources of the scalp-recorded amplitude-modulation following response. *J Am Acad Audiol.* 13, 188–204.

Lightfoot G, Sininger Y, Burkard R, Lodwig A. (2007) Stimulus repetition rate and the reference levels for clicks and short tone bursts: a warning to audiologists, researchers, calibration laboratories, and equipment manufacturers. *Am J Audiol.* 16, 94–95.

Maloff ES, Hood LJ. (2014) A comparison of auditory brain stem responses elicited by click and chirp stimuli in adults with normal hearing and sensory hearing loss. *Ear Hear.* 35, 271–282.

Moore BCJ. (2004) *An Introduction to the Psychology of Hearing.* 5th ed. San Diego, CA: Elsevier Academic Press.

Moore JK, Ponton CW, Eggermont JJ, Wu BJ, Huang JQ. (1996) Perinatal maturation of the auditory brain stem response: changes in path length and conduction velocity. *Ear Hear.* 17, 411–418.

Oates P, Stapells DR. (1997) Frequency specificity of the human auditory brainstem and middle latency responses to brief tones. I. High-pass noise masking. *J Acoust Soc Am.* 102, 3597–3608.

Orlando MS, Folsom RC. (1995) The effects of reversing polarity of frequency-limited single-cycle stimuli on the human auditory brain stem response. *Ear Hear.* 16, 311–320.

Picton TW. (1978) The strategy of evoked potential audiometry. In: Gerber SE, Mencher GT, eds. *Early Diagnosis of Hearing Loss.* New York: Grune and Stratton; pp 279–307.

Picton TW, Champagne SC, Kellett AJC. (1992) Human auditory evoked potentials recorded using maximum length sequences. *Electroencephalogr Clin Neurophysiol.* 84, 90–100.

Picton TW, Hillyard SA, Krausz HI, Galambos R. (1974) Human auditory evoked potentials: I. Evaluation of components. *Electroencephalogr Clin Neurophysiol.* 36, 179–190.

Picton TW, Ouellette J, Hamel G, Smith A. (1979) Brainstem evoked potentials to tone pips in notched noise. *J Otolaryngol.* 8, 289–314.

Pratt H, Sohmer H. (1976) Intensity and rate functions of cochlear and brain stem evoked responses to click stimuli in man. *Arch Otorhinolaryngol.* 212, 85–92.

Rance G, Beer DE, Cone-Wesson B, Shepherd RK, Dowell RC, King AM, et al. (1999) Clinical findings for a group of infants and young children with auditory neuropathy. *Ear Hear.* 20, 238–252.

Salamy A. (1984) Maturation of the auditory brainstem response from birth through early childhood. *J Clin Neurophysiol.* 1, 293–329.

Schoonhoven R. (1992) Dependence of auditory brainstem response on click polarity and high-frequency sensorineural hearing loss. *Audiology.* 31, 72–86.

Sininger YS. (1993) Auditory brain stem response for objective measures of hearing. *Ear Hear.* 14, 23–30.

Sininger YS. (1995) Filtering and spectral characteristics of average auditory brain-stem response and background noise in infants. *J Acoust Soc Am.* 98, 2048–2055.

Sininger YS. (2002) Auditory neuropathy in infants and children: implications for early hearing detection and intervention programs. *Audiol Today.* 14, 16–21.

Sininger YS, Cone-Wesson B, Folsom RC, Gorga MP, Vohr BR, Widen JE, et al. (2000) Identification of neonatal hearing impairment: auditory brain stem responses in the perinatal period. *Ear Hear.* 21, 383–399.

Sininger YS, Masuda A. (1990) Effect of click polarity on ABR threshold. *Ear Hear.* 11, 206–209.

Spivak L. (1993) Spectral composition of infant auditory brainstem responses: implications for filtering. *Audiology.* 32, 185–194.

Stangl S, Rentmeester L, Hood LJ. (2013) Auditory brainstem responses to clicks, chirps, tonebursts, and octave-band chirps. American Auditory Society Annual Meeting, Scottsdale, Arizona.

Stapells DR. (2000) Threshold estimation by the tone-evoked auditory brainstem response: a literature meta-analysis. *J Speech Lang Pathol Audiol.* 24, 74–83.

Stapells DR, Gravel JS, Martin BA. (1995) Thresholds for auditory brain stem responses to tones in notched noise from infants and young children with normal hearing and sensorineural hearing loss. *Ear Hear.* 16, 361–371.

Stapells DR, Linden D, Suffield JB, Hamel G, Picton TW. (1984) Human auditory steady state potentials. *Ear Hear.* 5, 105–113.

Starr A, Achor LJ. (1975) Auditory brain stem responses in neurological disease. *Arch Neurol.* 32, 761–768.

Starr A, Amlie RN, Martin WH, Sanders S. (1977) Development of auditory function in newborn infants revealed by auditory brainstem potentials. *Pediatrics.* 60, 831–839.

Starr A, McPherson D, Patterson J, Don M, Luxford W, Shannon R, et al. (1991) Absence of both auditory evoked potentials and auditory percepts dependent on timing cues. *Brain.* 114, 1157–1180.

Starr A, Picton TW, Sininger YS, Hood LJ, Berlin CI. (1996) Auditory neuropathy. *Brain.* 119, 741–753.

Starr A, Sininger YS, Pratt H. (2000) The varieties of auditory neuropathy. *J Basic Clin Physiol Pharmacol.* 11, 215–230.

Stockard JJ, Stockard JE, Westmoreland B, Corfits J. (1979) Brainstem auditory-evoked responses: normal variation as a function of stimulus and subject characteristics. *Arch Neurol.* 36, 823–831.

Stuart A, Yang EW, Green WB. (1994) Neonatal auditory brainstem response thresholds to air- and bone-conducted clicks: 0 to 96 hours postpartum. *J Am Acad Audiol.* 5, 163–172.

Teas DC, Eldredge DH, Davis H. (1962) Cochlear responses to acoustic transients: an interpretation of whole-nerve action potentials. *J Acoust Soc Am.* 34, 1438–1459.

Terkildsen K, Osterhammel P, Huis in't Veld F. (1975) Far-field electrocochleography: frequency specificity of the response. *Scand Audiol.* 4, 167–172.

Wegner O, Dau T. (2002) Frequency specificity of chirp-evoked auditory brainstem responses. *J Acoust Soc Am.* 111, 1318–1329.

Yang Y, Stuart A. (1990) A method of auditory brainstem response testing of infants using bone-conducted clicks. *JSLPA/ROA.* 14, 69–76.

Yoshinaga-Itano C, Sedey AL, Coulter DK, Mehl AL. (1998) Language of early- and later-identified children with hearing loss. *Pediatrics.* 102, 1161–1171.

Zeng FG, Oba S, Garde S, Sininger Y, Starr A. (1999) Temporal and speech processing deficits in auditory neuropathy. *Neuroreport.* 10, 3429–3435.

CHAPTER 15

Auditory Steady-State Response

Andrew Dimitrijevic and Barbara Cone

OVERVIEW

Auditory steady-state responses (ASSRs) are rhythmic brain potentials evoked by regularly repeating stimuli such as clicks, amplitude-modulated (AM) noise or tones, or frequency-modulated (FM) tones.

Imagine the waveform of an auditory brainstem response if two toneburst stimuli were presented within an averaging epoch. Each toneburst would be expected to produce a response, and so the response waveform would be repeated twice, within the averaged epoch. Now, imagine a 125-ms train of 2-1-2 cycle tonebursts, say at 2,000 Hz (carrier frequency [CF]), with an interstimulus interval between each burst at 12.5 ms (80 Hz). Imagine that the response averaging epoch is also 125 ms in duration. One thousand 125-ms trains are presented, and the response to each train is averaged. There are 10 responses represented in the time-averaged waveform for the 125-ms sample. The time-averaged waveform appears as a series of peaks or a periodic wave, with a 12.5-ms interpeak interval (Figure 15.1A). This is one way of conceptualizing an ASSR. The ASSR, furthermore, can be obtained for a wide range of modulation frequencies (MFs), although rates between 10 and 200 Hz have been most often investigated. For example (Figure 15.1B), the 40-Hz ASSR is obtained using clicks or tonebursts presented every 25 ms, or puretones or noise amplitude modulated at 40 Hz. Because the ASSR is periodic (repeating at the same rate as the MF), it can be analyzed using frequency-domain methods. The spectrum of the response will show a peak at the repetition rate, that is, at the MF. The latency of this periodic waveform can be expressed as a phase delay (360 degrees), relative to the onset of the start of the stimulus train, or onset modulation of tones or noise. When a response is present, the phase of the response is consistent (phase coherent) across subaverages and is "phase-locked," occurring at a specific phase relative to the MF. The phase coherence (PC)/phase-locking can be tested for statistical significance in addition to, or in place of, the statistical test of response spectral peak amplitude. The presence of an ASSR is dependent on the integrity of the auditory periphery (external, middle, and inner ear, auditory nerve) for the CF, as well as the integrity of higher levels of the auditory pathway.

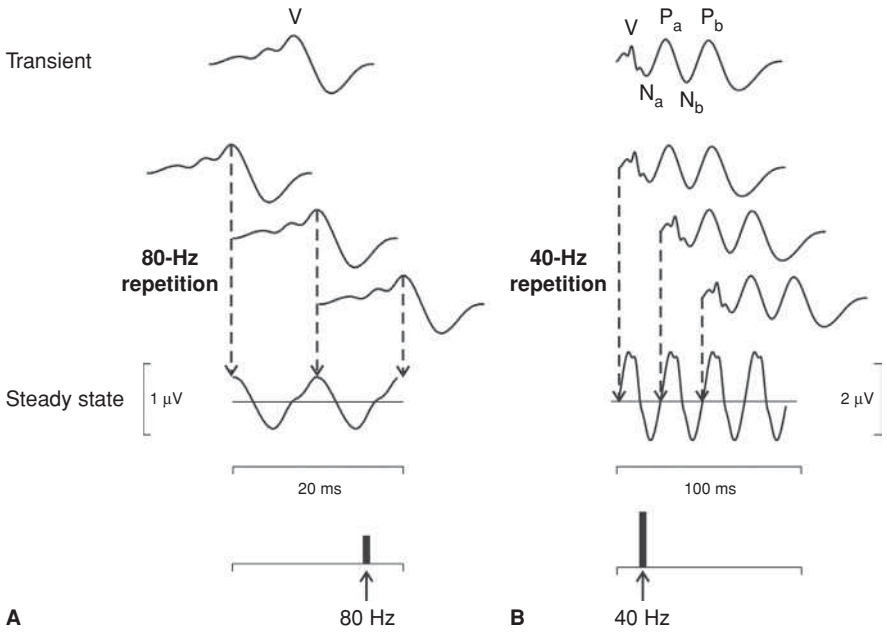


FIGURE 15.1 Examples of transient-evoked potentials and how they are related to steady-state potentials. [**A**] With a modulation frequency of 80 Hz, there are brainstem responses occurring every 12.5 ms, the same period as the modulated (tone) stimulus. [**B**] With a stimulus modulation frequency of 40 Hz, the responses occur every 25 ms, the same period as the stimulus modulation.

Since the first inclusion of ASSRs in the fifth edition of the *Handbook of Clinical Audiology,* there has been a significant increase in the clinical application of ASSRs. The primary clinical application of ASSR during the past two decades has been for estimating hearing thresholds in infants and children, which is the focus of this chapter. The ASSR is also a tool for investigating responses to complex stimuli at suprathreshold levels. These emerging applications will be highlighted in this chapter.

FUNDAMENTALS

Knowledge of neural generators, stimulus–response relationships, subject-related response factors, signal processing, and detection algorithms is necessary for interpreting ASSR results. Each of these areas will be reviewed.

Neural Generators

The generation of the ASSR at the level of the cochlea and nerve is schematized in Figure 15.2 (after Lins et al., 1995). An AM tone is the stimulus. The first step in sensory transduction occurs at the level of the inner hair cells. As the basilar membrane vibrates, the stereocilia on the inner hair cells move back and forth following the sound stimulation. This symmetric movement is shown in the figure as a sinusoid. Integrity of the outer and inner hair cell transduction systems is needed to obtain a normal response to the tone. The transmission of information from the inner hair cells to the auditory nerve involves the release of glutamate when the stereocilia move in one direction thereby initiating an action potential. Because the action potentials are generated by movement of stereocilia in only one direction, the stimulus (tone) undergoes half-wave rectification. Half-wave rectification provides energy at the modulation frequency. There is no stimulus energy at the modulation frequency, yet the half-wave rectification introduces energy at the modulation frequency. It is this energy at the modulation frequency that evokes the ASSR. Supporting evidence of this model is seen in recordings from auditory nerve made by Khanna and Teich (1989a, 1989b), who showed that presenting AM or FM stimuli evoked responses in the auditory nerve at the MFs, at the harmonics of the MFs, and at the CF.

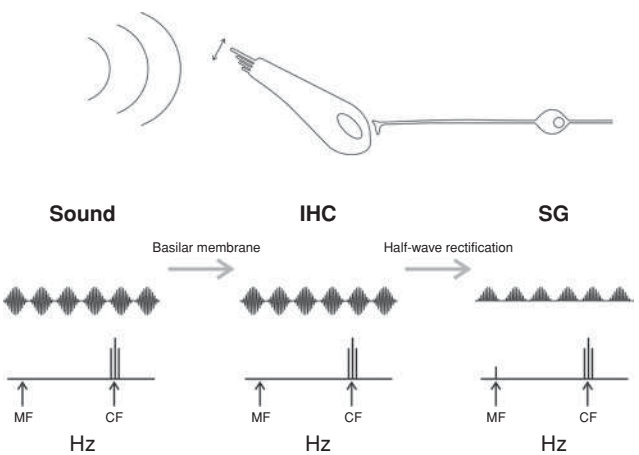


FIGURE 15.2 A model of ASSR generation at the level of the cochlea and eighth nerve. The modulated tone creates a basilar membrane vibration at the "best place" for the carrier frequency. There is no energy present at the modulation frequency. Inner hair cells release neurotransmitters to the peripheral processes of the spiral ganglion cells. This provides a half-wave rectification of the stimulus providing energy for the neural response at the modulation frequency.

Neurons of the eighth nerve (Ruggero, 1992), cochlear nucleus (Rhode and Greenberg, 1992), inferior colliculus (IC; Irvine, 1992), and primary auditory cortex (Clarey et al., 1992) are responsive to AM and FM signals, and so could be involved in the generation of the ASSR. One line of evidence that points to a relationship between MF and the underlying neural generator is that of ASSR latency. Measurement of the response phase spectrum (relative to the MF) can be used to estimate response latency. The predominant phase is used to characterize the latency of the response, and hence the generators are assumed to be the same as those for the transient-evoked response of similar latency. Modulation rates of 20 Hz or less will result in a response dominated by those generators that are responsible for the late cortical-evoked potential, specifically primary auditory cortex and association areas. For modulation rates higher than 20 Hz but lower than 50 Hz, the response characteristics are similar to those found for the middle latency auditory-evoked response (MLAER), with generators generally thought to be auditory mid-brain, thalamus, and primary auditory cortex (Kraus et al., 1994). Modulation rates higher than 50 Hz will be dominated by evoked potentials from brainstem sites, including those for Wave V and its subsequent negative trough, sometimes identified as SN-10 (Møller, 1994).

Chemical lesions of the auditory pathway were used by Kuwada et al. (2002) to determine neural generators of the ASSR. Using a rabbit model, they administered pharmacologic substances that reduced activity at selected levels of the auditory system, while recording ASSRs as MF was varied. As MF was increased, phase delay (latency) decreased. Estimated latency for MFs <100 Hz was 27 ms, suggesting a cortical generator. At rates above 100 Hz, latencies of 5 ms or less were more consistent with brainstem generators. When potassium chloride was administered topically to the cortex (to depress cortical activity), the ASSRs for MFs <100 Hz were significantly decreased, whereas those for MFs >100 Hz were stable. Szalda and Burkard (2005) recorded ASSRs from the IC and auditory cortex sites in awake and nembutal-anesthetized chinchillas, as the modulation frequency of a 2,000-Hz tone was varied from 29 to 249 Hz in 20-Hz steps. The IC responses were largest at modulation rates of 109 and 170 Hz. A different result was

obtained from the auditory cortex. In the awake state, the auditory cortex had large responses at 29 and 70 Hz, but when anesthetized, the amplitude of the ASSR was greatly reduced and the amplitudes were largest at 29 Hz. These results are consistent with those of Kuwada et al. (2002), in that more robust responses were found for higher modulation rates at the IC compared to auditory cortex, whereas the cortex had more robust responses to lower modulation rates.

Studies in human subjects, using the techniques of multichannel EEG/MEG for brain source analysis, functional magnetic resonance imaging (fMRI), and positron emission tomography (PET), indicate both brainstem and cortical neural generators of the ASSR. Herdman et al. (2002a) investigated the neural generators of ASSR for modulation rates of 12, 39, and 88 Hz in adults using dipole source modeling techniques. The results showed that the brainstem source was active for all three rates of stimulation, whereas the cortical sources were predominant for the two lower rates, although the ASSRs at 12 Hz were very low in amplitude. Estimated latencies of the ASSR were also consistent with a brainstem site of generation for the 88-Hz ASSR, and cortical site(s) for the 39- and 12-Hz modulation rates. PET was used to study the generators of the 40-Hz ASSR in adults (Reyes et al., 2004). The investigators distinguished the cortical areas activated by an AM tone from those activated by a puretone. They showed that bilateral activation of primary auditory cortices, left medial geniculate, and right middle frontal gyrus, as well as the right anterior cingulate gyrus and an area of right auditory cortex, was specifically by the AM stimulus. The PET technique used in this study would not be sensitive to brainstem sites of activation, so these cannot be ruled out. A recent study (Steinmann and Gutschalk, 2011) examined the use of fMRI and MEG for ASSR localization in the same adults on different testing days. A blood oxygen–level–dependent (BOLD) activation in the fMRI contrast between puretones and AM tones indicated medial Heschl gyrus activation with ASSRs. In the same subjects, there was a close correspondence between ASSR dipole source location and BOLD activation in the same group of subjects. This provided good correspondence with previous results showing medial activation of Heschl gyrus (more medial than for a transient-evoked response) with ASSRs (Herdman et al., 2002a).

In summary, the ASSR has multiple generators, although the contributions of the generators vary with MF. In humans, MFs >80 Hz are thought to be generated predominantly by brainstem sites, although the contribution of cortical generators is still present. At lower MFs, the medial geniculate body, auditory radiation, and primary auditory cortex are thought to contribute to the ASSR. As for other cortical-evoked responses, ASSRs at low modulation rates show laterality toward the hemisphere contralateral to the stimulated ear, although 40-Hz ASSRs show evidence of right hemispheric dominance (Ross et al., 2005).

Stimulus Factors

Carrier Frequency

Auditory sensitivity varies as a function of CF, as does ASSR threshold. The difference between ASSR and behavioral threshold also varies with CF. In general, the ASSR threshold is lowest in the mid-frequency range, at 1.5 and 2.0 kHz, in comparison to thresholds at lower or higher CFs. Phase delays decrease systematically with CF, reflecting the tonotopic organization of the cochlea, and in support of the data demonstrating that the ASSR is somewhat place specific (Herdman et al., 2002b).

The data about the effect of CF on ASSR are from experiments in which ASSR threshold was measured. Variability in the methods used in these studies contributes to the variability in the results. Some investigators report ASSR threshold in dB HL, for which thresholds are "normalized" to an audiometric calibration, and others report thresholds in dB SPL. Another consideration is that ASSR threshold is determined using algorithms based on an estimate of the signal-to-noise ratio (SNR). Longer averaging times decrease the background noise level, thus improving the SNR, yielding lower (better) thresholds. A further consideration is that there are relatively little threshold data for those with normal hearing; the bulk of published threshold data are for those with hearing loss. Table 15.1 includes ASSR threshold data as a function of frequency in adults and infants with normal hearing. The estimation of perceptual threshold for puretones from ASSR threshold is discussed in a later section of this chapter.

AM and FM Depth

John et al. (2001b) evaluated the effect of AM and FM depth on ASSRs obtained from adults with normal hearing. A sinusoidal function at 82 Hz was used to amplitude modulate a 60-dB SPL 1,000-Hz tone at 100%, 50%, 20%, 10%, and 5%. Then, the 1.0-kHz carrier was frequency modulated (at 82 Hz) at depths of 50%, 20%, 10%, 5%, and 2%. ASSR amplitude decreased with modulation depth by 0.5 nV/% over a 20% to 100% AM change, but the AM change was almost twice as steep for a change in FM depth. ASSR amplitudes in response to a 20% FM tone were larger than those for a 100% AM tone. There was no change in phase delay (latency) as modulation depth was varied. Fewer than 50% of responses reached statistical significance for AM depths less than 20% and for FM depths less than 5%.

John et al. (2001b) studied the advantage of using both AM and FM (called mixed modulation [MM]) for the same CF. ASSR amplitudes were significantly larger in response to MM tones in comparison to responses to AM alone. Part of the reason is the spread of spectral energy for the MM signal. Sidebands for the AM will be at the CF ± MF, and for FM at CF ± integer multiples of the MF. The interaction of

TABLE 15.1

80-Hz ASSR Thresholds in Adults and Infants with Normal Hearing

Study	Subjects (*N*)	Stimuli	Level	500 Hz	1,000 Hz	2,000 Hz	4,000 Hz
Adults							
Aoyagi et al. (1994c)	20	AM	dB HL	34 ± 15 (250 Hz)	28 ± 14	–	30 ± 15
Lins et al. (1996)	15	AM	dB SPL	39 ± 10	29 ± 12	29 ± 11	31 ± 5
Picton et al. (1998)	10	AM	dB SPL	37 ± 10	32 ± 15	30 ± 7	30 ± 7
Herdman and Stapells (2001)	10	AM	dB SPL	22 ± 12	19 ± 10	18 ± 9	20 ± 11
Perez-Abalo et al. (2001)	40	AM	dB SPL	40 ± 10	34 ± 9	33 ± 10	35 ± 10
Cone-Wesson et al. (2002a)	10	AM	dB SPL	52 ± 7	–	–	23 ± 10
Dimitrijevic et al. (2002)	14	MM	dB SL	17 ± 10	4 ± 11	4 ± 8	11 ± 7
Picton et al. (2005) (short-duration average)	10	MM	dB SL[a]	35 ± 16	16 ± 8	18 ± 9	23 ± 15
Picton et al. (2005) (long-duration average)	10	MM	dB SL[a]	21 ± 8	7 ± 8	9 ± 6	13 ± 7
Luts and Wouters (2005) "Master"	10	MM	dB SL[a]	24 ± 11	17 ± 9	14 ± 7	21 ± 11
Luts and Wouters (2005) "Audera"	10	MM	dB SL[a]	48 ± 21	40 ± 21	33 ± 10	30 ± 20
Van der Werff and Brown (2005)	10	MM	dB HL	29 ± 10	23 ± 11	16 ± 6	15 ± 10
Johnson and Brown (2005)	14	AM	dB SL[a]	–	22 ± 5[c]	14 ± 2[c]	–
Johnson and Brown (2005)	14	MM	dB SL[a]		16 ± 4[c]	14 ± 3[c]	–
Small and Stapells (2005)[b]	10	MM	dB HL[a]	22 ± 11	26 ± 13	18 ± 8	18 ± 11
D'haenens et al. (2009)	40	MM	dB HL	24 ± 9	18 ± 8	12 ± 8	16 ± 9
Ishida et al. (2011)[b]	10	MM	dB HL	39 ± 16	34 ± 15	42 ± 14	45 ± 15
Infants							
Rickards et al. (1994) (newborns)	337	MM	dB HL[d]	41 ± 10		24 ± 9 (1,500 Hz)	35 ± 11
Levi et al. (1995) (1 mo)	35	AM	dB SPL	42 ± 16	42 ± 11	34 ± 15	
Lins et al. (1996) (<12 mo)	23	AM	dB SPL	45 ± 13	29 ± 10	26 ± 8	29 ± 10
Savio et al. (2001) (newborns)	25	AM	dB nHL[e]	16 ± 11	22 ± 12	19 ± 12	23 ± 13
Savio et al. (2001) (7–12 mo)	13	AM	dB nHL[e]	9 ± 9	12 ± 10	7 ± 8	9 ± 9
Cone-Wesson et al. (2002c)	85	MM	dB HL[d]	39 ± 8	34 ± 10	26 ± 10	39 ± 12
Rance et al. (2005)	285	MM	dB HL	32 ± 7	32 ± 7	24 ± 6	28 ± 7
Ribeiro et al. (2010) (term)	27	MM	dB SPL	44 ± 10	28 ± 7	27 ± 6	33 ± 6
Ribeiro et al. (2010) (preterm)	21	MM	dB SPL	49 ± 9	26 ± 7	27 ± 8	36 ± 8

The number of subjects, stimulus type, and threshold level are given for each study. AM is a sinusoidally modulated puretone and MM is an amplitude- and frequency-modulated tone. AM modulation depth is 100% and FM modulation depth varies between 10% and 25% in various studies. There are considerable differences in ASSR recording methodology represented in these studies, with some using a single-frequency, sequential test strategy, whereas the others used multiple carriers presented simultaneously. Differences in averaging time, signal processing, and detection algorithm also vary between studies. "80-Hz ASSR" refers to modulation rates in the 70–110-Hz range.

[a]ASSR thresholds are ASSR threshold–puretone threshold for that carrier frequency.

[b]Bone-conduction stimuli.

[c]Threshold and standard error estimated from bar graph.

[d]Re: Adult thresholds.

[e]Re: Adult thresholds. 0 dB nHL = 51, 39, 39, and 34 dB SPL at 0.5, 1.0, 2.0, and 4.0 kHz, respectively.

[f]Cosine^3 (similar to AM^2).

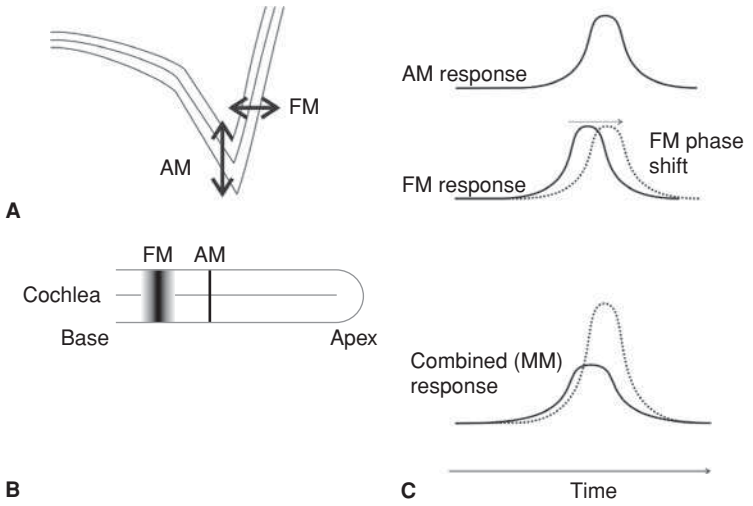


FIGURE 15.3 A model of combined AM and FM response activation. [**A**] The tuning curves for auditory nerve fibers and how the AM activates the fibers slightly apical to the best frequency and FM, slightly basally. [**B**] The area of the basilar membrane activated by both types of modulation. [**C**] The time course of response to each type of modulation and the time course of the response to combined [mixed modulation [MM]] AM and FM tones.

AM and FM accounts for increased MM responses. John et al. suggest that the benefit of MM is governed by the different responses of AM and FM. An FM response maximally stimulates neurons that are slightly higher in frequency than at the CF and therefore occurs slightly earlier than the AM response (Figure 15.3). If the FM phase is adjusted relative to the AM component, so that it is delivered slightly after the AM, the resultant ASSR will be optimized because the "peaks" of AM and FM occur at the same time. This results in a larger ASSR response in comparison to AM or FM alone, or when the phase relationships between AM and FM are not phase adjusted. The researchers demonstrated that when the phase of FM was varied with respect to AM, ASSR amplitude plotted as a function of FM phase delays varied in a sinusoidal fashion, with amplitude crests observed at relative phases of 0 and 270 degrees. Stimulus level and CF were also varied in this experiment, and phase delays decreased with increasing level and CF; this is homologous to the latency changes observed in ABR for changes in stimulus level and toneburst frequency.

This parametric study showed that MM stimuli yield larger ASSR amplitudes, and hence, a greater number of responses are detected, in comparison to results with pure AM stimuli. The study also showed the importance of the phase relationship between the AM and FM stimulus components, to obtain the largest, most easily detected responses. Of course, the spectrum of the stimulus is considerably broader for an AM + FM tone than for AM alone. This would be expected to affect the frequency specificity or cochlear place specificity of the ASSR.

Modulation Type

Clicks, modulated broadband, high-pass, or low-pass noise, will yield large-amplitude ASSRs with low detection thresholds. John et al. (2003) used noise and tones modulated with exponential functions to obtain ASSRs. In all cases, the ASSRs for the exponential modulations were larger than those for conventional, sinusoidal, and MM tones. This is predictable, given that exponential modulation results in steeper rise times that will broaden the stimulus spectra and, in fact, the shape approaches a toneburst. They suggest that high-pass– and low-pass–modulated noise or clicks could be used to quickly estimate threshold in the low- and high-frequency ranges of hearing. This could then be followed by using frequency-specific MM tones, to make more definitive puretone threshold estimates. If threshold can be estimated quickly using less frequency-specific stimuli, then more time can be spent acquiring ASSRs for frequency-specific stimuli at levels closer to threshold. Figure 15.4 shows time- and frequency-domain representations of sinusoidal AM, FM, MM tones, and tones modulated with an exponential function and modulated noise.

Chirps

ASSRs using chirps have been studied (Elberling et al., 2007) to create a stimulus that maximally excites the cochlea and produces a large ASSR. The chirp is an AM and FM stimulus designed to create a cochlear traveling wave that produces synchrony in response to the neural elements innervating the low-, mid-, and high-frequency portions of the cochlea. The chirp stimulus compensates for the normal phase delays introduced by the traveling wave by systematically presenting high frequencies slightly later than low frequencies. These frequency-specific time delays can occur over a broad frequency range (activating the entire cochlea) or be constrained to a narrow range (e.g., near 500 Hz). Stürzebecher et al. (2006) described a stimulus composed of cosine waves ranging from 270 to 810 Hz (average near 500). The phases of the individual components were adjusted so that neural activation would occur synchronously. These chirp stimuli evoked ASSRs with larger SNRs and that were detected earlier compared to those in response to nonadjusted stimuli. The advantage of the chirp stimulus is particularly important for ASSRs in response to low-frequency carriers (i.e., near 500 Hz) that have relatively low SNRs for conventional stimuli.

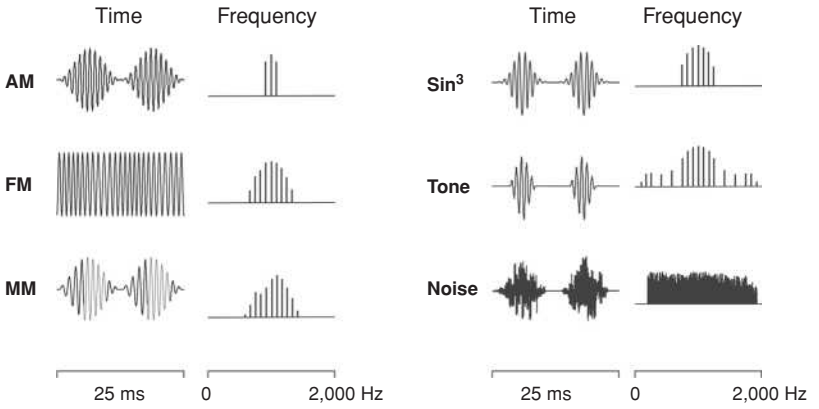


FIGURE 15.4 Time- and frequency-domain representation of stimuli used for ASSR. Waveforms and spectra are shown for (sinusoidal) amplitude modulation (*AM*), frequency modulation (*FM*), mixed modulation ([*MM*], AM+FM), exponential sine-wave modulation (sin^3), toneburst (linear ramp), and modulated noise.

Multiple Modulation and Carrier Frequencies

It is possible to present multiple AM CFs simultaneously and perform a separate analysis for each modulation frequency used in the complex stimulus. Lins and Picton (1995) were the first to show that it was possible to present up to four CFs in both ears and obtain multiple ASSRs. The CFs were 0.5, 1.0, 2.0, and 4.0 kHz and there were eight different modulation frequencies, with the modulation frequency varied for both ear and CF. When suprathreshold-level (60-dB SPL) stimuli were used, there was no difference in response amplitude for the single tone–alone condition, four stimuli combined in one ear, or four stimuli combined in both ears. ASSR threshold was also estimated using two CF tones (500 and 2,000 Hz) in each ear. In normal ears, there does not appear to be a difference in ASSR threshold for four CFs presented simultaneously, compared to when they are presented singly, as long as the CFs are separated by an octave, and the MFs (at 70 Hz or greater) are separated by 3 Hz. An illustration of this multifrequency stimulus is shown in Figure 15.5, with both time (waveforms) and frequency (spectra) domain representations of the stimulus components. Lins et al. (1996), using four simultaneously presented CFs to normal hearing adults, showed that the mean behavioral ASSR threshold difference was 12 dB.

Lins and Picton (1995) also showed that it was possible to measure ASSRs using the same CF (1.0 kHz) and up to four different modulation rates presented simultaneously, with modulation rates in the low (39 and 49 Hz) and high (81 and 97 Hz) ranges. This would appear to be a beneficial technique for testing patients who may be in a variable state of arousal, from alert to deep (stage 4) sleep, as might occur during a typical clinical test session. When adults were tested during an awake state, the amplitudes of the responses to the low MFs (presented individually) were up to three times those of the responses for high MFs (presented individually). When two different MFs were used in each ear, the responses to each MF were decreased by about 20%. In sleeping adults, when four different MFs were presented simultaneously (39, 49, 81, and 97 Hz), the amplitudes of the low MF response components decreased with sleep stage, whereas the amplitudes of the high MF response components remained unchanged. Although only a small group of adults

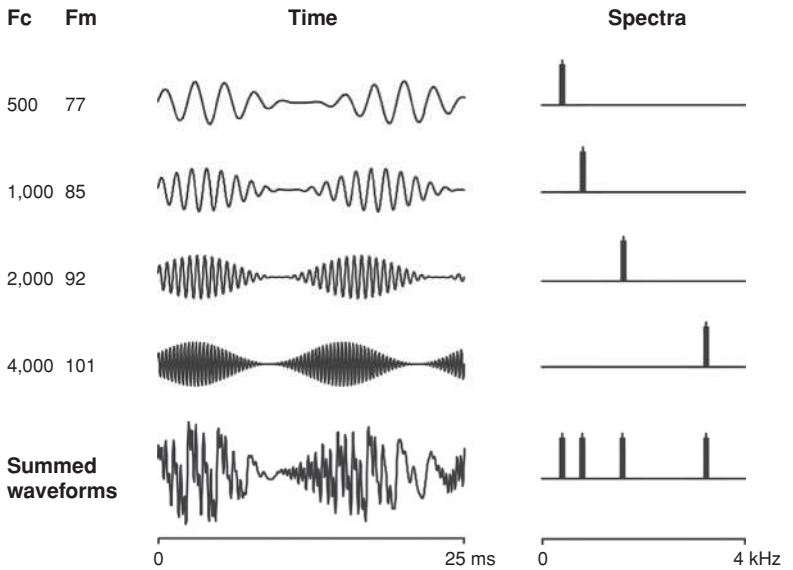


FIGURE 15.5 Time waveforms and spectra for each component of a multifrequency stimulus, consisting of simultaneously presented modulated tones. *Fc* is the carrier (tone) frequency, *Fm* is the modulation frequency (rate). Fcs should be separated by an octave, and Fms by at least 3 Hz to avoid interaction.

were tested in each of these conditions, and the 1,000 Hz CF was at a suprathreshold level, the implications for a clinical test protocol are compelling. That is, if threshold estimates are needed and the patient is in an awake state, the low MF components might be best, but at the same time, high MF components can also be tested. If the patient is drifting from wakefulness to drowsiness and sleep, the stability of the high MF components will yield adequate information.

Cochlear Place Specificity

The cochlear place specificity of the ASSR evoked by MM tones, the most commonly used stimuli for ASSR tests, was evaluated by Herdman et al. (2002b), using the classic high-pass masking derived-band technique. In this method, cochlear response areas are delimited by using masking noise in which the high-pass edge of the filter is systematically lowered, for example, from 8 to 20 kHz, to 4 to 20 kHz, then 2 to 20 kHz, and so forth. An ASSR is obtained for each masker setting. Cochlear response areas are "derived" by subtracting the response obtained for adjacent masking bands, that is, the 4- to 20-kHz ASSR from the 8- to 20-kHz condition. The derived or difference wave represents the response attributed to the 4- to 8-kHz cochlear area. Details about this technique can be found in Chapter 11. Herdman et al. used this technique, with masker high-pass cutoffs at 0.5 octave intervals between 0.250 and 16 kHz. The derived bandwidths for cochlear place of excitation varied from 1.02 to 1.21 octaves. These results indicate slightly narrower derived bandwidths compared to those obtained when a toneburst is used to evoke the ABR, or middle latency response (MLR). Even though the stimulus spectrum of an AM tone may have a narrower, more "frequency-specific" spectrum than does the spectrum of a toneburst, the response "place specificity" appears to be about an octave wide for modulation envelopes or tonebursts that are less than 10 ms in duration. Place specificity is better (narrowest in terms of octaves) for high-frequency CFs (>1.0 kHz) in comparison to low-frequency CFs. Picton et al. (2003) remind us that it is the cochlea that is the limiting factor for place specificity, not the stimulus. Audiogram estimates derived from ASSR tests using MM stimuli appear to be accurate, even when such steep audiometric slopes exist (Herdman and Stapells, 2003), and thus additional masking does not appear to be necessary.

Subject Factors

Age: Infants

ASSR threshold decreases with age during infancy. Rickards et al. (1994) were the first to establish ASSR thresholds in newborns. ASSR threshold was estimated from the results of over 480 tests conducted at 500, 1,500, and 4,000 Hz, using MFs of 72, 85, and 97 Hz, respectively. A statistical criterion of $p < 0.03$ was used to determine when a response was present, using a PC algorithm. The mean thresholds were 41, 24, and 34 dB HL (or 52.5, 30.5, and 44.5 dB SPL) for 500, 1,500, and 4,000 Hz, respectively. The ASSR in newborns shows stimulus–response characteristics, and derived latencies similar to those of the toneburst-evoked ABR.

The 4-CF combined MF technique was applied to infants aged 1 to 10 months by Lins et al. (1996). The MFs were between 75 and 110 Hz. All infants were considered "well-babies," tested during sleep, and assumed to have normal hearing. The mean ASSR thresholds found for the infants tested in a quiet room were within 10 dB of ASSR threshold in adults and were 45 dB SPL for 500 Hz, 29 dB SPL for 1,000 Hz, 26 dB SPL for 2,000 Hz, and 29 dB SPL for 4,000 Hz. The response amplitudes and phases were also measured and compared to those of adults. On average, response amplitudes were less than 50% of those found in adults, but phase measurements were similar.

Savio et al. (2001) showed that ASSR threshold improved with age in the first year of life. In the 0 to 1 month age group, ASSR thresholds were, on average, 13 dB higher than those obtained from infants aged 7 to 12 months. John et al. (2004) measured ASSR amplitude and detectability for AM, FM, MM, and exponentially modulated tones in neonates and also in older infants (3 to 15 weeks) using stimuli at a fixed level of 50 dB HL. ASSR amplitude and detectability increased with age, suggesting that threshold might also improve with age. Reponses were largest for MM and AM tones with exponential modulations, suggesting that these stimuli would be best for testing threshold in very young infants.

Rance and Tomlin (2006) performed longitudinal ASSR threshold measures over the first 6 weeks of a life in a cohort of full-term infants with normal hearing. They found an improvement in ASSR threshold of 11 dB at 0.5 kHz and 10 dB at 4.0 kHz when comparing thresholds measured in the newborn period to those measured at 6 weeks of age. These threshold differences were obtained after taking into account the level differences owing to the ear canal acoustics (obtained from in situ stimulus calibration).

Unlike the ABR, there have been no large-scale parametric studies of ASSR development in infants and young children, the population most likely to undergo testing of this nature to estimate threshold. Indeed, most of the published ASSR results in infants and children concern those who have hearing loss, and it is difficult to infer normal development from results obtained in pathologic ears. ASSR tests utilize modulation rates much higher than toneburst presentation rates typically used to evoke the ABR, rates at which considerable neural adaptation in both premature and full-term neonates is known to occur (Lasky, 1984). Optimization of ASSR test parameters for very young infants, particularly for threshold estimation applications, requires further research.

Age: Adults

Although there is experimental evidence that there is an increase from 38 to 46 Hz in the peak of the function

relating ASSR amplitude to modulation rate that occurs in middle age (Poulsen et al., 2007), the 40-Hz ASSR does not change significantly with increasing age in adulthood (Boettcher et al., 2001), although even slight-mild hearing loss among older adults may be a confounding variable. Larger amplitudes for the ASSR have been found among the elderly who have hearing levels at the "lower" end of normal (e.g., 20 to 25 dB HL), likely because of a recruitment-like phenomenon (Muchnik et al., 1993). Picton et al. (2003) report no age-related changes in the amplitude or phase of ASSRs for a 1,000-Hz puretone modulated at 3, 43, and 95 Hz in a group of normal hearing adults aged 20 to 81 years. They do, however, report high intersubject variability in amplitude and phase measures that may have precluded finding statistically significant age-related differences. Grose et al. (2009) found some decrement in ASSR amplitudes for older adults but only for high (>100 Hz) modulation frequencies.

Stimulus × Subject Interactions

MF × Subject State

The modulation frequency, modulation type(s), and CF are the primary determinants of ASSR properties. There are, however, some interactive effects of subject consciousness with MF and CF on the ASSR. These were first evaluated by Cohen et al. (1991). CFs of 250 to 4,000 Hz (octave steps) presented at 55 dB HL were used to evoke steady-state responses in awake adults at MFs of 30 to 185 Hz. MFs of 60 Hz or lower resulted in response latencies (calculated from phase delay data) in the range of 28 to 33 ms, clearly similar to the range for auditory MLRs. For modulation frequencies at 90 Hz and above, the latencies ranged from 11.6 ms for a CF of 250 Hz to 8.9 ms for a CF at 4.0 kHz, indicating a likely homology to toneburst-evoked ABRs. In both waking and sleeping adults, for CFs at 1.0 kHz or lower, an MF of 45 Hz yielded larger ASSR SNRs; however, this SNR advantage for a 45-Hz MF was not obvious for sleeping subjects tested with CFs at 2.0 or 4.0 kHz. At CFs of 2.0 and 4.0 kHz, MFs of 80 Hz and above yielded SNRs that were equivalent to those at lower MFs in sleeping subjects. The study by Cohen et al. established the efficacy of recording ASSR in sleeping subjects, using high (>80 Hz) MFs, for CFs in the audiometric frequency range.

Dobie and Wilson (1998) also determined the detectability of ASSRs in adults tested both in the awake state and during sedated sleep. MFs of 40 and 90 Hz yielded peaks in the detection function for both awake and sedated sleep states for the low-frequency (640-Hz) CF presented at a moderate level, but less than 75% of the trials conducted at 38 dB SPL resulted in a detectable response, regardless of MF. MFs at 50 Hz or lower detectability were considerably reduced in the sedated sleep state compared to the awake state. The results obtained by Dobie and Wilson indicate that both low (40 to 50 Hz) and high (90 Hz) MFs are effective in evoking an ASSR for a CF below 1.0 kHz in awake or sleeping adults.

Aoyagi et al. (1994a) tested adults with normal hearing during natural sleep using MFs of 20 to 120 Hz, and CFs of 0.5, 1.0, 2.0, and 4.0 kHz all presented at 50 dB HL at MFs of 20 to 120 Hz. Results were similar to those of Cohen et al. (1991), with peaks in the detectability versus MF functions found at 40 and 80 Hz for CFs at 0.5 and 1.0 kHz and at 80 Hz or higher for CFs at 2.0 and 4.0 kHz. It should be noted that responses were detected at all MFs except for 20 Hz.

Lins et al. (1995) conducted a parametric study of ASSR using MFs of 67 to 111 Hz and CFs of 0.5, 1.0, and 2.0 kHz. Adult subjects were tested as they read or slept, but the effects of subject state were not evaluated as a variable. For a CF of 1.0 kHz presented at 60 dB SPL, MFs at 83 and 91 Hz yielded the largest ASSR amplitudes, significantly different from amplitudes measured at MFs of 71 and 111 Hz. Holding MF constant at 91 Hz, and level constant at 60 dB SPL, they showed no significant difference in ASSR amplitude for CFs varied at 5.0, 1.0, and 2.0 kHz. Increasing the level of a 1.0-kHz (CF) tone modulated at 91 Hz from 20 to 90 dB SPL resulted in a systematic increase in amplitude, and a decrease in phase, equivalent to a 1.3-ms decrease in latency.

In summary, for CFs of ≤1.0 kHz, at near threshold levels, there may be some advantage to using MFs at around 40 Hz in awake or sleeping adult subjects. MFs at 80 Hz or higher are suitable for CFs greater than 1.0 kHz.

Stimulus × Subject Interactions in Infants and Children

Findings of Levi et al. (1993) indicate that modulation frequencies above 40 Hz, and particularly at 80 Hz, are preferable for testing young infants with AM tones. Using AM tones at 500 and 2,000 Hz, presented at 60 dB HL (~78 dB SPL), they measured response coherence, an estimate of response power relative to overall response plus noise power, as a function of MF. The largest coherence values were obtained at 80 Hz, regardless of CF. When a 500-Hz CF was used, statistically significant responses were obtained only for MFs of 40, 50, and 80 Hz, but not at 10, 20, or 30 Hz. Using a 2,000-Hz CF, only the 80-Hz MF yielded statistically significant responses for infants. Aoyagi et al. (1994b) showed that MFs in the 80-Hz range resulted in the most stable and reliable ASSR results among normal hearing infants and children (aged 4 months to 15 years), tested while sedated. Although only one CF (1,000 Hz) was used, the MF was varied from 20 to 200 Hz. Measures of PC were highest for 80 Hz, although peaks were also found at 120 and 160 Hz for infants and children less than 4 years of age; these additional peaks in the coherence functions were not clear for older children, nor for a group of normal hearing adults. There was a clear advantage for the 80-Hz MF compared to the 40-Hz MF for all except children older than 9 years or for adults.

A large-scale study of newborns completed by Rickards et al. (1994) provides compelling evidence for the efficacy of modulation rates higher that 60 Hz for obtaining responses to tones with both AM (100%) and frequency modulation (20%). CFs of 500, 1,500, and 4,000 Hz (at 55 dB HL) were used to obtain ASSRs at MFs ranging from 35 to 185 Hz. As CF increased, so did the best MF for response detection. MFs in the range of 65 to 100 Hz yielded the best detection efficiencies in sleeping newborns. In addition, latencies calculated from the response phase were in the 11- to 14-ms range, with a systematic decrease in latency with increased frequency. Both the range and the type of latency change suggest that the ASSR recorded at high MFs in sleeping newborns are generated by the brainstem.

These studies indicate that MF should be varied with CF to get the largest amplitude responses. They further show that for CFs <1.0 kHz, ASSRs obtained with MFs of 30 to 50 Hz are larger than ASSRs at MFs >80 Hz. This may be because at lower MFs, the modulation envelope is of longer duration, allowing greater temporal summation, and thus, a larger response. It is also likely because of the fact that the ASSRs from the cortex, that is, at the lower MFs, are larger than those from the brainstem (at the higher MFs). The relationship between MF and ASSR amplitude is illustrated in Figure 15.6. Although Levi et al. (1993) were able to obtain ASSRs for low-frequency MF–CF combinations in very young infants, the ASSRs for the 0.5-Hz CF at 80-Hz MFs were more consistently present and of larger amplitude than were those obtained at lower MFs. Previous research in sleeping infants and young children using MFs at 40 Hz indicates that the ASSR is unstable (Stapells et al., 1988). This is not the case, however, for adults, in whom ASSRs for low (<1.0 kHz) CF and low (<50 Hz) MF stimuli are present during either sleep or wakefulness.

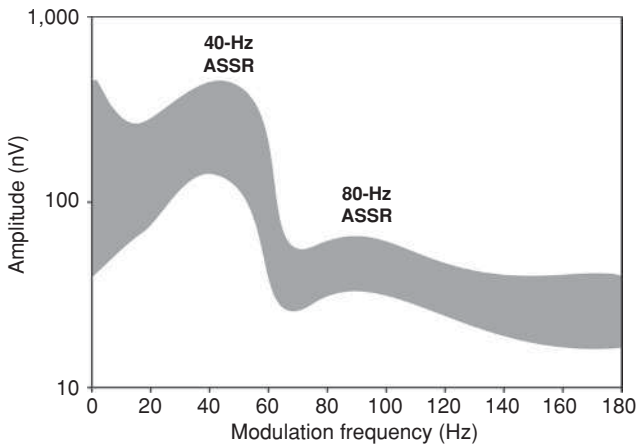


FIGURE 15.6 ASSR amplitude as a function of modulation frequency. Data are modeled from adults tested awake or asleep. ASSR amplitudes at 40 Hz are two to three times the amplitudes of ASSRs at 80 Hz. ASSR amplitudes for MFs <20 Hz are variable in wakefulness, and even more so during sleep.

Signal Processing and Acquisition Variables

Filtering

Filtering is a crucial tool that is used in recording any type of evoked response. Filtering can be in either analog (online) or digital (offline), or a combination of both. Filtering increases the SNR by removing unwanted activity at frequencies that are not of interest, and allowing focus on the frequencies at which the responses are located.

The choice of filter cutoff frequencies depends on the frequency of the intended recorded signal. If a filter's cutoff frequency is placed too close to the MF used to obtain the ASSR, then correction factors must be applied to compensate for the attenuation of the signal because of the filter. Common high-pass filter settings used in ASSR studies are 1 Hz up to 30 Hz, with low-pass filters of 300 Hz. As the major energy of the ASSR is at the MF, and the FFT acts as a narrow-band filter centered on the MF, the filter cutoff points are largely unimportant, unless there is a danger of saturating the bioamplifier.

Electrode Montage

ASSRs are readily recordable using electrode configurations similar to those used for the ABR. A number of factors contribute to the optimal placement of electrodes. Some of these include location of the ASSR generator and noise sources.

The magnitude of a scalp-recorded evoked response varies depending on the orientation of the equivalent dipole of the underlying generator. The 80-Hz ASSRs have a major component in the brainstem oriented vertically and therefore yield large responses when using centrally placed noninverting electrodes such as Cz, Fpz, or Fz, with inverting electrodes placed at the mastoid, inion, or nape of neck (C7). The 40-Hz ASSR has neural generators that are both at the brainstem and at the primary auditory cortex levels and therefore will also have large responses recorded from the midline. A study by Van der Reijden et al. (2004) evaluated the effect of electrode placement on ASSRs obtained from young infants. They recorded ASSRs from an array of 57 scalp electrodes and determined the montages that yielded the best ASSR-to-noise ratios. They showed a Cz-Mi (vertex-ipsilateral mastoid) montage resulted in the largest ASSR-to-noise ratios. The practical result is that using the Cz-Mi montage will result in time-savings during an ASSR evaluation, because criterion SNRs are reached more quickly. More recently, Van Dun et al. (2009) recorded ASSRs with six scalp electrodes and combined ASSRs across electrodes in various combinations. They showed that in quiet, "clean" EEG recordings, the addition of multiple recording electrodes does not provide an SNR advantage for the ASSR. However, in situations of higher EEG noise, as is typical in the clinic, a clear SNR advantage was obtained with "spatial averaging" across the Oz, P3, and Mi electrode sites, when Cz was used as reference.

Noise Sources

Every electrode placed on the scalp will have noise sources, either electrical or physiological. Electrical noise can most often be reduced by ensuring that the contact between electrode and skin has low impedance (under 5 kΩ). One source of physiological noise results from placing an electrode over a muscle. Tonic muscle activity is particularly problematic because it contains energy at frequencies (20 to 50 Hz) that are close to those of the ASSR. For example, a large amount of tonic neck muscle activity is present in an otherwise quiet subject sitting upright. If an electrode is placed at the nape of neck, the recording will be contaminated with this muscle noise. In such cases, placing head supports behind the subject's neck often relieves the strain on neck muscles ensuring less muscle activity and increased comfort for the subject.

Averaging

Just like every other evoked response, the ASSR becomes more easily detected through the process of averaging (see Chapter 11). By definition, an ASSR has a stable amplitude and phase. The ASSR detection algorithms (see the next section) are based primarily on the SNR; that is, the ASSR signal must be significantly larger than the noise for the ASSR to be detected. ASSRs near threshold have very low amplitudes, and averaging for a substantial time period is required so that the ASSR can be detected.

Another assumption of averaging, and of the ASSR detection algorithms, is that the background noise is stationary; however, biologic noise (coughing, sneezing, blinking, yawning, swallowing) is anything but. Invariably, when recording at near threshold levels, the subject transiently generates muscle noise (i.e., swallowing, or gross limb movements), creating a large "noise burst" that significantly alters the SNR, thus resulting in the detection algorithm indicating no response. A couple of options exist for such cases. One is to simply record for a longer period of time until the transient noise has been "averaged out." Another option is to use artifact rejection. Simply, if the voltage of an EEG sample exceeds a predetermined value (i.e., 80 mV), that sample is discarded. The disadvantage of artifact rejection is that the response is discarded along with the noise, so that longer test times are needed to obtain a result.

Weighted averaging (Elberling and Wahlgreen, 1985; John et al., 2001a) is another signal processing method used to reduce the effect of transient noise in evoked potential recordings. The general concept in weighted averaging is that noisy sections of the recording, that is, noisy EEG samples, contribute proportionally less to the overall average. Using online calculations, the "weight" of each sample can be determined by considering its variance. Noise will increase the variance of a sample. A weighted-averaging algorithm will assign the higher variance samples lesser weights, and the low-variance samples higher weights. Samples with large (noise) variance will contribute proportionally less to the overall average.

Detection Methods

One of the major reasons that ASSRs are gaining widespread use is the fact that "real-time" statistical methods may be used for response detection. It is only when an evoked response is detected using statistical methods that the technique is truly "objective."

Time- and Frequency-Domain Methods

Most modern techniques of ASSR detection involve transformation of the response from the time domain to the frequency domain. These transformations are usually accomplished using the Fourier transform. In the frequency domain, the ASSR can be represented as an addition of sinusoids each with its own frequency, amplitude, and phase.

The Fourier transform can be implemented in either analog or digital form. In analog form, the EEG sample is fed into a Fourier analyzer and is multiplied by the sine and cosine of the modulation frequency. After multiplication, the ASSR at the modulation frequency is observed as a sustained or DC output, whereas noise yields an oscillatory or AC output. The Fourier analyzer output is further low-pass filtered and yields values x (from the cosine multiplication) and y (from the sine multiplication). The ASSR amplitude, a, is calculated using the formula (Stapells et al., 1984)

$$a = (x^2 + y^2)^5$$

Response phase, θ, is calculated using the formula

$$\theta = \tan^{-1}(y/x)$$

The output of the Fourier transformation for a particular (modulation) frequency of interest is two-dimensional, with real and imaginary components in rectangular coordinates, or as a magnitude and phase in polar coordinates; that is, a Fourier coefficient is a complex number. These two dimensions may be graphed on a polar plot with response amplitude shown by the length of the vector and response phase (latency) as the angle of the vector in either radians (from 0 to 2π) or in degrees (from 0 to 360).

There are two general strategies for objective statistical analyses of the ASSR. One strategy involves repeated measures of ASSR phase and amplitude as obtained from the Fourier transformation. The other strategy evaluates the variability of the ASSR and adjacent noise amplitudes in the spectrum of the response.

Phase Coherence Measures

PC is related to the signal (response)-to-noise (background EEG and myogenic) ratio. The basic concept is that the phase delay of the response is measured relative to the MF. Each averaged response can be subjected to a fast Fourier transformation (FFT). For PC, the phase of the major peak at the MF frequency can be plotted in polar coordinates. The sine and cosine of the angles formed by each phase

vector (for each sample) are calculated.* The general idea for detecting ASSRs using PC is that measurements of phase (from the Fourier transformation) are taken for a number of EEG samples. If an ASSR is indeed present, then its phase will be consistent, phase-locked to the MF, across the samples. If sample phases are random, then the ASSR cannot be distinguished from background noise.

PC values vary from 0.0 to 1.0. When the sample phases are in phase with one another, there is high coherence and the values will be closer to 1.0. When the sample phases are random, there is low coherence (values close to 0), as would be found if the samples contained only noise, with no ASSR. The statistical significance of the resulting PC value can be determined. That is, the probability that the samples come from a distribution of phase values that are randomly distributed can be tested using a variety of statistics. Usually when a significance level of $p < 0.05$ is obtained, the null hypothesis (samples of phases and sample of noise are equal) is rejected, and the samples can be considered phase-locked or phase coherent, and an evoked response is deemed to be present. The amplitude, or length, of the phase vectors is not used in this statistical test. Very small amplitude responses that demonstrate high PC will be detected as easily as large-amplitude responses with the same degree of PC.

Dobie and Wilson (1989a, 1989b, 1993, 1995) have employed magnitude-squared coherence† (MSC) methods for detecting and defining the ASSRs. This method uses both the amplitude and phase information from the FFT. MSC (γ^2) estimates the power of the averaged response divided by the average power of the individual responses. γ^2 will vary from 0 (no response) to 1 (high SNR). Since the response consists of both signal (response) and noise, the MSC can be viewed as a signal plus noise-to-noise estimate. Theoretical distributions of MSC have been determined, so that it is possible to determine critical values to be used in the objective detection of an ASSR. When a critical value of MSC is exceeded by the EEG samples obtained in response to an AM tone, the null hypothesis (sample containing only noise) can be rejected and an ASSR has been detected. Another method for determining the significance of the ASSR phase and amplitude distribution employs the Hotellings T^2 test, which is similar to a *t*-test, except that it calculates significance in two dimensions (amplitude and phase). Victor and Mast (1991) introduced the T^2_{circ}, which assumed equal variances in both real and imaginary dimensions. This results in confidence limits with a circular shape. Mathematically, the T^2_{circ} and γ^2 are equivalent (Dobie and Wilson, 1993).

If amplitude information is ignored, that is, if all amplitude vectors are set to a value of 1, then $\text{MSC} = \text{PC}^2$, or $\text{PC} = (\text{MSC})^{1/2}$. The advantage of MSC is that amplitude increases in the evoked potential will serve to increase the MSC value obtained, and enhance detection, as compared to methods that measure phase alone. The disadvantage of MSC is that fluctuations in background noise will have a greater effect on the MSC value compared to PC or PC^2 (PCS).

Dobie and Wilson (1995) compared MSC at two alpha levels, 0.01 and 0.10, to human visual detection of the time-domain waveform for a 40-Hz auditory-evoked potential. The sensitivity and specificity of each method were determined, and a d' calculated. Values for d' were higher for MSC at both alpha levels, compared to human observers. Although thresholds were not estimated, it is clear from their data that the alpha level for MSC would have an effect on estimated threshold. In general, as the statistical criterion is relaxed, the estimated threshold decreases, but at the expense of decreased specificity (more false positives, or responses detected when there is no stimulus).

Spectral Measurements

The basis for spectral measurements of ASSR detection comes from performing an FFT on a grand averaged recording. The result is a frequency spectrum of the entire EEG. The peaks in the resulting spectrum, and the amplitude and phase of the spectral peak, can be measured. A steady-state response evokes activity at the MF, and, therefore, the stronger the signal the more power there is at the MF. In this measurement, the noise is defined as activity that is not at the MF. The significance of the signal is determined by comparing the power (voltage squared) of the signal to the power of the noise (a few Hertz above and below the MF). An *F*-test can then be calculated, where the numerator is the power of the signal and the denominator is the power of the noise.

Picton et al. (2003) have described the method of spectral analysis for ASSR detection:

> "The level of the background noise in a recording can be estimated by measuring the activity at frequencies in the spectrum other than that of the stimulus and response. Comparing the power of the signal to the powers at other frequencies is the basis of the F-test for hidden periodicity (Dobie and Wilson, 1996; Lins et al., 1996). The procedure calculates an F ratio of the power in the signal frequency bin (*s*) to the mean power in *N* adjacent bins:

$$F = N\left(x_s^2 + y_s^2\right) \Bigg/ \sum_{\substack{j=s-N/2 \\ j\neq s}}^{s+N/2} \left(x_j^2 + y_j^2\right)$$

> This is distributed as *F* with degrees of freedom 2 and 2 *N*. The F-test is essentially the same as the magnitude

*Phase coherence is

$$\text{PC} = [(1/n\Sigma\cos\phi_I)^2 + (1/n\Sigma\sin\phi_I)^2]^{1/2}$$

where n is the number of successive samples and ϕ is the phase of the Ith frequency component in the Fourier series.

†Magnitude-squared coherence, γ^2, is

$$\text{MSC} = ((1/n\Sigma A_I\phi_I)^2 + (1/n\Sigma\sin A_I\phi_I)^2)^{1/2}/(1/n\Sigma A_I^2)$$

where n is the number of subaverages and ϕ is the phase and A the amplitude of the Ith frequency component in a Fourier series.

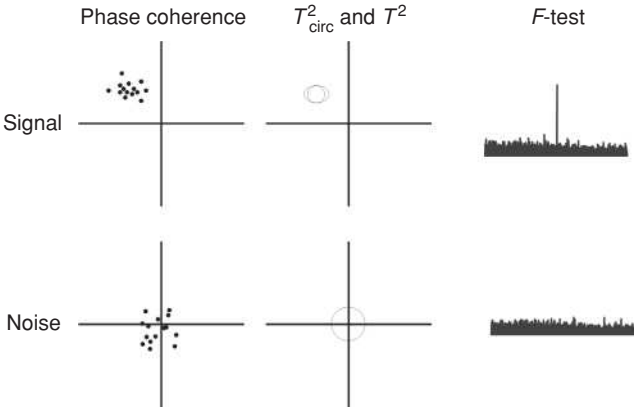


FIGURE 15.7 Examples of phase coherence, circular T^2-, and *F*-tests for a statistically significant response (*signal*) versus no response (*noise*). Each dot in the phase coherence quadrant represents the end-point of a phase vector, which emanates from the 0,0 point of the vertical and horizontal axes. Similarly, the position and size of the circle drawn on the T^2 quadrant represents the presence of a statistically significant response. A result containing only noise (no response) is indicated by a *circle* that is at or close to the crossing of the *X*- and *Y*-axes. For the *F*-test, the amplitude of the response spectrum peak at the MF will be significantly larger than the amplitude at adjacent frequencies.

squared coherence when the number of individual measurements for calculating the coherence equals one less than the number of adjacent points used in the F-test, i.e., the degrees of freedom are the same (Dobie and Wilson, 1996).

The F-test has several advantages over tests based on repeated measurements of the response. First, the number of adjacent frequency-bins to which the signal response is compared can be increased beyond any easily obtained number of separate measurements of the signal response. Second, the technique can easily be adapted to omit certain frequency bins from the calculation. In this way a noise estimate can be obtained that is uncontaminated by line noise or by responses at other frequencies (if one is recording responses to multiple simultaneous stimuli.)"

Figure 15.7 shows the results for PC, circular *t*-tests, and *F*-tests for trials in which responses are present compared to those in which only noise is present.

CALIBRATION

Calibration of modulated tones is straightforward. The common practice is to measure the SPL of the modulated tone in the same way as for a puretone. Commercially available test instruments allow the user to select levels using dB SPL or dB HL levels. In the latter case, the 0 dB HL at each frequency would have the same SPL as a puretone at 0 dB HL, for example, using published audiometer standards such as ANSI S3.6-2004 American National Standard Specification for Audiometers, or equivalent international standards (ISO-389-2: 1994; ISO-389-1:1998). For example, using ANSI 3.6-2004, the SPL of a 2.0 kHz at 0 dB HL is 2.5 dB when presented through insert phones (calibrated in an H1 A coupler).

It must be recognized that the power of a modulated tone is less than that for a puretone of the same peak amplitude. The long-term average power of a sinusoidally amplitude-modulated (SAM) waveform is $(1+m^2/2)I_0$, where I_0 is the average power when *m*, the modulation index, = 0 (Viemeister, 1979). The relative increment in power, $\Delta I/I_0$, for a modulated tone is $m^2/2$. Viemeister's classic study of temporal modulation transfer functions (1979) addressed the issue of modulation detection thresholds by measuring modulation thresholds for power-compensated wide-band noise $[(1+m\sin\omega_m t/(1+m^2/2)^{1/2}]$ compared to those obtained with no compensation $[(1+m\sin\omega_m t)]$. At modulation frequencies below 560 Hz, there was no effect of power compensation on modulation thresholds. Although the threshold of modulation detection is different from the threshold for a stimulus with modulation, it is worth keeping in mind that the power of modulated versus unmodulated signals is different, and that there will be a discrepancy of around 2 to 3 dB when thresholds for 100% AM tones are compared to those for unmodulated tones. These differences are not compensated for when using dB HL calibration.

CLINICAL APPLICATIONS OF ASSR

The primary application of ASSR in audiology is for hearing threshold estimation in those at risk for hearing loss, that is, the prediction of the audiogram. To this end, a number of studies have compared ASSR thresholds and behavioral thresholds in infants, children, and adults with hearing loss and published regression formulae that relate ASSR threshold to puretone threshold. Alternatively, the difference between ASSR and puretone thresholds has been calculated, and these "correction" factors have been used to interpret the ASSR thresholds. In addition, ASSR thresholds to bone-conducted stimuli can be used to determine the presence and extent of a conductive impairment. The determination of a sensory versus neural hearing loss, another element of site-of-lesion evaluation, is possible, under some circumstances. ASSRs have also been employed in hearing aid and cochlear implant (CI) evaluations. Experiments with ASSR tests employing complex and dynamic suprathreshold stimuli hold some promise for estimating psychophysical and speech perception abilities. These applications are reviewed below.

Audiogram Prediction/Hearing Threshold Estimation

A driving concept in ASSR research is the objective determination of the puretone audiogram. The goal of objectivity

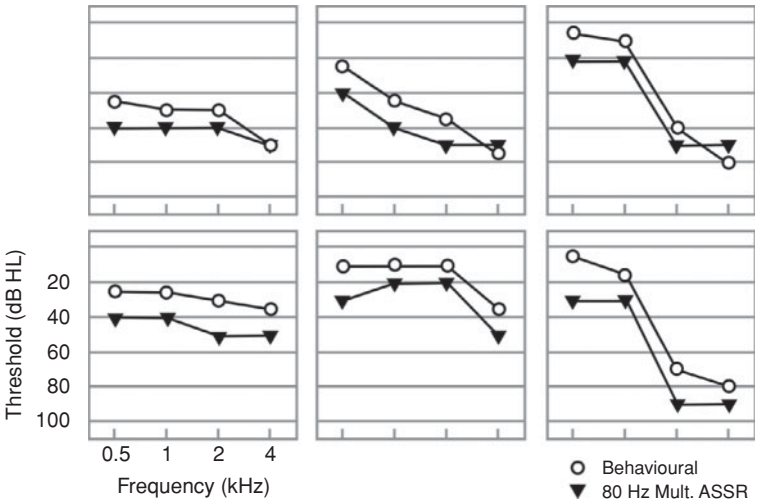


FIGURE 15.8 ASSR results from Van Maanen and Stapells (2005) showing a variety of audiometric configuration can be estimated using ASSRs. *White circles* represent behavioral thresholds whereas *black triangles* are ASSR thresholds derived using multiple ASSRs.

should be met in three ways. First, the threshold determination is based on a physiological response, not on a subjective perception of the subject. Second, the presence of a response is determined by the use of statistical tests, or "objective" detection algorithms. Third, the interpretation of the results is also bound by objective methods and decision-making rules.

Figure 15.8 illustrates how different audiometric configurations can be predicted using ASSRs (data adapted from Van Maanen and Stapells, 2005).

A comprehensive study of hearing threshold prediction using ASSR, in a sample that included hearing impaired children and adults, was reported by Rance et al. (1995). Participants had hearing losses that were moderate or worse, including some with profound hearing loss. These hearing losses were purely sensory/neural; those with conductive loss were specifically excluded from the study. ASSR threshold estimates were made using CFs at 250, 500, 1,000, 2,000, and 4,000 Hz, all presented at an MF of 90 Hz. Pearson product–moment correlations between puretone and ASSR threshold were at 0.96 for 250 Hz and as high as 0.99 for 2,000 and 4,000 Hz. Regression formulae were developed, to predict behavioral thresholds from ASSR thresholds. Table 15.2 shows these formulae for each CF. The *Y*-intercepts demonstrate that subjects with normal hearing (e.g., 10 dB HL) have ASSR thresholds elevated by as much as 40 dB with respect to puretone threshold in the low frequencies, whereas in mid and high frequencies, ASSR thresholds are closer to puretone thresholds. This is similar to reports of ABR thresholds to toneburst stimuli in normal hearing subjects (Stapells, 2000). As hearing loss increases and as CF increases, there is less discrepancy between behavioral and ASSR threshold, shown by an analysis of the standard deviations of the regressions by degree of hearing loss and by CF (Rance et al., 1995). Errors in prediction of behavioral thresholds from ASSR thresholds show standard deviations ranging from 3.6 dB for severe-profound losses at 2 kHz, to 11.9 dB for mild-moderate losses at 250 Hz. These findings are very similar both qualitatively and quantitatively to the findings of Stapells et al. (1995), who developed regression formulae relating toneburst ABR threshold to puretone threshold in infants and young children.

In a related study, the Melbourne group (Rance et al., 1998) demonstrated the advantages of using ASSRs to determine residual hearing thresholds for those infants and children from whom ABRs could not be evoked (at 100 dB nHL) using click stimuli. Again, ASSRs were obtained using CFs of 250 to 4,000 Hz with an MF of 90 Hz. In a sample of 109 children, whose hearing losses ranged from moderate to profound, the average discrepancy between ASSR and behavioral thresholds was only 3 to 6 dB (although the standard deviations were 6 to 8 dB), with larger discrepancies and standard deviations found at 250 and 500 Hz, as in the previous study. ASSR thresholds were within 20 dB of puretone threshold for 99% of the comparisons and less than or equal to 10 dB for 82% of the comparisons. The findings demonstrated the efficacy of ASSRs for estimating the audiogram in infants and children who can benefit from the amplification of their residual hearing.

More recently, Rance et al. (2005) published a large series of ASSR and behavioral thresholds in infants. Clinical findings from seven audiology centers within the state of Victoria, Australia, were pooled. All centers used the GSI-Audera (GSI-Nicolet) or its predecessor, the ERA System (ERA Systems), in the collection of ASSR thresholds. Only those infants who had ASSR thresholds measured at ≤3 months of age, who subsequently yielded reliable conditioned behavioral audiometric thresholds, and who had evidence of normal middle-ear function at the time of ASSR and behavioral tests were included. This sample was composed of 575 infants (1,091 ears), whose ASSR thresholds were obtained at a mean age of 2.6 months and behavioral thresholds obtained at a mean age of 9.8 months. There were 285 (of 575) infants who demonstrated normal hearing

TABLE 15.2

Regression Formulae Relating ASSR Threshold to Behavioral Threshold, Where *x* = ASSR Threshold

Carrier Frequency	500 Hz	1,000 Hz	2,000 Hz	4,000 Hz
Cone-Wesson et al. (2002a)	1.39*x* – 35	1.24*x* – 15	0.94*x* + 5	1.34*x* – 24
Dimitrijevic et al. (2002)	0.88*x* – 9	0.92*x* – 1	0.89*x* – 0	0.99*x* – 8
Herdman and Stapells (2003)	0.77*x* – 6	0.91*x* – 4	0.92*x* – 6	1.04*x* – 5
Rance et al. (1995)	1.30*x* – 40	1.18*x* – 26	1.05*x* – 19	1.19*x* – 24
Rance and Rickards (2002)	1.39*x* – 49	1.35*x* – 44	1.28*x* – 36	1.31*x* – 39
Rance et al. (2005)	1.37*x* – 45	1.33*x* – 40	1.23*x* – 28	1.32*x* – 37
Van der Werff and Brown (2005)	1.12*x* – 23	1.03*x* – 13	1.11*x* – 14	1.11*x* – 13

Regression formulae: (1) Cone-Wesson et al. (2002a) based on a sample of 51 infants, mean age 16 months, 16 with near normal or mild loss, 18 with moderate loss, and 17 with severe-profound loss. Whereas 31 were SNHL, 10 were conductive and the remainder had normal hearing or mixed-type loss. (2) Dimitrijevic et al. (2002) based on a sample of 45 adults, 31 with hearing loss and 14 with normal hearing. In the hearing loss group, there were 17 ears with mild, 19 with moderate, and 16 with severe loss. (3) Herdman and Stapells (2003) based on a sample of 27 adults with SNHL ranging from mild to severe. (4) Rance et al. (1995) based on a sample of 25 children with moderate to profound SNHL and 35 adults with hearing ranging from normal to profound SNHL. (5) Rance and Rickards (2002) based on a sample of 211 infants with a mean age of 3.2 months at the time of ASSR and a mean age of 7.9 months at the time of behavioral hearing tests. Infants with evidence of conductive or progressive losses were excluded. (6) Rance et al. (2005) based on 575 infants tested at a mean age of 2.6 months, with behavioral tests completed at a mean age of 9.8 months. Whereas 285 infants had normal hearing thresholds (by behavioral tests) 271 had SNHL. (7) Van der Werff and Brown (2005) based on 30 subjects, 10 with normal hearing, 10 with sloping hearing losses, and 10 with flat hearing losses.

(thresholds ≤15 dB HL). The mean ASSR thresholds in this group were 32.3, 32.5, 23.3, and 28.1 dB HL for CFs at 0.5, 1.0, 2.0, and 4.0 kHz, respectively, with standard deviations ranging from 6.3 dB (2.0 kHz) to 7.5 dB (0.5 and 4.0 kHz).

For the infants with sensory/neural hearing loss (N = 271), regression formulae were developed to relate the ASSR and behavioral thresholds. These are shown in Table 15.2 and are similar to those published by Rance et al. (1995) and Rance and Rickards (2002). In general, the slope of the regression function indicates that as hearing loss increases in severity, and CF increases in frequency, there is a closer correspondence of ASSR and behavioral threshold.

Several studies have demonstrated that ASSR thresholds have a strong relationship with puretone thresholds in adults with well-defined hearing losses. Dimitrijevic et al. (2002) tested 59 ears of 31 adults with primarily sensory/neural hearing impairments ranging in severity from mild to severe, with nearly equal representation among mild, moderate, and severe degrees of loss. The ASSR thresholds showed a high correlation with the puretone thresholds, with r = 0.92 for carriers in the range of 500 to 4,000 Hz. The differences between ASSR and puretone threshold ranged from 13 ± 11 dB at 0.5 kHz and 5 to 8 dB ± 8 to 11 dB for carriers at 1.0 to 4.0 kHz. Herdman and Stapells (2003) tested 31 male adults with sensory/neural hearing losses, some with very steep configurations, and demonstrated that the ASSR versus puretone threshold differences were, on average, 14, 8, 10, and 3 dB for CFs at 0.5, 1.0, 2.0, and 4.0 kHz, respectively. Van der Werff and Brown (2005) and Picton et al. (2005) obtained ASSRs from adults with normal hearing and those with sensory/neural hearing loss. Both studies showed that the difference between ASSR and puretone threshold was smaller in those with sensory/neural hearing loss than in the normal hearing subjects. This is in agreement with Rance et al. (1995). Furthermore, the amplitude-growth functions were steeper in those with sensory/neural hearing loss, indicating a physiological recruitment-like phenomenon (Picton et al., 2005). Van der Werff and Brown (2005) showed ASSR–puretone differences in the range of 8 to 18 dB HL at 0.5 and 1.0 kHz, but only 6 to 8 dB HL at 2.0 and 4.0 kHz, among adults with sensory/neural hearing losses. On the other hand, Picton et al. (2005) obtained ASSR–puretone differences of less than 5 dB HL in their group of elderly adults with sensory/neural hearing loss. One reason for these differences may be that Van der Werff and Brown (2005) averaged ASSRs for about 4 minutes, whereas Picton et al. (2005) averaged for greater than 9 minutes, thus allowing resolution of responses with smaller SNRs.

Regardless of stimulus procedure, whether it be single frequency (Rance et al., 1995) or multiple frequencies (Dimitrijevic et al., 2002; Herdman and Stapells, 2003; Picton et al., 2005; Van der Werff and Brown, 2005) or averaging time (as short as 90 seconds in Rance et al., 1995, as long as 9 minutes in Picton et al., 2005), it is clear that the ASSR provides a clinically useful estimate of puretone thresholds, even in sloping audiometric configurations. The largest discrepancies between ASSR and puretone threshold are obtained in those with normal cochlear function (i.e., normal or conductive hearing loss), which are on

the order of 25 to 40 dB for low-frequency carriers and 10 to 20 dB for mid- and high-frequency carriers. The size of the ASSR–puretone difference is not really the issue, as this can be accounted for with a correction factor; it is the variability that is troublesome. For example, even if the ASSR–puretone difference was 45 dB at every frequency, if there was little variability (±5 dB), puretone threshold would be easy to estimate. What is observed, however, is that both the ASSR–puretone difference and the variability are dependent on CF and degree of hearing loss. As the CF and degree of hearing loss increase, the ASSR–puretone difference and the variability decrease. That is, predictive error decreases with increases in severity of hearing loss and CF. Both subtractive and regression formulae methods of estimating puretone thresholds from ASSR thresholds can take this into account. Table 15.3, after Picton et al. (2003), updated with studies published since 2003, summarizes 80-Hz ASSR threshold data from multiple clinical laboratories.

Comparison of ASSR with ABR

Because a primary application of ASSR is in the estimation of threshold, it is appropriate to compare the results obtained from ASSR to those obtained from ABR tests using tonebursts. There are, however, a number of differences in methodology that could lead to fairly substantial differences, including stimulus spectrum, modulation envelope/toneburst shape, rate, and response detection methods.

Aoyagi et al. (1999) directly compared toneburst-evoked ABR threshold estimates to ASSR threshold estimates, primarily in hearing impaired children tested during sedated sleep. They used a 1,000-Hz tone modulated at 80 Hz to evoke the ASSR and a 1,000-Hz toneburst (2 ms rise/fall time, 1 ms plateau) with a 53-ms interstimulus interval to evoke an ABR. Puretone thresholds ranged from 10 to 110 dB HL in the group of children tested with ABR and ASSR. The correlation of puretone threshold (in dB HL) with ABR threshold (in dB nHL) was 0.83, whereas for ASSR the correlation with behavioral threshold was 0.86. The difference in correlation coefficients was not statistically significant. The mean difference between behavioral and ASSR threshold was 3.8 dB (12.9 s.d.) and for ABR the difference was 6.8 dB (14.1 s.d.). It is not known if the dB nHL reference was the same for the AM tone and the toneburst.

TABLE 15.3

ASSR Threshold–Behavioral Hearing Threshold Difference, in Adults, Children, and Infants with Hearing Loss

Study	Subjects	Stimulus	500 Hz	1,000 Hz	2,000 Hz	4,000 Hz
Van der Werff and Brown (2005) (flat loss)	10	MM	11 ± 5	8 ± 4	7 ± 5	6 ± 5
Van der Werff and Brown (2005) (sloping loss)	10	MM	18 ± 8	10 ± 7	8 ± 6	5 ± 4
Picton et al. (2005)	10	MM	11 ± 18	–4 ± 9	2.5 ± 11	5 ± 12
Luts and Wouters (2005) ("Master")	10	MM	17 ± 12	12 ± 8	17 ± 8	19 ± 12
Luts and Wouters (2005) ("Audera")	10	MM	20 ± 8	14 ± 7	13 ± 7	14 ± 13
Herdman and Stapells (2003)	29	AM	14 ± 13	8 ± 9	10 ± 10	3 ± 10
Dimitrijevic et al. (2002)	31	MM	13 ± 11	5 ± 8	5 ± 9	8 ± 11
Rance and Briggs (2002)	184	MM	6 ± 9	6 ± 7	4 ± 8	3 ± 11
Van Maanen and Stapells (2005)	23	MM	17 ± 11	15 ± 7	19 ± 9	4 ± 10
D'haenens et al. (2009)	21, 18, 13, 4 (mild)	MM	14 ± 11	13 ± 8	14 ± 7	13 ± 6
	11, 15, 20, 27 (mild)	MM	14 ± 7	10 ± 10	9 ± 6	11 ± 9
Lin et al. (2009)	142	AM	17 ± 14	15 ± 9	14 ± 8	11 ± 87
Ishida et al. (2011)[a]	13 (flat)	MM	6 ± 12	4 ± 13	3 ± 13	2 ± 11
	10 (gradual slope)	MM	18 ± 8	7 ± 10	4 ± 13	0 ± 9
	15 (steep slope)	MM	25 ± 13	16 ± 11	11 ± 12	2 ± 10

The number of subjects and stimulus type are given for each study. AM is a sinusoidally modulated puretone and MM is an amplitude- and frequency-modulated tone. Rance and Briggs (2002) is the only study in which ASSR – PT differences were published for infants and children. All other studies report differences in adults. All studies used multiple carrier frequencies presented simultaneously, except Rance and Briggs (2002) and Luts and Wouters (2004) "Audera," in which single-frequency, sequential testing was used.

[a]Bone conduction.

Cone-Wesson et al. (2002a) performed a direct comparison of toneburst-evoked ABR and ASSR threshold measures in a group of 10 normal hearing adult subjects. ABRs were evoked using 0.5- and 4-kHz tonebursts, with (Blackman window) onset and offset ramps of two cycles, and a one cycle plateau, presented with an interstimulus interval of 40 ms. ASSRs were evoked using CFs of 0.5 and 4 kHz, tested at 41 Hz, and also at 74 Hz for the 0.5-kHz CF and at 95 Hz for the 4-kHz CF. Sampling for each ABR trial proceeded until an Fsp criterion of 3.1 ($p < 0.01$) was met, or 6,000 artifact-free samples were obtained. Visual detection by an expert observer was used as an additional measure of ABR presence. For each ASSR trial 64 samples of 1.486 seconds duration were obtained and subjected to PC analysis using a filter centered at the MF. A response was considered present if the PC statistic reached a criterion of $p < 0.01$. Thresholds for the 46-Hz ASSR and toneburst ABR thresholds were not statistically different. Thresholds for the 74-Hz MF–0.5-kHz CF were elevated with respect to the ABR threshold for a 0.5-kHz toneburst (and 46-Hz ASSR), but thresholds for the 95-Hz MF–4-kHz toneburst were 15 dB better than those for the 4.0-kHz toneburst ABR.

This study is the only one that attempted to compare threshold estimates using both ABR and ASSR methods and also employed similar statistical criteria for judging a response to be present. There is no difference in evoked potential threshold (expressed as dB SL) when adults are tested with tonebursts (for ABR) or CFs modulated at 41 Hz (for ASSRs), and when both ABR and ASSR are detected using an appropriate statistical technique. These results are in agreement with Cohen et al. (1991) and Dobie and Wilson (1998), who show that a low MF (in this case 41 Hz) is generally advantageous testing *adults* at low CFs (1,000 Hz or lower) whereas the higher MFs (above 60 Hz) are generally better for high CF tones. In normal hearing adults, furthermore, ABRs and ASSRs can generally be detected within 20 dB of behavioral threshold.

Van der Werff et al. (2002) obtained click and toneburst ABR and ASSR thresholds from 32 infants and young children, all tested during sedated sleep. All the participants in the study were being evaluated as candidates for CIs, and so they were known to have significant hearing losses. They found a 0.97 correlation between click ABR thresholds and those found for ASSRs at 2.0 and 4.0 kHz. The correlation between the 500-Hz toneburst ABR and the 500-Hz ASSR thresholds was statistically significant, but lower (0.86) than the correlations between click ABR and high-frequency (2- and 4-kHz) ASSR. In 33% of cases, when the toneburst ABR was absent for stimuli presented at the highest stimulus level available, an ASSR was present, albeit at elevated levels consistent with a moderately severe or greater hearing loss. Also, 58% of ears with absent click-evoked ABRs had an ASSR response. This result replicates the findings of Rance et al. (1998) that ASSR thresholds may reveal some residual hearing when click or toneburst ABRs are absent. This property has helped to establish ASSR as a valuable test for infants and young children undergoing evaluation for cochlear implantation.

Johnson and Brown (2005) measured ASSRs and toneburst-evoked ABRs at 1.0, 1.5, and 2.0 kHz in adults with normal hearing and those with flat or sloping sensory/neural hearing losses. ASSR thresholds were determined as the lowest level at which a statistically significant response was obtained, whereas ABR thresholds were determined by visual inspection of the time-domain waveforms. Overall, ABR thresholds were "closer" to behavioral thresholds than ASSRs, except in the case of steeply sloping sensory/neural hearing losses, for which ASSRs were better estimates of threshold. Both ASSR and ABR provided accurate estimates of threshold among those with sensory/neural hearing loss.

In summary, the experience of those who use the ASSR technique for predicting audiometric threshold in hearing impaired infants and children appears to be comparable to that of those who use toneburst-evoked ABRs. Comparisons between the two techniques can be made to help formulate the most efficient and sensitive methods for this purpose. Each technique has particular strengths and limitations. An advantageous feature of the ASSR technique is that objective detection algorithms rather than visual detection methods are always used to determine the presence or absence of a response. This is a particular advantage for techniques claiming to be "objective" measures.

It is difficult to determine whether or not the ASSR is detected at lower SPLs than a toneburst-evoked ABR response at the same center frequency, owing to differences in stimulus calibration and response detection method. In normal hearing subjects, visual detection of the toneburst-evoked ABRs yielded lower thresholds, although when both toneburst ABR and ASSR were detected with automatic detection algorithms, the thresholds were equal (Cone-Wesson et al., 2002a).

40-Hz ASSR Threshold Tests

The bulk of the literature concerning audiometric applications of the ASSR employs modulation frequencies of 80 Hz or higher. When it was shown that ASSR responses to 40 Hz were unstable or absent in sleeping infants and children, much of the interest in its audiometric applications diminished. Thus, there is limited data on the use of the 40-Hz ASSR for puretone threshold estimation. Aoyagi et al. (1993) showed that 40-Hz ASSR was present at 11 to 18 dB above puretone threshold in normal hearing adults and at 8 to 13 dB for adults with hearing loss. There is reason to believe that the 40-Hz ASSR threshold estimates would be as good, if not better, than those for the 80-Hz ASSR, but this has not been carefully determined. The 40-Hz ASSR has a larger amplitude than the 80-Hz ASSR; however, background EEG and other biologic noise are larger in that

TABLE 15.4

40-Hz ASSR Threshold–Puretone Threshold Difference (Adults)

Study	Subjects	Stimulus	500 Hz	1,000 Hz	2,000 Hz	4,000 Hz
Klein (1983)	30 N	TB	16 ± 10	14 ± 7	16 ± 7	19 ± 7
Szyfter et al. (1984)	31 N	TB	15 ± 9	13 ± 7		
Dauman et al. (1984)	30 H	TB	11 ± 10	9 ± 10		
Lynn et al. (1984)	40 H	TB	–2 ± 12	8 ± 11		
Sammeth and Barry (1985)	16 N	TB	9 ± 7	10 ± 10	9 ± 5	16 ± 7
Kankkunen and Rosenhall (1985)	20 M	TB	8 ± 11	5 ± 9	4 ± 7	3 ± 8
Rodriguez et al. (1986)	15 N	TB	3 ± 5		20 ± 5	
	10 H	TB	4 ± 10		5 ± 10	
Stapells et al. (1987)	6 M	TB	1 ± 3		2 ± 4	
Milford and Birchall (1989)	22 H	TB		27 ± 10	23 ± 15	16 ± 15
Chambers and Meyer (1993)	10 M	AM	1 ± 5	2 ± 5		
Aoyagi et al. (1993)	15 N	AM	11 ± 10	11 ± 11	13 ± 10	18 ± 12
	18 H	AM	8 ± 7	9 ± 6	13 ± 8	12 ± 6
Van Maanen and Stapells (2005)	23 M	MM	14 ± 7	11 ± 6	12 ± 6	0 ± 9
Tomlin et al. (2006)	36 N	MM	17 ± 10			42 ± 14
	Mild/moderate $27_{500\ Hz}/20_{4,000\ Hz}$	MM	10 ± 9			24 ± 8
	Severe/profound $3_{500\ Hz}/10_{4,000\ Hz}$	MM	10 ± 13			22 ± 19
Ozdek et al. (2010)	23 N	MM	15 ± 12	10 ± 7	14 ± 8	15 ± 9
	38 H	MM	8 ± 6	9 ± 6	9 ± 7	14 ± 10

The number of subjects and their hearing status, and stimulus type are given for each study. For stimulus type, TB is the toneburst, AM is the sinusoidally modulated puretone, and MM is the amplitude- and frequency-modulated tone. For hearing status of subject group, N is normal hearing, H means subjects had hearing loss, and M indicates the group tested had members with hearing loss and normal hearing.

frequency region as well, so that achieving a criterion SNR may require as much averaging as for a smaller amplitude response. Table 15.4 summarizes 40-Hz ASSR threshold as a function of CF in adults.

A study by Van Maanen and Stapells (2005) performed the first comparison of 40-Hz ASSRs, 80-Hz ASSRs, and the slow cortical potential (N1/P2) in adults with normal hearing and sensory/neural hearing loss. Their results demonstrated that multiple 40-Hz ASSRs showed the smallest difference between physiological and behavioral thresholds compared to the other two measures. Moreover, the recording time for the 40-Hz ASSRs and the slow cortical potential was less than for the 80-Hz ASSRs. The authors concluded that the method of choice for estimating threshold in adults was 40-Hz ASSRs.

A growing trend in ASSR research, although not for threshold testing, has been to evaluate brain function using rates below 40 Hz. The general trend from the early work (Picton and Skinner, 1987; Rees et al., 1986) has demonstrated that ASSRs with AM rates from 2 to 35 Hz could be reliably recorded; however, the recordings had high levels of background EEG noise. Also noted was that the lower modulation rates had responses at the modulation rate harmonics. This effect was recently explored by Tlumak et al. (2012) in both children and adults. ASSRs were elicited by tone bursts ranging from 0.75 Hz all the way up to 80 Hz. Children showed larger responses at harmonics of the modulation rate than at the primary modulation frequency. This effect was most prominent at low modulation rates. It is possible that these harmonics represent the auditory system responding to the modulation change in both directions, in effect responding twice for one cycle of the stimulus. For example, with a low AM rate of 10 Hz, the rise time of the stimulus would be 50 ms (half modulation cycle) and 50 ms fall time (the second half of the modulation cycle), 50 ms being likely enough time for the auditory system to respond before the next half cycle of modulation.

Tlumak et al. (2007) performed a meta-analysis of the literature to test six assumptions about threshold estimation with ASSR. The first was that threshold differences between ASSR and behavioral thresholds decrease with the degree of hearing loss and CF. Second, that threshold estimates are expected to be better with MM stimuli compared to SAM tones. Third, that longer test duration or a greater number of sweeps are associated with lower threshold estimates because of the improvement in the response to noise ratio. Fourth, that there are no differences in the accuracy of threshold estimation for monaural compared to binaural stimulation.

Fifth, that there are no differences in threshold estimation owing to electrode montage; and sixth, that threshold differences for 80-Hz versus 40-Hz ASSR are similar to those found for ABR versus MLR. Meta-analyses were performed for studies that had participants with normal hearing, those with hearing loss, and then by combining the two samples. Assumptions that held true for the group with hearing loss were not always the same for the normal hearing group. In the normal hearing population, the threshold differences between ASSR and behavioral measures were smallest at 1.0 and 2.0 kHz, differences in threshold for MM versus SAM were significant at 1.0 kHz only, and there were no differences for the number of sweeps required to reach threshold for MM versus SAM tones. There was no difference in threshold for stimuli delivered monaurally versus binaurally nor did electrode montage have an effect. The analogy with ABR and MLR was confirmed for both normal hearing and hearing impaired groups. For the hearing impaired group, both modulation type and number of sweeps had significant effects on threshold, as did monaural versus binaural stimulation. As in the normal hearing group, there were no differences in threshold owing to electrode montage for the hearing impaired group.

Bone Conduction

The mainstay of audiometry is the determination of puretone air- and bone-conduction thresholds, for the purpose of determining whether a conductive component exists and its severity. In general, an air–bone gap of >10 dB is considered indicative of a conductive hearing loss. There are a number of studies that have explored the techniques for and the results of ABR BC threshold tests (for review, see Cone-Wesson, 1995). The results of these studies are relevant to the problem of estimating BC threshold with ASSR, for which there are fewer published results. In general, there are two methods for obtaining BC thresholds. First, the stimuli used for the AC test are presented through a bone vibrator, and the difference in threshold for AC and BC test conditions is measured. The second method is to present masking noise by a bone vibrator and to determine the noise level needed to mask the response to an AC stimulus. This is known as the "sensory/neural acuity level" (SAL) technique. The level of effective BC noise needed to mask the response to the AC signal is used as the BC threshold (Ysunza and Cone-Wesson, 1987). The air–bone gap is calculated as the difference between the AC threshold and the BC effective masking level. Both techniques require careful calibration. The first requires determination of psychophysical threshold for the stimuli used for the BC test. The SAL technique requires physiological calibration of the BC noise masker for a panel of normal hearing listeners. Both conventional BC thresholds (Dimitrijevic et al., 2002; Lins et al., 1996; Small and Stapells, 2005) and the SAL technique (Cone-Wesson et al., 2002c) have been used to estimate air–bone gaps from ASSR threshold tests. The advantage of the first "direct" method is that the procedure mimics that which is typically done during behavioral testing, and so has the comfort of face validity. A disadvantage of obtaining ASSRs to BC stimuli is that the electromechanical artifact of the BC stimulus is "steady state" and is present during the entire recording and so can obscure the neural response or, worse, cause the detection algorithm to return a "false positive," that is, an artifactual response (Small and Stapells, 2004). An artifactual response may arise if the sampling rate of the signal is a harmonic of the CF. There are two methods for reducing or eliminating this artifact: (1) Change the digital-to-analog conversion rate so that it is not a harmonic of the CF or (2) use a steep antialiasing (low-pass) filter (Picton and John, 2004). Some commercially available instrumentation may not allow these procedures, in which case the chance of artifact during BC testing is very high.

The advantage of the SAL method is that the stimulus for both the AC and BC threshold tests is the same, that is, the AC stimulus. There is no difference in transducer. Also, the artifact produced by the BC oscillator is noise, and so should not be mistaken for the response. The level of the artifact would be expected to diminish with averaging. The disadvantage is that the effective masking levels must be measured physiologically, not psychophysically. Table 15.1 summarizes the studies which established ASSR threshold for BC signals. The real conundrum in BC ASSR (or ABR) tests is not really the stimulus or masker, but the fact that the skulls of infants less than 1 year of age transduce BC stimuli much differently than in adults. Studies (Cone-Wesson and Ramirez, 1997; Yang et al., 1987) have shown that the immature skull appears to "focus" the BC signal at the temporal bone, leading to higher effective stimulus levels than in an adult. Thus, infants exhibit very low ABR thresholds compared to adults, and air–bone gaps exceeding 10 to 15 dB are not uncommon. Furthermore, there are only case reports of how ASSRs for air- and bone-conducted stimuli may be used to detect hearing loss because of middle-ear pathology, and there are no controlled cohort studies demonstrating the clinical efficacy. There are only two studies (Hulecki and Small, 2011; Casey and Small, 2014) that undertook measurement of psychophysical air- and bone-conduction puretone thresholds in infants under the age of 1 year and compared them to ASSR threshold estimates. The range of behavioral versus ASSR threshold estimates was very large (−10 to 30 dB) limiting clinical applicability until the sources of variability can be determined and controlled.

Hearing Aid Fitting and Cochlear Implant Mapping

There is considerable interest in using "objective" measures to fit and demonstrate benefit from amplification, particularly in preverbal infants and toddlers. It is possible to

measure ASSR thresholds in the unaided and then aided conditions (Dimitrijevic et al., 2004; Picton et al., 1998) and demonstrate functional gain. One advantage of using ASSRs for this purpose, compared to ABR, is that the AM (or MM) tones appear to be transduced by hearing aid microphones and circuitry more accurately than are click or toneburst stimuli, at least when used in a linear mode. It is still necessary, however, to measure the fidelity of this transduction and to calibrate the sound field, before making this type of measurement. At the present time, measures of functional gain, using either behavioral or electrophysiological measures, are not recommended (Scollie and Seewald, 2002). Rather, the careful determination of threshold as a function of frequency, and verification of target gains (based on the threshold data) using in situ electroacoustic measures, is preferred. The ASSR test, then, has a role in hearing aid fitting, by providing an accurate estimate of threshold on which hearing aid targets can be based. Yet, to quote Picton et al. (2003):

> …demonstrating that the hearing aid is causing sounds to activate responses in the brain at intensities where there was no response without the aid is an important confirmation of the benefit of the aid. This is essential in patients who do not have clear or reliable thresholds (either behavioral or physiologic) without aids. (pg. 211)

ASSR methods for the estimation of the loudness discomfort levels, another crucial variable in hearing aid fitting, have not yet been developed. ASSRs may be present at higher stimulus levels (when toneburst-evoked ABRs are absent at the upper limits of the instrumentation), and thus can more accurately indicate the severity of a hearing loss and residual hearing levels. This is always a consideration when cochlear implantation is being considered. As in the case of hearing aids, the ASSR provides the audiometric data on which implantation decisions can be made.

There are a limited number of studies examining the use of ASSRs in evaluating CI function in humans (Hoffman and Wouters, 2010, 2012; Menard et al., 2004; Yang et al., 2008). Recording an ASSR in subjects with CIs is problematic because the CI itself creates a large electrical signal that can obscure the ASSR. The CI artifact problem arises because the CI extracts the envelope of an incoming signal and uses that derived envelope to modulate electrical pulses stimulating the auditory nerve. Therefore, the stimulus modulation frequency used to elicit the ASSR becomes part of the CI electrical artifact. The algorithms used to detect the ASSR are not able to distinguish between the ASSR and the CI electrical signal. It is not unreasonable to assume that that the CI artifact will be larger with greater stimulus intensities (assuming "automatic gain" functions are switched off) and therefore it is difficult to interpret studies that report ASSR thresholds similar to behavioral thresholds (e.g., Yang et al., 2008) if no measures are taken to reduce CI artifacts. Menard et al. (2004) examined the feasibility of recording ASSRs in the presence of CI artifacts by manipulating the CI pulse train duration and amplitude. To hear a sound, there must be sufficient charge density (pulse amplitude and duration) required to stimulate the auditory nerve. It was assumed that the amplitude of the CI artifact grew as a function of stimulus intensity and was linearly related to CI pulse amplitude. By varying the CI pulse duration and different pulse amplitudes the authors showed that some portion of the recorded ASSR does contain a physiological response, although it was impossible to know how much of the response was artifact, particularly at high stimulus levels. Overall the study found a reasonable relationship between ASSR threshold and behavioral threshold.

Another approach taken by Hofman and Wouters (2010) was to perform operations to reduce the CI artifact. First, they used the CI itself to generate short-duration biphasic pulses of alternate polarities at the desired modulation rate. A very obvious 100-μV stimulus artifact was observed. The averaged ASSR (across both polarities) still had a sizable artifact (roughly 20 μV) suggesting the alternating polarities are not entirely symmetrical. The next step in the artifact reduction process involved "cutting out" the artifact by interpolating the EEG time points between the remaining pulse artifacts. This last process reduced the recorded response to 500 nV (in the range for a 40-Hz ASSR). Correlations of $r > 0.96$ were observed between ASSR thresholds and behavioral thresholds. One drawback of the study was that the stimulus rates used (near 40 Hz) were not close to everyday clinical pulse rates (near 1,000 Hz). Additionally, if there were residual CI artifacts present (after removal) there is the possibility of false ASSR detection. In an effort to address these issues, the authors conducted a follow-up study (2012) in which new stimuli were constructed and new statistical methods employed. The two novel stimuli elicited larger ASSRs with artifacts 10 times larger than the single pulses. The authors employed a novel detection paradigm based on the assumption that ASSR phase, being a neural response, changes as a function of modulation rate whereas an artifact does not. Overall, strong correlations were documented between ASSR and perceptual thresholds. Obtaining ASSRs in response to the electrical stimuli provided by the implant is technically challenging. It remains to be determined if there are any advantages to using electrically evoked ASSRs compared to current techniques of electrically evoked compound nerve action potentials or ABRs, at least for threshold estimation. Because ASSRs provide a method to quantify suprathreshold temporal envelope-encoding ability, a critical feature of speech perception with a CI, there may be some impetus to overcome the technical hurdles. At this time, testing ASSRs in response to CI stimulation using standard, commercially available equipment is not recommended.

Differential Diagnosis of Sensory versus Neural Losses

There is limited information on the effect of neurologic compromise on the 80-Hz ASSR. The conditions for which there are published data are for auditory neuropathy (Rance et al., 1999, 2005), neurologic compromise because of confirmed lesions to the central auditory nervous system (Shinn and Musiek, 2007), and neurologic compromise because of prematurity (Cone-Wesson et al., 2002b). A summary of ASSR findings in 19 children with auditory neuropathy showed that there is no correspondence between behavioral hearing sensitivity and ASSR threshold (Rance et al., 2005). Puretone sensitivity was widely distributed between normal hearing and profound hearing loss, but the average ASSR threshold, regardless of CF, was around 85 to 90 dB HL. Correlations between puretone and ASSR thresholds averaged 0.51 in this group, compared to an average of 0.97 in the group with normal hearing or sensory/neural hearing loss. ASSRs cannot be used to estimate the puretone sensitivity of those with auditory neuropathy or retrocochlear or brainstem lesions. One problem that may occur, however, is in the case of an infant with auditory neuropathy for whom evoked otoacoustic emissions are absent. Unless one specifically tested for the presence of the cochlear microphonic, the absence of acoustic reflexes and an absent ABR might be interpreted as a severe-profound SNHL. Because ASSRs are known to be present in cases of severe-profound SNHL when ABRs are absent (Vander Werff et al., 2002), the presence of ASSR at elevated levels could be mistaken for an SNHL.[‡]

A discrepancy between behavioral thresholds and ASSR thresholds may be used as an indicator of neurologic dysfunction. Shinn and Musiek (2007) obtained 40-Hz ASSR thresholds in a group of patients with well-defined brain lesions. The author showed that the discrepancy between behavioral threshold and ASSR threshold was greater in the patient group, when compared to a control group of adults with normal neurologic status. This is qualitatively similar to the results of Rance et al. (2005), who also show large discrepancies between behavioral and ASSR threshold in the patients with auditory neuropathy.

It is unwise to use ASSR thresholds to estimate perceptual thresholds when the status of the central auditory system is unknown. It should be possible, though, to use ABR in conjunction with ASSRs to help determine the impact of neural hearing loss. ABR Wave I–V interwave intervals and/or abnormal waveform morphology (i.e., missing components, abnormal amplitude and latency of components) are useful for determining the presence of brainstem dysfunction (see Chapter 13). The combination of a suprathreshold click-evoked ABR along with ASSR threshold may be a rational way to approach an electrophysiologic assessment of the brainstem auditory system.

[‡]It is also possible to evoke a response from the vestibular system when high-level modulated stimuli are used. These may be mistaken for auditory responses unless steps to rule out other sources of artifact are undertaken (Gorga et al., 2004).

Other ASSR Applications

Although the vast majority of research efforts have focused on the hearing threshold estimation, applications of ASSRs in other domains are becoming more common. A sample of this work is given here.

Dimitrijevic et al. (2001) developed an innovative stimulus, composed of tones that are independently amplitude- and frequency-modulated (IAFM). They obtained significant correlations between the IAFM ASSRs and word-recognition scores in normal hearing adults. Specifically, they measured word recognition across stimulus level and compared the scores across level to the number of ASSRs present for an IAFM complex stimulus consisting of four CFs, each CF amplitude- and frequency-modulated at different rates. The number of ASSR components detected at each stimulus level was significantly correlated with the word-recognition score at a similar level. In a follow-up study (Dimitrijevic et al., 2004), the IAFM stimulus was refined to better represent the AM and FM components in natural speech. Results in normal hearing and hearing impaired subjects (with and without hearing aids) showed significant correlations between the word-recognition scores and the number of ASSR components present. These investigators suggest that ASSRs for multiple modulated tones correlate with word-recognition scores because both speech and multiple modulated tones contain information that varies rapidly in intensity and frequency. The ASSR "score" (i.e., the number of response components present for the 8-component stimulus) was modeled as an indicator of how much acoustic information was available to the listener. The more information available in the speech-frequency range, and for which the auditory system can process rapidly changing intensity and frequency cues, the better the word-recognition (speech discrimination) capabilities. More recently, Alaerts et al. (2009) found high correlations between ASSRs and phoneme identification and sentence perception in normal hearing and hearing impaired adults. Their ASSR stimulus was a speech-weighted noise carrier with AM at 4, 10, 20, and 38 Hz. These rates were chosen because much of the speech envelope contains information below 40 Hz (Rosen, 1992; Drullman, 1994). A combined measurement of ASSRs at 4, 10, and 20 Hz showed high correlations with phoneme and sentence identification. Interestingly, the higher modulation rate, 38 Hz, did not show a significant relationship with either phoneme or sentence scores.

The principle of using a complex ASSR stimulus for estimating speech perception abilities was applied in a cohort of infants under the age of 1 year by Cone and Garinis (2009).

They trained infants to perform a speech-feature detection task using an operant-conditioned response. Speech token levels were presented at several different levels to develop a performance-level function. ASSRs for the complex stimulus were also measured as a function of level. There was a strong correspondence between the ASSR and behavioral performance-level functions. As there are no methods for determining speech perception ability in preverbal infants, an electrophysiologic indicator, such as ASSR, could have benefit.

ASSRs in response to suprathreshold modulated noise have been correlated with temporal gap detection and the detection of modulation (Purcell et al., 2004). Young and old adult listeners had ASSRs recorded for modulated noise, in which the frequency of modulation was swept across the range of 20 to 600 Hz. They also underwent psychophysical tests of gap detection and modulation detection. First, the highest modulation frequency at which an ASSR was detected (using a 25% modulation depth) was significantly correlated ($r = 0.72$) with the modulation detection threshold. Second, the amplitude and phase (latency) of the ASSRs in several ranges of modulation were also correlated with modulation detection. Third, several of the ASSR response parameters were also correlated with gap detection. Because temporal processing is crucial for speech understanding, it is appropriate to develop electrophysiological methods by which temporal processing may be assessed. The ASSR may provide a means of doing so.

Related to temporal processing is phonemic awareness and discrimination. These abilities are also thought to be the basis of reading, and an impairment of temporal processing and/or phonemic awareness is believed to be the basis of some reading disabilities, that is, dyslexia (Goswami, 2011). Adults with dyslexia were shown to have lower 40-Hz ASSR amplitudes than typical readers (McAnally and Stein, 1997). More recent work has focused on differences in laterality of ASSRs at modulation rates associated with syllabic (<4 to 7 Hz) versus phonemic (20 to 40 Hz) processing (Vandermosten et al., 2013). Poelmans et al. (2012) measured group (dyslexic vs. controls) differences in response strength and also laterality for ASSRs at 20 Hz, but not for 80 or 4 Hz. They interpreted this as indicative of dysfunction for the "cortical phonemic processing rate" whereas the "cortical syllabic processing rate" responses (at 4 Hz) and the brainstem responses (at 80 Hz) were not different between the two groups. Vandermosten et al. (2013) used diffusion tensor imaging (DTI) of white matter in conjunction with ASSRs for 4- and 20-Hz modulation to test the hypotheses that dyslexics lack appropriate laterality in white matter and that this is related to the processing deficits identified by Poelmans et al. (2012). They found that, as a group, dyslexic adults demonstrated differences in white matter distribution for the superior posterior temporal gyrus, and arcuate fasciculus compared to typical readers. There was less pronounced left white matter dominance in dyslexics compared to the typical readers. The results from DTI and ASSR were interpreted as indicating both structural and functional differences in the brain that could be the bases for the phonemic processing problems demonstrated by those with dyslexia. The correlations between perceptual, electrophysiologic, and anatomical findings appear to be in support of the hypothesis that basic mechanisms of spectro-temporal processing are atypical or hypofunctional in those who are dyslexic.

Proposed ASSR Threshold Estimation Protocol

There are several ways to optimize ASSR threshold estimation tests. These take into account stimulus, acquisition, and patient factors that would be expected to influence the results.

Stimulus

For adults, modulation frequencies may be at 40 ± 5 Hz. As the effects of MM stimuli have not been formally evaluated at 40 Hz, SAM tones are recommended for 40-Hz ASSRs. For infants and children, the modulation rates should be at ≥80 Hz but ≤120 Hz, MM should be used, and the MF should increase with increasing CF. If more than one CF is presented at a time, the MFs for each CF should be separated by at least 3 Hz. The threshold for modulated noise should be determined prior to that for modulated tones. The threshold for modulated noise can be used to determine the level for initiating a threshold search for specific CFs. A 5-dB step size should be used for ASSR threshold searching with puretones, although a 10-dB step size may be useful for initial testing with modulated noise.

Acquisition

Filter settings should be at 1 to 300 or 30 to 300 Hz. The analog-to-digital conversion rate should be at 1,000 Hz or higher (but most commercial instruments do not allow choice of an A/D rate). Artifact rejection, if available, should be employed. The electrode montage for infants and young children should be Cz (vertex) to Mi (ipsilateral mastoid) with the common electrode on the opposite mastoid or forehead. For adults, an Fpz (high forehead) to Oz (inion) may be used.

Patient

Infants and young children should be in quiet sleep during the estimation of thresholds. A sedative or light anesthesia may be needed, if the infant is not able to maintain quiet

sleep for the duration of a complete test (40 to 60 minutes). Adults may be awake, but should be encouraged to recline in a comfortable chair, relax, and be still.

Test Method

Testing should not be initiated until the patient is sufficiently quiet. This can usually be determined by observing the patient and the ongoing EEG. Threshold is determined by decreasing the stimulus by 10 dB for each level at which the response criterion is met (i.e., the detection algorithm returns a result that meets the $p < 0.05$ level) and increased by 5 dB when no response is detected. How many averages does it take to determine that a response is *not* present? Some decisions must be made a priori, regarding the amplitude of the response that is to be detected. This decision, then, determines the amount of averaging needed, because it is the averaging process that allows the response to be resolved out of the background noise, as noise decreases with increased averaging. For example, detection of a response of 15 nV would require averaged noise levels to be lower than 10 nV. Depending on how "noisy" the recording is, it may take 10 to 12 minutes to obtain such fine resolution. A 10-nV "noise" criterion has been recommended as a stopping rule for terminating a trial averaging (Picton et al., 2003). Averaging should then proceed for as long as it takes to meet this criterion, or until a response is detected, whichever comes first. If the noise criterion is not met, and a response is not detected, this should be reported as a failure to achieve the a priori criterion. In such cases, a "threshold" cannot be determined. Raising the noise criterion will mean that ASSR thresholds are elevated in comparison to published norms for which the noise criterion was met.

There are other rules that may be employed at the discretion of the clinician, however, when application of these rules would be expected to affect threshold. For example, following the custom of "repeating" a trial as for ABR, some would require that a response be present for two independent trials given at the same level. This means that the criterion that a response is present has been made stricter. Using a stricter criterion will result in elevated thresholds in comparison to the more lax criterion. To avoid spurious "false-positive" responses, some require that the ASSR also be present at 10 dB above the lowest level for which a response is detected (to the same stimulus). False positives are sometimes seen when using a multifrequency technique wherein the ASSR thresholds vary as a function of frequency. For example, the threshold for a 500-Hz CF may be at 35 dB HL and for 2,000 Hz at 15 dB HL. In testing the 2,000-Hz response to threshold, a "response" to the 500-Hz CF may be detected at 15 dB HL, but not at 20, 25, or 30 dB HL. Thus, the "response" obtained at 15 dB HL is not considered valid.

Interpretation of Thresholds

The ASSR thresholds should be interpreted with respect to published data that have established the relationship between ASSR and puretone threshold. This may involve the use of regression formulae (see Table 15.2) or correction factors (see Table 15.3). An important aspect of interpreting ASSR thresholds in this way is to acknowledge the sample characteristics on which they were based, such as the age (infants, children, or adults), the type of hearing losses (conductive, sensory/neural, or mixed), and the range of hearing losses represented.

Case Study

Some of these principles are illustrated in the following case (Figure 15.9). The response spectra are shown in the left panel, with open triangles denoting responses for tones presented to the right ear and filled triangles for the left ear. The audiograms are shown on the right, with open circles denoting behavioral thresholds and filled squares indicating the ASSR thresholds. For the right ear, ASSRs at 500 Hz and 1.0 kHz are present at 40 dB HL; at 2.0 and 4.0 kHz, ASSRs are present at 60 and 70 dB HL, respectively. For the left ear, a response to 500 Hz is seen at 70 and 50 dB HL, but not at 60 dB HL. The response to 1.0 kHz is present at 40 dB HL, and to 2.0 kHz at 50 dB HL. The ASSR threshold at 4.0 kHz is 80 dB HL. Is ASSR threshold at 0.5 kHz at 70 dB HL or 50 dB HL? A steep upward slope to the audiogram between 0.5 and 1.0 kHz is not unheard of, yet not likely, given the overall configuration of the audiogram. Other information, such as tympanometry and acoustic-reflex thresholds, may also be used to interpret ASSR thresholds. Finally, the 0.5-kHz response is absent at 40 and 30 dB HL, suggesting that 50 dB HL is the threshold. Yet, if one adopted a conservative criterion, requiring the response to be present at 10 dB above the lowest level detected, then the threshold would be judged to be 70 dB HL.

Using ASSR–puretone threshold differences determined from a study of adults with sensory/neural hearing loss (Herdman and Stapells, 2003), the right ear puretone thresholds would be estimated to be 26, 32, 50, and 67 dB HL for octave frequencies at 0.5 to 4.0 kHz, respectively, and left ear thresholds would be estimated at 36, 32, 40, and 77 dB for the same frequencies. A mild-to-severe sloping bilateral loss is indicated. Comparing the estimated puretone thresholds to the true puretone thresholds, some discrepancies are obvious, but none exceed 10 dB.

Threshold Rules for ASSR Tests

Consider the threshold test findings in Table 15.5. This case is riddled with many problems. At 500 Hz, significant

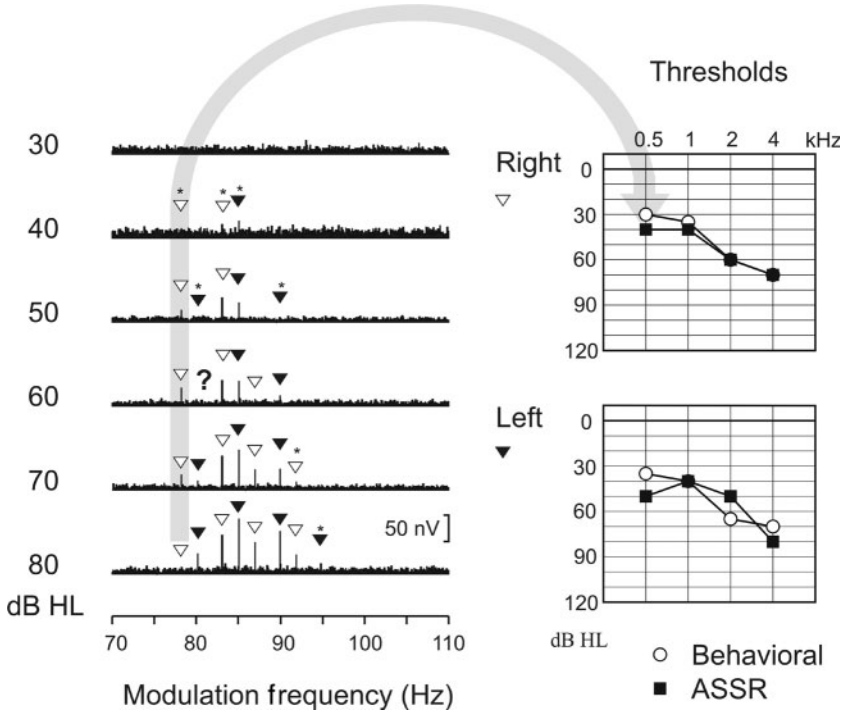


FIGURE 15.9 Response spectra and audiograms for case example. The response spectra are shown in the **left panel,** with *open triangles* denoting responses for tones presented to the right ear and *filled triangles* for the left ear. The audiograms are shown in the **right panel,** with *open circles* denoting behavioral thresholds and *filled squares* indicating the ASSR thresholds.

responses are obtained at 0 and 20 dB HL, but not at 10 dB HL. Is threshold at 0 or 20 dB HL? At 1,000 Hz, responses are present at 0, 20, and 40 dB HL, but not at 10 or 30 dB HL. Where is the threshold? The significant responses at 0 dB may be a false positive (with a statistical criterion of $p > 0.05$, there is a 5% probability of a false positive). On the other hand, the nonsignificant response at 10 dB may be a false negative. In these types of scenarios, it is essential to establish rules for ASSR threshold determination prior to the interpretation of the results. These rules should be reported in the results.

TABLE 15.5

Test Results as a Function of Level

	Carrier Frequency (Hz)			
Level (dB HL)	500	1,000	2,000	4,000
–10	0	0	X	0
0	X	X	0	0
10	0	0	0	0
20	X	X	0	0
30	X	0	0	0
40	X	X	X	0
50	X	X	X	X
60	X	X	X	0

X refers to a significant response and 0 to a nonsignificant response. See text for a discussion of threshold determination given these patterns of response/no response.

Examples of Rules

i. If there is one nonsignificant response at a stimulus level greater than a significant response, and significant responses are obtained at all higher stimulus levels, then it is assumed that the nonsignificant response is a false negative. In the example given in Table 15.5, threshold for 500 Hz is 0 dB HL because the 10-dB HL result is assumed to be a false negative, as results at 20 dB and higher were all significant. At 1,000 Hz, the threshold is at 20 dB HL because the 30-dB HL response is considered a false negative. The 0-dB HL response is a false positive.

ii. A significant response must be obtained at 20 dB above the lowest level at which there is a significant response. This is important to rule out any potential false positives. In the example above, threshold for 2,000 Hz would be 40 dB HL, and the 4,000-Hz threshold is unknown because the significant response at 50 dB HL could be a false positive.

iii. When in doubt, repeat tests at levels for which there are questionable responses.

Stopping Rules for ASSR Tests

For those who use instrumentation that allows the user to increase the number of sweeps for an average, the decision to stop recording must also be rule bound. The problem is the determination of no response. Specifically, if there is no response, how would we know if sampling for another 5 to 10 minutes might have resulted in a significant response? Usually, a "low-noise" and/or "time" rule can be used. Some potential stopping rules include the following:

i. Stop after 3 to 5 minutes when responses are significant (i.e., statistical significance must be maintained over a 3- to 5-minute period).
ii. Stop after 12 to 15 minutes when no responses are significant.
iii. Stop when averaged residual noise levels are at 10 to 15 nV (for 80-Hz ASSR) or 60 to 90 nV (for 40-Hz ASSRs).
iv. Stop after 12 minutes, or when averaged residual noise levels are 10 nV, whichever comes first.

As in the case of threshold rules, it is imperative to report the stopping rules used.

SUMMARY AND CONCLUSION

The clinical application of ASSRs, especially for threshold estimation in infants and young children, continues to expand. This is reflected in the steady flow of published studies on humans ($N = 189$) since 2005, the existence of several commercially available systems for recording ASSRs, and the pediatric audiology practice guidelines that acknowledge the role of ASSRs as an electrophysiologic assessment technique.

ASSRs provide an excellent estimate of hearing threshold across the audiometric range (250 to 8,000 Hz), particularly for those with moderate and greater degrees of hearing loss. Threshold tests can be completed in an objective fashion, owing to the detection algorithms indicating statistically significant responses. Results can also be interpreted objectively using regression formulae or correction factors for conversion of ASSR thresholds to (behavioral) puretone threshold estimates. Thresholds can be estimated for both air- and bone-conducted stimuli. Beyond threshold tests, ASSRs have been shown to be correlated with some aspects of suprathreshold hearing, particularly, word-recognition ability and atypical phonologic awareness found in those with dyslexia.

No single audiometric test stands alone as a diagnostic measure and neither does the ASSR. ASSRs should be used in conjunction with tests of middle-ear function (tympanometry and acoustic-reflex tests), cochlear function (evoked otoacoustic emissions), and other evoked potentials (ABR) and, when possible, behavioral hearing tests. Tympanometry and acoustic-reflex tests will aid in the interpretation of elevated ASSR thresholds, especially in the case of young infants with conductive hearing losses. The presence of otoacoustic emissions when ASSRs indicate significantly elevated thresholds is an indicator of auditory neuropathy. Click-evoked ABR tests provide important information about neural synchrony and brainstem integrity through the absolute and relative latencies of its constituent peaks; this information is not yet available from phase measurements of ASSRs. Although ASSRs are often used to estimate threshold in those too young or disabled to yield reliable behavioral thresholds, there should also be systematic attempts to document the infant's behavioral response to sound.

The goal is always to obtain the most information possible about the patient's hearing ability. ASSRs contribute a substantial amount toward that goal.

FOOD FOR THOUGHT

1. If asked, what is the better test of hearing threshold, toneburst ABR or toneburst ASSR, how would you answer? What are your reasons for doing so?
2. What are the major reasons why toneburst ASSR versus toneburst ASSR may yield different threshold estimates in a person with normal hearing? In a person with a conductive hearing loss? In a person with a sensory/neural hearing loss?
3. What behavioral tests should we use in conjunction with ASSR to learn more about typical and atypical auditory processing?

REFERENCES

Alaerts J, Luts H. (2009) Cortical auditory steady-state responses to low modulation rates. *Int J Audiol.* 48 (8), 582–593.

Aoyagi M, Furuse H, Yokota M, Kiren T, Suzuki Y, Koike Y. (1994a) Detectability of amplitude-modulation following response at different carrier frequencies. *Acta Otolaryngol.* 511 (suppl), 23–27.

Aoyagi M, Kiren T, Furuse H, Fuse T, Suzuki Y, Yokota M, et al. (1994b) Effects of aging on amplitude modulation-following response. *Acta Otolaryngol.* 511 (suppl), 15–22.

Aoyagi M, Kiren T, Furuse H, Suzuki Y, Yokota M, Koike Y. (1994) Pure-tone threshold prediction by 80-Hz amplitude-modulation following response. *Acta Otolaryngol Suppl.* 511, 7–14.

Aoyagi M, Kiren T, Kim Y, Suzuki Y, Fuse T, Koike Y. (1993) Frequency specificity of amplitude-modulation-following response detected by phase spectral analysis. *Audiology.* 32, 293–301.

Aoyagi M, Suzuki Y, Yokota M, Furuse H, Watanabe T, Ito T. (1999) Reliability of 80-Hz amplitude modulation-following response detected by phase coherence. *Audiol Neurootol.* 4, 28–37.

Boettcher FA, Poth EA, Mills JH, Dubno JR. (2001) The amplitude-modulation following response in young and aged human subjects. *Hear Res.* 153, 32–42.

Casey KA, Small SA. (2014) Comparisons of auditory steady state response and behavioral air conduction and bone conduction thresholds for infants and adults with normal hearing. *Ear and Hearing*. doi:10.1097/AUD.0000000000000021.

Chambers RD, Meyer TA. (1993) Reliability of threshold estimation in hearing-impaired adults using the AMFR. *J Am Acad Audiol.* 4, 22–32.

Clarey JC, Barone P, Imig TJ. (1992) Physiology of thalamus and cortex. In: Popper AN, Fay RR, eds. *The Mammalian Auditory Pathway: Neurophysiology.* New York: Springer-Verlag.

Cohen LT, Rickards FW, Clark GM. (1991) A comparison of steady state evoked potentials to modulated tones in awake and sleeping humans. *J Acoust Soc Am.* 90, 2467–2479.

Cone-Wesson B. (1995) Bone-conduction ABR tests. *Am J Audiol.* 4 (3), 14–19.

Cone-Wesson B, Dowell RC, Tomlin D, Rance G, Ming WJ. (2002a) The auditory steady-state response: comparisons with the auditory brainstem response. *J Am Acad Audiol.* 13, 173–187.

Cone-Wesson B, Parker J, Swiderski N, Rickards FW. (2002b) The auditory steady-state response: full term and premature neonates. *J Am Acad Audiol.* 13, 260–269.

Cone-Wesson B, Ramirez GM. (1997) Hearing sensitivity in newborns estimated from ABRs to bone-conducted sounds. *J Am Acad Audiol.* 8, 299–307.

Cone-Wesson B, Rickards FW, Poulis C, Parker J, Tan L, Pollard J. (2002c) The auditory steady-state response: clinical observations and applications in infants and children. *J Am Acad Audiol.* 13, 270–282.

Dauman R, Szyfter W, Charlet de Sauvage R, Cazals Y. (1984) Low frequency thresholds assessed with 40 Hz MLR in adults with impaired hearing. *Arch Otorhinolaryngol.* 240, 85–89.

D'haenens W, Dhooge I, Maes L, Bockstael A, Keppler H, Philips B, et al. (2009) The clinical value of the multiple-frequency 80-Hz auditory steady-state response in adults with normal hearing and hearing loss. *Arch Otolaryngol Head Neck Surg.* 135 (5), 496–506.

Dimitrijevic A, John MS, Picton TW. (2004) Auditory steady-state responses and word recognition scores in normal hearing and hearing-impaired adults. *Ear Hear.* 25, 68–84.

Dimitrijevic A, John MS, Van Roon P, Purcell DW, Adamonis J, Ostroff J, et al. (2002) Estimating the audiogram using multiple auditory steady-state responses. *J Am Acad Audiol.* 13, 205–224.

Dimitrijevic A, John S, Van Roon P, Picton TW. (2001) Human auditory steady-state responses to tones independently modulated in both frequency and amplitude. *Ear Hear.* 22, 100–111.

Dobie RA, Wilson MJ. (1989a) Analysis of auditory evoked potentials by magnitude-squared coherence. *Ear Hear.* 10 (1), 2–13.

Dobie RA, Wilson MJ. (1989b) Objective response detection in the frequency domain. *Electroencephalogr Clin Neurophysiol.* 88, 516–524.

Dobie RA, Wilson MJ. (1993) Objective response detection in the frequency domain. *Electroencephalogr Clin Neurophysiol.* 88 (6), 516–524.

Dobie RA, Wilson MJ. (1995) Objective versus human observer detection of 40-Hz auditory-evoked potentials. *J Acoust Soc Am.* 97, 3042–3050.

Dobie RA, Wilson MJ. (1996) A comparison of the t-test, F-test and coherence methods of detecting steady-state auditory-evoked potentials, distortion product otoacoustic emissions or other sinusoids. *J Acoust Soc Am.* 100, 2234–2246.

Dobie RA, Wilson MJ. (1998) Low-level steady-state auditory evoked potentials: effects of rate and sedation on detectability. *J Acoust Soc Am.* 104, 3482–3488.

Drullman R, Festen JM, Plomp R. (1994) Effect of reducing slow temporal modulations on speech reception. *J Acoust Soc Am.* 95 (5 Pt 1), 2670–2680.

Elberling C, Don M, Cebulla M, Stürzebecher E. (2007) Auditory steady-state responses to chirp stimuli based on cochlear traveling wave delay. *J Acoust Soc Am.* 122 (5), 2772–2785.

Elberling C, Wahlgreen O. (1985) Estimation of auditory brainstem response, ABR, by means of Bayesian inference. *Scand Audiol.* 14, 89–96.

Gorga MP, Neely ST, Hoover BM, Dierking DM, Beauchaine KL, Manning C. (2004) Determining the upper limits of stimulation for auditory steady-state response measurements. *Ear Hear.* 25, 302–307.

Goswami U. (2011) A temporal sampling framework for developmental dyslexia. *Trends Cogn Sci.* 15, 3–10.

Grose JH, Mamo SK, Hall JW 3rd. (2009) Age effects in temporal envelope processing: speech unmasking and auditory steady state responses. *Ear Hear.* 30, 568–575.

Herdman AT, Lins O, Van Roon P, Stapells DR, Scherg M, Picton TW. (2002a) Intracerebral sources of human auditory steady-state responses. *Brain Topogr.* 15, 69–86.

Herdman AT, Picton TW, Stapells DR. (2002b) Place specificity of multiple auditory steady-state responses. *J Acoust Soc Am.* 112, 1569–1582.

Herdman AT, Stapells DR. (2001) Thresholds determined using the monotic and dichotic multiple steady-state response technique in normal-hearing subjects. *Scand Audiol.* 30, 41–49.

Herdman AT, Stapells DR. (2003) Auditory steady state response thresholds of adults with sensorineural hearing impairment. *Int J Audiol.* 42, 237–248.

Hofmann M, Wouters J. (2010) Electrically evoked auditory steady state responses in cochlear implant users. *J Assoc Res Otolaryngol.* 11 (2), 267–282.

Hofmann M, Wouters J. (2012) Improved electrically evoked auditory steady-state response thresholds in humans. *J Assoc Res Otolaryngol.* 13 (4), 573–589.

Hulecki LR, Small SA. (2011) Behavioral bone-conduction thresholds for infants with normal hearing. *J Am Acad Audiol.* 22 (2), 81–92.

Irvine D. (1992) Physiology of the auditory brainstem. In: Popper AN, Fay RR, eds. *The Mammalian Auditory Pathway: Neurophysiology.* New York: Springer-Verlag.

Ishida I, Cuthbert B, Stapells D. (2011) Multiple auditory steady state response thresholds to bone conduction stimuli in adults with normal and elevated thresholds. *Ear Hear.* 32 (2), 373–381.

John MS, Brown DK, Muir PJ, Picton TW. (2004) Recording auditory steady-state responses in young infants. *Ear Hear.* 25, 539–553.

John MS, Dimitrijevic A, Picton TW. (2001a) Weighted averaging of steady-state responses. *Clin Neurophysiol.* 112, 555–562.

John MS, Dimitrijevic A, Picton TW. (2003) Efficient stimuli for evoking auditory steady-state responses. *Ear Hear.* 24, 406–423.

John MS, Dimitrijevic A, Van Roon P, Picton TW. (2001b) Multiple auditory steady-state responses to AM and FM stimuli. *Audiol Neurootol.* 6, 12–27.

Johnson TA, Brown CJ. (2005) Threshold prediction using the auditory steady-state response and the toneburst auditory brainstem response: a within-subject comparison. *Ear Hear.* 26, 559–576.

Kankkunen A, Rosenhall U. (1985) Comparison between thresholds obtained with pure-tone audiometry and the 40-Hz middle latency response. *Scand Audiol.* 14, 99–104.

Khanna SM, Teich MC. (1989a) Spectral characteristics of the responses of primary auditory-nerve fibers to amplitude-modulated signals. *Hear Res.* 39, 143–157.

Khanna SM, Teich MC. (1989b) Spectral characteristics of the responses of primary auditory nerve fibers to frequency-modulated signals. *Hear Res.* 39, 159–175.

Klein AJ. (1983) Properties of the brain-stem response slow-wave component. I. Latency, amplitude and threshold sensitivity. *Arch Otolaryngol.* 109, 6–12.

Kraus N, McGee T, Stein L. (1994) The auditory middle latency response. In: Jacobsen JT, ed. *Principles and Applications in Auditory Evoked Potentials.* Boston: Allyn and Bacon; pp 123–154.

Kuwada S, Anderson JS, Batra R, Fitzpatrick DC, Teissier N, D'Angelo WR. (2002) Sources of the scalp-recorded amplitude-modulation following response. *J Am Acad Audiol.* 13, 188–204.

Lasky RE. (1984) A developmental study on the effect of stimulus rate on the auditory evoked brain-stem response. *Electroencephalogr Clin Neurophysiol.* 59, 411–419.

Levi EC, Folsom RC, Dobie RA. (1993) Amplitude-modulation following response (AMFR): effects of modulation rate, carrier frequency, age and state. *Hear Res.* 68, 42–52.

Levi EC, Folsom RC, Dobie RA. (1995) Coherence analysis of envelope-following responses (EFRs) and frequency-following responses (FFRs) in infants and adults. *Hear Res.* 89, 21–27.

Lin Y-H, Ho H-C, Wu H-P. (2009) Comparison of auditory steady-state responses and auditory brainstem responses in audiometric assessment of adults with sensorineural hearing loss. *Auris, Nasus, Larynx*. 36 (2), 140–145.

Lins OG, Picton P, Picton TW, Champagne SC, Durieux-Smith A. (1995) Auditory steady-state responses to tones amplitude-modulated at 80–110 Hz. *J Acoust Soc Am.* 97 (1), 3051–3063.

Lins OG, Picton TW. (1995) Auditory steady-state responses to multiple simultaneous stimuli. *Electroencephalogr Clin Neurophysiol.* 96, 420–432.

Lins OG, Picton TW, Boucher BL, Durieux-Smith A, Champagne SC, Moran LM, et al. (1996) Frequency specific audiometry using steady-state responses. *Ear Hear.* 17, 81–96.

Luts H, Wouters J. (2004) Hearing assessment by recording multiple auditory steady-state responses: the influence of test duration. *Int J Audiol*. 43 (8), 471–478.

Luts H, Wouters J. (2005) Comparison of MASTER and AUDERA for measurement of auditory steady-state responses. *Int J Audiol.* 44, 244–253.

Lynn JM, Lesner SA, Sandridge SA, Daddario CC. (1984) Threshold prediction from the auditory 40-Hz evoked potential. *Ear Hear.* 5, 366–370.

McAnally KI, Stein JF. (1997) Scalp potentials evoked by amplitude-modulated tones in dyslexia. *J Speech Lang Hear Res.* 40, 939–945.

Menard M, Galleyo S, Truy E, Berger-Vachon C, Durrant JD, Collet L. (2004) Auditory steady-state response evaluation of auditory thresholds in cochlear implant patients. *Int J Audiol.* 43 (suppl 1), S39–S43.

Milford CA, Birchall JP. (1989) Steady-state auditory evoked potentials to amplitude-modulated tones in hearing-impaired subjects. *Br J Audiol.* 23, 137–142.

Møller AR. (1994) Neural generators of auditory evoked potentials. In: Jacobsen JT, ed. *Principles and Applications in Auditory Evoked Potentials.* Boston: Allyn and Bacon; pp 23–46.

Muchnik C, Katz-Putter H, Rubinstein M, Hildesheimer M. (1993) Normative data for 40-Hz event-related potentials to 500-Hz tonal stimuli in young and elderly subjects. *Audiology.* 32, 27–35.

Ozdek A, Karacay M, Saylam G, Tatar E, Aygener N, Korkmaz MH. (2010) Comparison of pure tone audiometry and auditory steady-state responses in subjects with normal hearing and hearing loss. *Eur Arch Otorhinolaryngol*. 267 (1), 43–49.

Perez-Abalo MC, Savio G, Torres A, Martin V, Rodriguez E, Galan L. (2001) Steady-state responses to multiple amplitude-modulated tones: an optimized method to test frequency-specific thresholds in hearing-impaired children and normal-hearing subjects. *Ear Hear.* 22, 200–211.

Picton TW, Dimitrijevic A, Perez-Aballo M-C, Van Roon P. (2005) Estimating audiometric thresholds using auditory steady-state responses. *J Am Acad Audiol.* 16, 140–156.

Picton TW, Durieux-Smith A, Champagne SC, Whittingham J, Moran LM, Giguere C, et al. (1998) Objective evaluation of aided thresholds using auditory steady-state responses. *J Am Acad Audiol.* 9, 315–331.

Picton TW, John MS. (2004) Avoiding electromagnetic artifacts when recording auditory steady-state responses. *J Am Acad Audiol.* 15, 541–554.

Picton TW, John MS, Dimitrijevic A, Purcell D. (2003) Human auditory steady-state responses. *Int J Audiol.* 42, 177–219.

Picton T, Skinner C. (1987) Potentials evoked by the sinusoidal modulation of the amplitude or frequency of a tone. *J Acoust Soc Am*. 82 (1), 165–178.

Poelmans H, Luts H, Vandermosten M, Boets B, Ghesquière P, Wouters J. (2012) Auditory steady state cortical responses indicate deviant phonemic-rate processing in adults with dyslexia. *Ear Hear.* 33, 134–143.

Poulsen C, Picton TW, Paus T. (2007) Age-related changes in transient and oscillatory brain responses to auditory stimulation in healthy adults 19–45 years old. *Cereb Cortex*. 17 (6), 1454–1467.

Purcell DW, John SM, Schneider BA, Picton TW. (2004) Human temporal auditory acuity as assessed by envelope following responses. *J Acoust Soc Am.* 116, 3581–3593.

Rance G, Beer DE, Cone-Wesson B, Shepherd RK, Dowell RC, King AM, et al. (1999) Clinical findings for a group of infants and young children with auditory neuropathy. *Ear Hear.* 20, 238–252.

Rance G, Briggs RJS. (2002) Assessment of hearing in infants with moderate to profound impairment: the Melbourne experience with auditory steady-state evoked potential testing. *Ann Otol Rhinol Laryngol.* 111, 22–28.

Rance G, Dowell RC, Rickards FW, Beer DE, Clark GM. (1998) Steady-state evoked potential and behavioral hearing thresholds in a group of children with absent click-auditory brain stem response. *Ear Hear.* 19, 48–61.

Rance G, Rickards FW. (2002) Prediction of hearing threshold in infants using auditory steady-state evoked potentials. *J Am Acad Audiol.* 13, 236–245.

Rance G, Rickards FW, Cohen LT, DeVidi S, Clark GM. (1995) The automated prediction of hearing thresholds in sleeping subjects using auditory steady state evoked potentials. *Ear Hear.* 16, 499–507.

Rance G, Roper R, Symons L, Moody LJ, Poulis C, Dourlay M, et al. (2005) Hearing threshold estimation in infants using auditory steady-state responses. *J Am Acad Audiol.* 16, 291–300.

Rance G, Tomlin D. (2006) Maturation of auditory steady-state responses in normal babies. *Ear Hear.* 27, 20–39.

Rees A, Green GG, Kay RH. (1986) Steady-state evoked responses to sinusoidally amplitude-modulated sounds recorded in man. *Hear Res.* 23, 123–133.

Reyes SA, Salvi RJ, Burkard RF, Coad ML, Wack DS, Galantowicz PJ, et al. (2004) PET imaging of the 40 Hz auditory steady state response. *Hear Res.* 194, 73–80.

Rhode WS, Greenberg S. (1992) Physiology of the cochlear nuclei. In: Popper AN, Fay RR, eds. *The Mammalian Auditory Pathway: Neurophysiology.* New York: Springer-Verlag.

Ribeiro FM, Carvallo RM, Marcoux AM. (2010) Auditory steady-state evoked responses for preterm and term neonates. *Audiol Neurootol.* 15 (2), 97–110.

Rickards FW, Tan LE, Cohen LT, Wilson OJ, Drew JH, Clark GM. (1994) Auditory steady-state evoked potential in newborns. *Br J Audiol.* 28, 327–337.

Rodriguez R, Picton T, Linden D, Hamel G, Laframboise G. (1986) Human auditory steady state responses: effects of intensity and frequency. *Ear Hear.* 7, 300–313.

Ross B, Herdman AT, Pantev C. (2005) Right hemispheric laterality of human 40 Hz auditory steady-state responses. *Cereb Cortex.* 15, 2029–2039.

Rosen S. (1992) Temporal information in speech: acoustic, auditory and linguistic aspects. *Philos Trans R Soc Lond B Biol Sci.* 336 (1278), 367–373.

Ruggero MA. (1992) Physiology and coding of sound in the auditory nerve. In: Popper AN, Fay RR, eds. *The Mammalian Auditory Pathway: Neurophysiology.* New York: Springer-Verlag.

Sammeth CA, Barry SJ. (1985) The 40-Hz event-related potential as a measure of auditory sensitivity in normals. *Scand Audiol.* 14, 51–55.

Savio G, Cardenas J, Perez-Abalo M, Gonzalez A, Valdes J. (2001) The low and high frequency auditory steady state responses mature at different rates. *Audiol Neurootol.* 6, 279–287.

Scollie SD, Sweewald RC. (2002) Evaluation of electroacoustic test signals I: comparison with amplified speech. *Ear Hear.* 23, 477–487.

Shinn JB, Musiek FE. (2007) The auditory steady state response in individuals with neurological insult of the central auditory nervous system. *J Am Acad Audiol.* 18 (10), 826–845.

Small SA, Stapells DR. (2004) Stimulus artifact issues when recording auditory steady-state responses. *Ear Hear.* 25, 611–623.

Small SA, Stapells DR. (2005) Multiple auditory steady-state responses to bone-conduction stimuli in adults with normal hearing. *J Am Acad Audiol.* 16, 172–183.

Stapells DR. (2000) Threshold estimation by the tone-evoked auditory brainstem response: a literature meta-analysis. *J Speech Lang Pathol Audiol.* 24, 74–83.

Stapells DR, Galambos R, Costello JA, Makeig S. (1988) Inconsistency of auditory middle latency and steady-state responses in infants. *Electroencephalogr Clin Neurophysiol.* 71, 289–295.

Stapells DR, Gravel JS, Martin BA. (1995) Thresholds for auditory brainstem responses to tones in notched noise from infants and young children with normal hearing or sensorineural hearing loss. *Ear Hear.* 16 (4), 361–371.

Stapells DR, Linden D, Suffield JB, Hamel G, Picton TW. (1984) Human auditory steady state potentials. *Ear Hear.* 5, 105–113.

Stapells DR, Makeig S, Galambos R. (1987) Auditory steady-state responses: threshold prediction using phase coherence. *Electroencephalogr Clin Neurophysiol.* 67, 260–270.

Steinmann I, Gutschalk A. (2011) Potential fMRI correlates of 40-Hz phase locking in primary auditory cortex, thalamus and midbrain. *NeuroImage.* 54 (1), 495–504.

Stürzebecher E, Cebulla M, Elberling C, Berger T. (2006) New efficient stimuli for evoking frequency-specific auditory steady-state responses. *J Am Acad Audiol.* 17 (6), 448–461.

Szalda K, Burkard R. (2005) The effects of nembutal anesthesia on the auditory steady-state response (ASSR) from the inferior colliculus and auditory cortex of the chinchilla. *Hear Res.* 203, 32–44.

Szyfter W, Dauman R, de Sauvage RC. (1984) 40 Hz middle latency responses to low frequency tone pips in normally hearing adults. *J Otolaryngol.* 13, 275–280.

Tomlin D, Rance G, Graydon K, Tsialios I. (2006) A comparison of 40 Hz auditory steady-state response (ASSR) and cortical auditory evoked potential (CAEP) thresholds in awake adult subjects. *Int J Audiol.* 45 (10), 580–588.

Tlumak AI, Durrant JD, Delgado RE, Boston JR. (2012) Steady-state analysis of auditory evoked potentials over a wide range of stimulus repetition rates in awake vs. natural sleep. *Int J Audiol.* 51 (5), 418–423.

Tlumak AI, Rubinstein E, Durrant JD. (2007) Meta-analysis of variables that affect accuracy of threshold estimation via measurement of the auditory steady-state response (ASSR). *Int J Audiol.* 46 (11), 692–710.

Van der Reijden CS, Mens LH, Snik AF. (2004) Signal-to-noise ratios of the auditory steady-state response from fifty-five EEG derivations in adults. *J Am Acad Audiol.* 15 (10), 692–701.

Van Dun B, Wouters J, Moonen M. (2009) Optimal electrode selection for multi-channel electroencephalogram based detection of auditory steady-state responses. *J Acoust Soc Am.* 126 (1), 254–268.

Vander Werff KR, Brown CJ. (2005) The effect of audiometric configuration on threshold and suprathreshold auditory steady-state responses. *Ear Hear.* 26, 310–326.

Vander Werff KR, Brown CJ, Gienapp BA, Clay KMS. (2002) Comparison of auditory steady-state response and auditory brainstem response thresholds in children. *J Am Acad Audiol.* 13, 227–235.

Vandermosten M, Poelmans H, Sunaert S, Ghesquiere P, Wouters J. (2013) White matter lateralization and interhemispheric coherence to auditory modulations in normal reading and dyslexic adults. *Neuropsychologica.* 51, 2087–2099.

Van Maanen A, Stapells DR. (2005) Comparison of multiple auditory steady-state responses (80 vs 40 Hz) and slow cortical potentials for threshold estimation in hearing-impaired adults. *Int J Audiol.* 44, 613–624.

Victor JD, Mast J. (1991) A new statistic for steady-state evoked potentials. *Electroencephalogr Clin Neurophysiol.* 78, 378–288 [published erratum in *Electroencephalogr Clin Neurophysiol.* (1992), 83, 270].

Viemeister NF. (1979) Temporal modulation transfer functions based upon modulation thresholds. *J Acoust Soc Am.* 66, 1364–1380.

Yang CH, Chen HC, Hwang CF. (2008) The prediction of hearing thresholds with auditory steady-state responses for cochlear implanted children. *Int J Pediatr Otorhinolaryngol.* 72 (5), 609–617.

Yang EY, Rupert AL, Moushegian G. (1987) A developmental study of bone conduction auditory brainstem response in infants. *Ear Hear.* 8 (4), 244–251.

Ysunza A, Cone-Wesson B. (1987) Bone conduction masking for brainstem auditory-evoked potentials (BAEP) in pediatric audiological evaluations. Validation of the test. *Int J Pediatr Otorhinolaryngol.* 12, 291–302.

CHAPTER 16

Intraoperative Neurophysiological Monitoring

Paul R. Kileny and Bruce M. Edwards

OVERVIEW

Intraoperative neurophysiological monitoring (IOM) is the term we will use in this chapter for this specialized aspect of audiology. Other similar terms include intraoperative neuromonitoring and cranial nerve monitoring. The purpose of IOM is to reduce the incidence of injury to the patient's central and peripheral nervous systems during surgery. This is accomplished by using a variety of electrophysiological methods during surgery. By virtue of their education and additional training, scope of practice, licensure, and/or additional specialty certification, audiologists and/or physicians are permitted to interpret IOM results. Further, audiologists own and supervise IOM programs that are staffed by other audiologists and in some cases they work remotely with trained technicians. This aspect of audiology has grown in sophistication and acceptance in the past 20 to 30 years.

We hope that this chapter will be instructive and illuminating as well as encourage the reader to seek additional information about this interesting, clinically effective field. Although intended primarily for audiology students and practicing audiologists, we believe that the contents of this chapter will be of assistance to other healthcare providers seeking information about intraoperative monitoring.

The earliest form of "intraoperative monitoring" was the brainchild of the renowned neurosurgeon Harvey Cushing who while in medical school contributed to the development of blood pressure monitoring during surgery and developed the prototype of the flow sheet used to monitor patients' vital signs during general anesthesia. With some modifications, this type of data tracking, now computerized, is still used today in countless operating rooms around the world. In the last five to six decades, there has been a remarkable evolution in the efficacy and safety of surgical techniques performed near or directly involving the central and peripheral nervous systems. For instance, as recently as midway through the previous century acoustic neuroma resection was considered to be successful if the tumor was completely resected and the patient survived with minimal or no significant neurological sequelae. Surgery performed to remove an acoustic neuroma was not initially concerned with the preservation of cranial nerve function. However, with advances in the understanding of surgical anatomy and with developments in instrumentation including the introduction of the operating microscope and associated microsurgical techniques, a burgeoning interest in the preservation of neural function occurred. Acoustic neuroma surgery is an example of a setting in which technologic advances have led to improved functional outcomes. In otology and neurotology in particular, refinements in temporal bone surgery have brought about an increased emphasis on the preservation of auditory and facial function. Similar advances may be found in head and neck surgery for selected primary parotidectomy, revision parotidectomy, and selected thyroid procedures. Now, goals typically include functional maintenance of the extracranial seventh facial nerve and the recurrent laryngeal nerve, a branch of the vagus nerve, during these often complex procedures.

ABOUT INTRAOPERATIVE NEUROPHYSIOLOGICAL MONITORING

The goal of preserving a patient's neural function intraoperatively faces several challenges for a surgical team. For example, an expanding tumor can compress a cranial nerve within a closed space making identification of specific neural structures daunting. Although the surgeon's skill level is crucial to a successful outcome, a limited ability to simply visualize targeted cranial nerves in the setting of an expansive mass lesion makes even more valuable one's ability to continuously monitor and properly interpret electromyography (EMG) and auditory-evoked potentials when a goal of surgery includes maintaining both the structural integrity of the target nerve and its function, too.

Interest in and continued developments in neurodiagnostic techniques by audiology and other disciplines represents an important step in the growth and acceptance of intraoperative monitoring and continued improvement in patients' functional outcomes. A landmark contribution to this field is one that every audiologist should recognize: the description and introduction into clinical practice of the auditory brainstem response (ABR), described by Jewett et al. (1970). The advent of this robust and replicable evoked potential, present even in a state of deep anesthesia, generated an explosion of clinical investigations involving the ABR. It became one

of numerous neurophysiological auditory diagnostic techniques used clinically and was joined by several forms of facial muscle EMG, too. The diagnostic applications of auditory-evoked potential and electromyographic techniques led to an improved understanding of pathological changes involving affected anatomic structures and the responses associated with those neural structures. Retrospectively, these discoveries were necessary and important precursors to current uses of neurophysiological measures in the operating room.

In the late 1970s, to the early 1980s interest was focused on preserving neural function and structures, improving microsurgical techniques, and the broadening of intraoperative use as well as the variety of neuropsychological techniques that led to improved surgical outcomes. These intraoperative methods were modifications of methods used in outpatient diagnostics and were redeveloped with a goal of being capable of providing feedback about the status of neural structures during surgery. Neurophysiological intraoperative monitoring has developed over time and now contributes to improved preservation of facial and auditory function, as well as preservation of various cranial nerves and associated function in otolaryngology-head and neck surgery such as acoustic neuroma resection, retrolabyrinthine vestibular nerve section, repair of semicircular canal dehiscence, microvascular decompression of cranial nerves V, VII, or VIII, as well as preserved cranial nerve function when operating on patients with congenital temporal bone anomalies and cancer of the salivary and endocrine glands.

At this point, it is important to state that IOM should be considered an adjunct to the surgically trained and skilled physicians who operate on or in the vicinity of neural structures. It is also important to note that despite improved technology, it is the authors' opinion that certain "automated," nonattended monitoring devices and techniques based on acoustic alarms are no substitute for the skill, experience, and interactive ability of a skilled clinician who actively interprets monitored activity and provides continuous feedback about one or more neural structures.

The individual responsible for monitoring must have extensive experience in clinical neurophysiology, must understand the pathological effects of the lesion to be treated surgically, and must be familiar with surgical anatomy and technique. In addition, the individual providing intraoperative monitoring should appreciate the pace and flow of the surgical procedure in which they participate. The training needed to become proficient in IOM is determined by an individual's background, motivation, clinical skill, and talents. Solid footing in the principles of neural stimulation and recording, electrophysiology, and surgical anatomy should be fundamental components of a training program in this specialty area. Precept or apprentice-type teaching may provide a valuable method of obtaining practical experience in the operative room. Although these educational activities cannot guarantee quality, they can serve as guidelines in developing a training program.

WHY IS THIS CLINICAL SUBSPECIALTY NEEDED?

Complex primary and revision surgery that risks neural structures, important anatomical components, and blood supply requires that a variety of intraoperative procedures be conducted to monitor, interpret, and continuously report on the integrity of those structures. Thus, the single most important role for the audiologist who performs intraoperative monitoring is to ensure the integrity of neural pathways and associated structures. This is accomplished by identifying and attempting to prevent unintended complications during the surgical procedures. Continuous monitoring with real-time interpretation and prompt intervention can result in improved outcomes for patients, assuming that the audiologist is able to differentiate between changes in baseline activity associated with, for example, anesthetic intervention versus significant surgical events (Edwards and Kileny, 2000). Properly conducted intraoperative monitoring can identify impending neurological insult in settings such as the dissection of a vestibular schwannoma that has wrapped itself around the seventh, fifth, or lower cranial nerves or with manipulation of an offending intracranial structure during microvascular decompression of cranial nerves V, VII, or IX. Properly trained individuals with appropriate knowledge and experience can assist the surgical team to quickly carry out their work.

The necessity for IOM was underscored by the now-expired National Institutes of Health (NIH) Consensus Statement regarding the management of acoustic neuromas and the role of facial nerve monitoring in those procedures (NIH, Acoustic Neuroma Consensus Statement, December, 1991). The conclusions and recommendations of the statement reported that ". . . the benefits of routine intraoperative monitoring of the facial nerve have been clearly established. This technique should be included in surgical therapy for vestibular schwannoma. Routine monitoring of other cranial nerves should be considered." The authors continue to agree with this now historically significant document and suggest that intraoperative monitoring has further expanded in its scope and importance to the profession of audiology. Numerous key purposes exist for continuous intraoperative monitoring in surgeries that involve the brain and brainstem, the spinal cord, and the peripheral nervous system including:

a. enabling changes in the course of surgical procedures to avoid postoperative complications,
b. prediction of patients' postoperative outcomes, and
c. retrospective review of monitoring data to enhance future surgical procedures (Sala et al., 2002).

We advocate that properly utilized instrumentation allows for the collection of data that could be valuable in certain medicolegal situations and also as components in an educational program for trainees. The training and education of

audiology students and resident surgeons is an added value for providers of IOM.

SAFEGUARDING PATIENT OUTCOMES BY STANDARDIZING IOM

Standardizing an intraoperative monitoring service is essential to avoid the many hazards and pitfalls present in all operating rooms and to ensure a consistent approach to providing the service. Edwards and Kileny (2000) have identified key components useful for creating and conducting a systematic, thoughtful IOM service. Attending to these items prior to the surgical procedure enables the audiologist to safeguard against unplanned or unfortunate incidents. Table 16.4 lists selected components useful in planning and establishing an intraoperative neurophysiological monitoring program that can serve multiple surgical services.

Before carrying out any form of intraoperative monitoring, important questions to answer should include the following:

- "Why is the patient having surgery?"
- "What type of equipment will be most useful for this case?"
- "What motor and/or sensory cranial nerves could come into play during this procedure?"
- "What are key portions of this surgical procedure that involve IOM?"
- "What are the anticipated outcomes of IOM and surgery?"

The specific monitoring setup for each operative procedure should be well thought out before one's entry into the operating room and should be formed in part by the patient's medical and surgical history. Further, the comprehensive plan for monitoring should be discussed with stakeholders including the patient, the attending surgeon, anesthesiology, and nursing. In this way, everyone is aware of the planned monitoring setup for the patient, the anesthetics that may best support nerve monitoring, the benefits and limitations that IOM has to offer, and the cost-carrying supplies that will be needed by the monitoring team.

Table 16.1 provides a simple planning scheme for intraoperative cranial nerve monitoring in selected surgical procedures. This could be used to standardize initial, presurgical preparation for a variety of operative procedures that involve cranial nerves. In the table, it can be seen that some operative cases involve monitoring just one cranial nerve (see revision mastoidectomy or parotidectomy), whereas other surgical interventions benefit either from multimotor nerve monitoring (see plans for surgical approaches to masses in the parapharyngeal space) or multimodality cranial nerve

TABLE 16.1

Simple Planning Scheme for Intraoperative Cranial Nerve Monitoring in Selected Surgical Procedures

Procedure	CN III	CN IV	CN V	CN VI	CN VII	CN VIII	CN IX	CN X	CN XI	CN XII
Otology										
Revision mastoidectomy					✓					
Aural atresia					✓					
Cochlear implant					✓	✓[a]				
Vestibular Nerve Section					✓	✓				
Head and neck										
Parotidectomy					✓					
Thyroidectomy								✓		
Radical neck dissection					✓				✓	
Neurotology										
Vestibular schwannoma			✓		✓	✓	✓	✓	✓	✓
Auditory brainstem implant					✓	✓	✓	✓	✓	
Skull based/other										
Petroclival meningioma	✓	✓		✓						
Jugular foramen							✓	✓	✓	
Sphenoid wing	✓			✓	✓					
Parapharyngeal space					✓			✓	✓	✓
Microvascular decompression		✓	✓		✓	✓	✓			

[a]Procedure where electric auditory brainstem response measures may be useful.

monitoring (see vestibular schwannoma or microvascular decompression for suggestions about motor and sensory nerve monitoring). With due consideration paid to at-risk cranial nerves during otologic, head and neck, as well as skull-based procedures, certain pitfalls may be avoided, leading to fewer technical interruptions, improved surgical outcomes, and continued confidence in monitoring services.

PATIENT PREPARATION

Careful patient preparation and an understanding of the electrical environment in the operating room are key components for obtaining replicable recordings in the operating room. However, electrical artifacts that may obscure, distort, or mask the intended responses are not completely avoidable. In terms of their temporal characteristics, such artifacts may be thought of as continuous or transient in nature.

A constant electrical artifact or interference is a major problem in the operating room because it may result in an inability to monitor electrophysiological function by obscuring the intended signals. Such artifacts may originate from equipment that is poorly grounded or inadequately shielded and may originate in the host operating room or even in an adjacent room that shares electrical wiring. One of the reasons this type of artifact is extremely troublesome is because of its constant nature and because its source is not easily identifiable. The most common artifact is the 60-cycle interference that may take the form of a 60-cycle sign wave that masks all electrophysiological recordings or it may be a sign wave or a more complex wave composed of 60-cycle harmonics. Poor grounding of the patient or the neurodiagnostic equipment used for monitoring may cause such interference. Sixty-cycle interference is more likely when monitoring equipment shares an outlet with other electronic equipment used in the operating room such as patient warmers, sequential compression devices, electrocautery, operating microscopes, and ultrasonic dissecting devices. Therefore, it is desirable to make every attempt to identify and use a properly grounded outlet that is not shared with other equipment.

Constant or continuous interference may also take the form of very high frequency signal that may originate from monitor screens. Monitor screens are in abundance in many operating rooms associated with the anesthesia equipment, video equipment including monitors linked to an operating microscope mounted camera, monitors used to view imaging studies, or monitors linked to frameless, stereotactic neuronavigation instrumentation.

Proximity of neurophysiological monitoring leads to electrocardiography (EKG) leads may result in a continuous artifact. Given the relatively short time base used for neuromonitoring, this type of artifact may not look like typical EKG. However, if one is able to capture this activity in an extended epoch, the various peaks of the EKG waveform are recognizable.

These continuous artifacts may be resolved in a systematic manner. The monitoring team should be familiar with the characteristics of various types of artifacts unique to specific operating rooms. Often, these artifacts can be resolved by repositioning recording electrode leads, patient interface connectors, or preamplifiers. If the artifact causes an evoked potential such as the ABR to be unidentifiable, slight changes in the stimulation rate (even by one or two decimals) may improve the ability to record a response. Other times recording parameters may need to be changed, especially the cutoff settings of the high-pass or low-pass filters; this may apply to both the ABR and to free-running and triggered EMG modalities. A cautious approach to stimulating and recording parameter alterations is recommended: One must be mindful of parametric changes on monitored activity as they may impact the continuous interpretation of responses or the comparison of ongoing activity with previously recorded "baseline" activity.

Transient artifacts are common during surgical procedures but are generally less disruptive and easier to resolve. Such artifacts may be associated with the temporary use of specific powered surgical instrumentation such as electrocoagulation, a powered scalpel, or an ultrasonic dissecting device. However, it is important that everyone in the room understands that electrophysiological responses are obscured while such instrumentation is used, owing to the massive interference artifact created. If it is necessary to use these instruments for lengthy periods of time, it is useful to have an agreement with the surgical team to provide periodic breaks to allow effective neurophysiological monitoring.

Patient preparation also includes the setup, or the arrangement of monitoring electrodes, transducers, extensions, stimulators, headboxes, and amplifiers on or about the patient and the operating table. Table 16.2 provides suggested recording sites for selected types of surgical procedures in which the authors routinely participate. Using the placement sites in this table, and with the suggestions offered in this section of the chapter, the audiologist will have a significantly increased opportunity to standardize the IOM setup within and across cases. This will help to improve the likelihood that a patient's outcome will be acceptable for all members of the care team, providing that the audiologist brings to the operating room the requisite training, skill, and experience required to perform IOM.

ANESTHESIA

One of the fundamental components of a standardized, well-conceived approach to offering neurophysiological monitoring to a variety of surgical departments includes the ability to work in a collaborative manner with anesthesiology colleagues. Careful selection and delivery of anesthetic agents coupled with information shared between anesthesiologists and audiologists about the effects of anesthesia on neurophysiological modalities impacts the success of monitoring

TABLE 16.2

Suggested Recording Sites for Properly Conducted IOM

Cranial Nerve(s)	Recording Muscle Site(s)	Comments
Oculomotor (III)	Extraocular muscles: Medial, superior, inferior recti	Useful in surgery involving the cavernous sinus
Trochlear (IV)	Superior oblique	Access challenging
Trigeminal (V)	Masseter or temporalis	Helpful as adjunct to facial nerve monitoring in cases of large vestibular schwannomas; possibly useful in trigeminal nerve microvascular decompression surgery
Abducens (VI)	Lateral rectus	Useful in surgery involving the cavernous sinus
Facial (VII)	1. Upper division and lower division montage (frontalis, orbicularis oculi in pairs and orbicularis oris, mentalis in pairs, respectively) for otologic and neurotologic surgery 2. Temporal, zygomatic, buccal, marginal mandibular branches of extracranial distribution of facial nerve (frontalis, orbicularis oculi, orbicularis oris, mentalis muscles, respectively)	Arguably the most frequent monitored motor cranial nerve in otologic, head and neck, posterior skull base, pediatric, and neurosurgical operative procedures
Cochleovestibular (VIII)	Electrode montage: Vertex or nape of neck (active), shoulder or forehead (ground), ipsilateral ear anterior to tragus for auditory brainstem responses; tympanic membrane surface for electrocochleography; cranial nerve for direct nerve recording (various reference sites selected for individual or combined auditory-evoked potentials)	Useful in planned hearing preservation surgery as well as during microvascular decompressions, vestibular neurectomy, endolymphatic sac procedures
Glossopharyngeal (IX)	Soft palate, pharyngeal wall	Effective for neurosurgical procedures including mass lesions involving the lower cranial nerves
Vagus (X)	Vocal cords, posterior cricoarytenoid, vocalis muscles (recurrent laryngeal nerve branch)	Hookwire or surface electrodes on endotracheal tube; used during thyroid surgery or cases involving brainstem or lower cranial nerves
Spinal accessory (XI)	Trapezius or sternomastoid muscles	May be used during radical neck dissection, anterior approaches to cervical spine
Hypoglossal (XII)	Ipsilateral tongue, laterally	Used with IX/X setup in posterolateral skull base surgery

and patient outcomes. Primary goals for an anesthesiology service in the operating room include keeping the patient properly immobile and pain-free (Banoub et al., 2003). There are a host of anesthetic agents available to achieve those goals and many of these medications have the power to mildly or profoundly affect cranial nerve monitoring in either the motor or the sensory modalities. Thus, it is crucial for the monitoring team to have pre- or perioperative discussions with members of the anesthesiology team. It is important to avoid surprises during the operative procedure that could result in a reduced ability to provide neurophysiological monitoring or that may interfere with nerve monitoring for the entire case. Long-lasting neuromuscular blockades, such as doxacurium, pipecuronium, and tubocurarine, are contraindicated when monitoring schemes include the provision of free-running and triggered EMG. Intermediate effects resulting from the use of atracurium, cisatracurium, pancuronium, and vecuronium could be a problem if

TABLE 16.3

Pharmacologic and Physiologic Effects on IOM

Pharmacologic Agents	Effects
Modality: Electrocochleography	Resistant to anesthesia effects
Modality: Auditory brainstem response	
Inhalational agents (enflurane, halothane, isoflurane, etc.)	≥0.5–1.0 ms latency shift in wave V; inhibition on averaged responses varies among agents but can prolong wave I–V interpeak latencies with end-tidal concentrations >1.5%; children more resistant than adults
Injectable agents (lidocaine, thiopental, pentobarbital, propofol)	Depending on dosages, amplitude reductions, and increases in absolute latencies, particularly wave V are noted
Modality: Electromyography	
Neuromuscular blockade: Doxacurium, pipecuronium, tubocurarine have long-lasting effect; atracurium, cisatracurium, pancuronium, vecuronium have intermediate effects; mivacurium, rocuronium, succinylcholine are short-acting agents	Free-running (motor unit recruitment) and triggered activity (compound muscle action potentials) abolished for minutes to hours depending on the agent selected; each has dose-dependent effects which may be chemically reversed with anticholinesterase inhibitors
Local anesthetics (lidocaine, tetracaine, bupivacaine, etc.)	Amplitude reduction and delay in electrically evoked responses because of altered axonal propagation
Physiologic Events	
Local and/or systemic decreases in temperature	Effects noted in absolute as well as interpeak latencies of averaged auditory-evoked potentials, triggered electromyography
Compression of tissue from retraction, crush, etc.	Degraded or abolished waveform activity depending on structural tolerance of instrument pressure
Insufficient ventilation, hemodilution, systemic hypotension, regional ischemia	Decrease in oxygen supply to hearing end organ leads to reduced endocochlear potentials, impaired cochlear output, loss of averaged responses

After Edwards BM, Kileny PR. (2000) Intraoperative monitoring of cranial nerves. In: Canalis RF, Lambert PR, eds. *The Ear: Comprehensive Otology*. Philadelphia, PA: Lippincott Williams & Wilkins.

the surgical procedure quickly involves a monitored cranial motor nerve, as in the case of a pediatric revision mastoidectomy. Importantly, an anesthesiologist may be able to select or alter anesthetic techniques to facilitate monitoring while affording maximal patient safety (Nuwer, 2002).

In the majority of cases that the authors have monitored over three decades, surgical, anesthesia, nursing, and monitoring teams have worked in full cooperation to achieve good patient outcomes. Nonetheless, pharmacologic, physiological, and mechanical factors under the control of the surgeon and the anesthesiologist must be continuously scrutinized by the audiologist. Any changes in IOM related to the previously mentioned factors should be reported to the room so that interventions can be made in time to avert suboptimal results. For an excellent review of the topic of anesthetic effects on sensory and motor nerve activity, the reader is referred to the comprehensive paper by Banoub et al. (2003).

Table 16.3 offers a selected list of routinely employed pharmacologic agents available to anesthesiology members of the operating room team. These therapeutic drugs are commonly found in operating rooms, and their effects must be well known to the monitoring audiologist to avoid a scenario wherein monitoring is rendered useless.

ELECTROMYOGRAPHY APPLIED TO IOM

To understand the principles and clinical applications that can be provided by clinical EMG, one must be familiar with the anatomy and physiology of the nerve–muscle complex. Striate or voluntary muscle fibers have contractile properties and consist of myofibrils made up of numerous myofilaments composed of actin and myosin which are the building blocks of muscle tissue. A motor unit (Figure 16.1) is an entity consisting of a neuron and its axon divided into several synaptic terminals, each innervating muscle fibers at the myoneural junction. On activation, each synaptic terminal/muscle fiber generates an electric signal, the motor potential. The sum of motor potentials from numerous muscle fibers innervated by the same axon results in a larger motor unit action potential (MUAP). Thus, the MUAP represents the electrical activity of several muscle fibers innervated by the same motor unit.

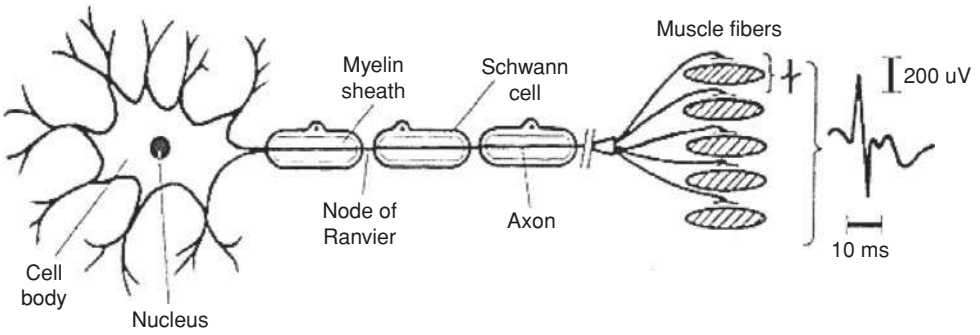


FIGURE 16.1 The motor unit.

How is one able to observe so-called "free-running" EMG during surgical procedures? For these electrical potentials to be generated, the axon innervating these muscle fibers must first be activated, or depolarized. This happens when a motor movement is initiated in the brain's motor cortex; muscle fibers innervated by the same axon generate single fiber potentials, and the sum of these single fiber potentials originating from the same axon results in a larger, single fiber potential we know as the MUAP. This response is recorded from the appropriate muscle using an intramuscularly placed needle electrode. The triphasic MUAP amplitude is determined by first, the number of single fiber potentials that constitute the motor unit potential and second, the proximity of the recording electrode to the source of this potential. The frequency or density of these MUAPs in a specific epoch depends on the magnitude of the muscular effort. Healthy, normally innervated muscle at rest generates no spontaneous activity; however, with increasing effort, motor units are recruited ultimately resulting in a high-frequency signal. As the magnitude of the voluntary effort diminishes, the recruitment pattern becomes less dense until at complete rest no motor units are seen. Figure 16.2 illustrates motor unit potential recruitment recorded from the orbicularis oculi muscle demonstrating initial muscle rest or inactivation, followed by an increase in effort of eye closure resulting in recruitment of motor unit activity, followed by relaxation of the orbicularis oculi muscle as motor units disappear. This figure illustrates the absence of motor unit potentials at rest and the recruitment of motor units present with effort. For neurodiagnostic purposes one can isolate a single motor unit potential and evaluate and examine its configuration. During intraoperative monitoring applications, one observes spontaneous or free-running EMG observing some of the applications and techniques just described.

Another component of neuromuscular function used intraoperatively is the compound muscle action potential (CMAP), a triggered response that is similar to an averaged evoked potential in that it requires a stimulus with the ability to stimulate or activate in a synchronized manner a relatively large number of MUAP. Unlike free-running EMG that depends on voluntary effort, so-called "triggered EMG" responses require that electrical pulses be delivered somewhere along or in proximity to a motor cranial nerve. This results in the contraction of muscle fibers innervated by this particular cranial nerve or a branch of the nerve. Here, one stimulates not individual axons but an entire nerve trunk. This CMAP response may be recorded using either intramuscular needle electrodes or surface electrodes; the stability of the latter option makes it impractical in the operating room where procedures can last for hours. The CMAP's configuration is a biphasic sine wave which is the result of the synchronized depolarization of a large number of axons

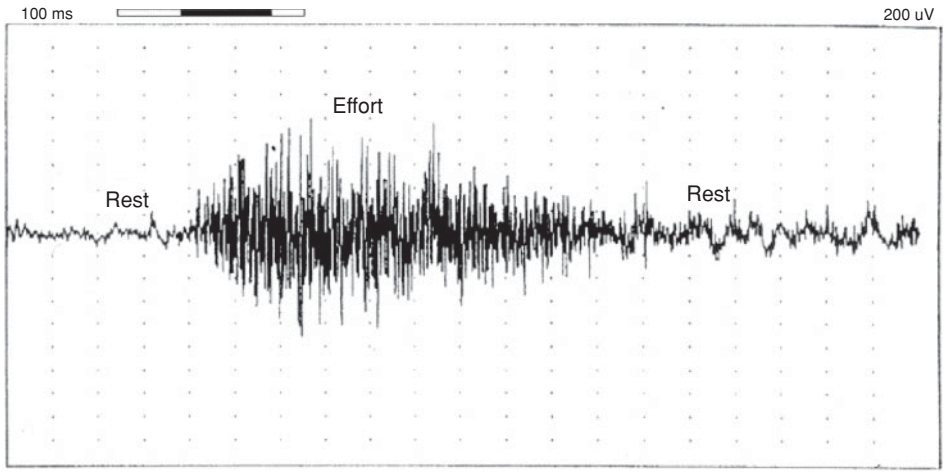


FIGURE 16.2 Recruitment of motor unit potentials.

resulting in the simultaneous activation of numerous MUAP. To measure this response for diagnostic purposes, the electrical pulse is delivered superficially along the facial nerve as it exits the stylomastoid foramen. The intraoperative application involves the use of a handheld surgical stimulator and direct electrical stimulation of the exposed motor cranial nerve (or tissue that may be in continuity with the target nerve). This specific component of EMG used in the operating room will be addressed in more detail in this chapter.

ANATOMY OF THE FACIAL NERVE

The facial nerve is physiologically and functionally complex as it courses through the posterior cranial fossa and the temporal bone before innervating peripheral facial muscles (Figure 16.3). Intracranially, the facial nerve emerges from the brainstem at the pontomedullary junction after receiving fibers from the facial motor and superior salivatory nuclei. After crossing the subarachnoid space of the cerebellopontine angle (CPA), the facial nerve passes into the internal auditory canal (IAC) joining the vestibulocochlear nerve. The intracranial segment of the facial nerve lacks an epineural sheath which is the fibrous sheath protecting the more peripheral segments of the facial nerve trunk. Consequently, even a healthy facial nerve which is not compressed and attenuated because of the presence of a tumor is at a relatively high risk for surgical injury because of this inherent weakness when compared to its more peripheral segments. The facial nerve traverses the IAC and then enters the fallopian canal of the temporal bone at the point where the fallopian canal has the smallest diameter—the meatal foramen. The facial nerve then makes several turns to form

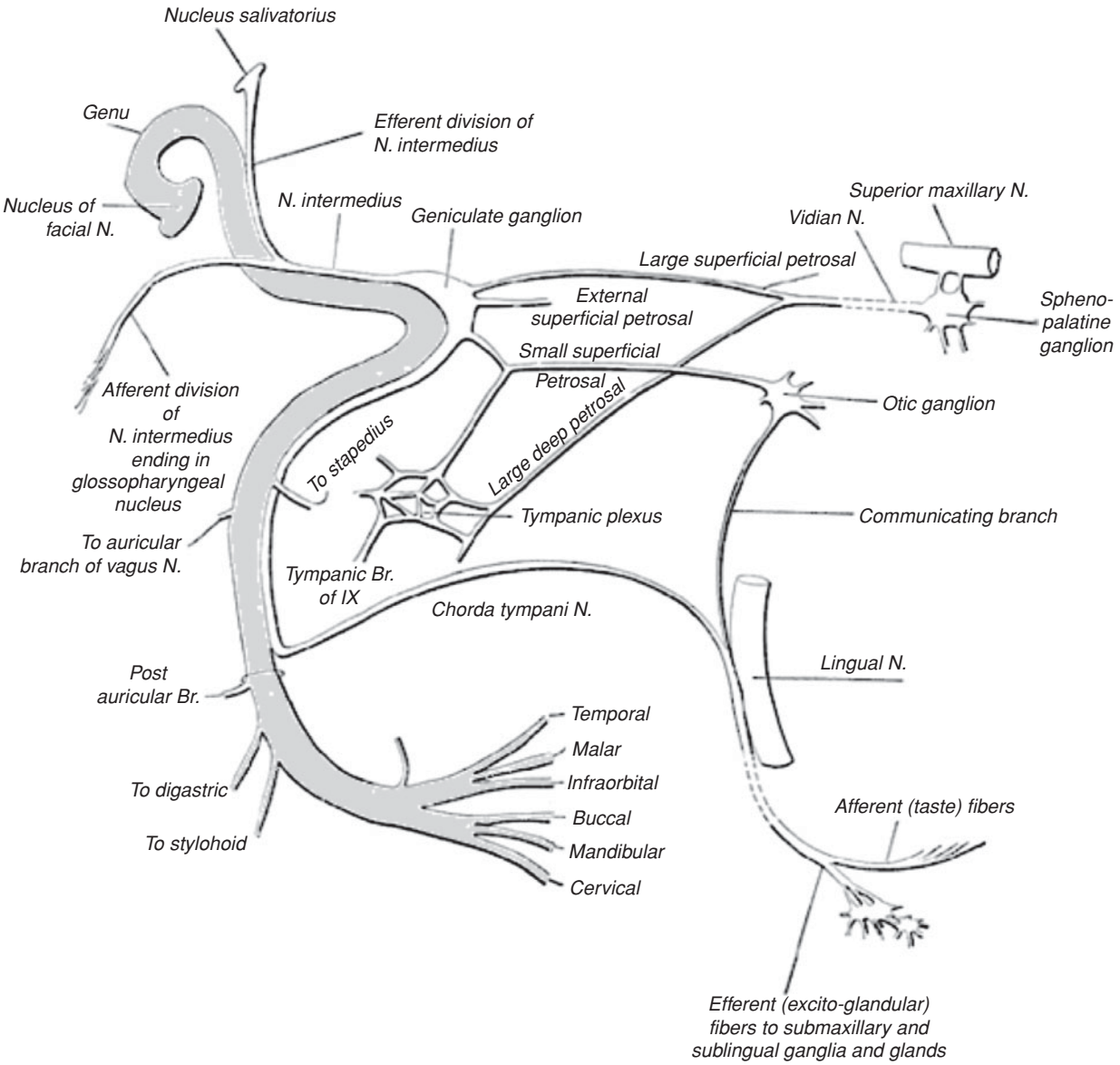


FIGURE 16.3 Course of the facial nerve from the pons to its distribution in the face.

the (1) labyrinthine, (2) geniculate, (3) tympanic, and (4) mastoid portions of the fallopian canal to exit from the skull base via the stylomastoid foramen. Following its exit through the stylomastoid foramen, the extratemporal segment of the facial nerve enters the parotid gland where it is contained in between the superficial and deep lobe of the parotid, and this is where it branches into several peripheral branches before it finally terminates on the motor end plates of the 16 muscles of facial expression on each side. The facial nerve also supplies the posterior belly of the digastric muscle, the stylohyoid muscle, and the stapedial muscle. As audiologists know, the acoustic stapedial reflex depends on an intact and functioning facial nerve. The facial nerve also provides parasympathetic innervation to the lacrimal, submandibular, and sublingual glands as well as to the mucous membrane of the nasopharynx and the hard and soft palate. Taste sensation for the anterior two-thirds of the tongue is also provided by the facial nerve as well as taste sensation to the hard and soft palates. The facial nerve also has a general sensory component providing general sensation for the skin of the concha of the auricle and a small area posterior to the auricle. Whereas the motor fibers of the facial nerve constitute the largest portion of the nerve, the remaining three components (the parasympathetic, the special sensory (taste), and general sensory components) are bound in a distinct fascial sheath and the branch of the facial nerve containing these components is referred to as the nervus intermedius.

Depending on the specific surgical procedure, facial or other motor nerve monitoring is directed to accomplish one or more of the following goals (Niparko et al., 1989):

1. early recognition and identification of impending surgical trauma to the nerve with timely feedback made available to the surgical team;
2. distinguishing the targeted cranial nerve from the adjacent soft tissue, tumor, or other cranial nerves;
3. facilitation of tumor excision by electrically mapping and confirming that the regions of the tumors are remote from the facial nerve;
4. confirmation of nerve stimulability following tumor removal; and
5. identification of the site and degree of neural degeneration in selected patients undergoing nerve exploration for suspected facial nerve neoplasm or decompression of acute facial palsy.

Events associated with intraoperative monitoring of cranial motor nerves are influenced by the preoperative status of the nerve and the anatomy and morphology of the nerve affected by a compressive tumor. Clearly, neurophysiological monitoring may have a limited role in cases with complete preoperative facial paralysis as determined by clinical exam and electrophysiological diagnostic measures (Kileny et al., 1999). In the presence of partial neurodegeneration, nerve-related monitored neural activity varies with the degree of ischemia and compression of the nerve. With compression, local irritation of the nerve results in increased sensitivity to mechanical stimulation that may be associated with surgical dissection, and thus the more likely appearance of spontaneous EMG activity. Demyelinating neuropathies are also associated with increases in spontaneous firing and mechanosensitivity of peripheral nerves attributable to the increased sodium channels present in the remaining myelinated nerve fibers (Bergmans, 1983). Increased spontaneous electromyographic activity associated with the mechanical stimulation of the facial nerve has been demonstrated in cases where the facial nerve was extensively involved with tumor (Prass and Luders, 1986).

COMMENTS REGARDING TRIGGERED ELECTRICAL STIMULATION OF CRANIAL MOTOR NERVES

Intracranial surgery in the vicinity of cranial nerves entails dissection through a complex array of tissues often surrounded by a fluid media that has the propensity to shunt the electrical current used for stimulation (Moller and Jannetta, 1984). Stimulus delivery to the nerve may be attenuated by that fluid, increasing the chance of false-negative stimulation. To counteract this problem, a constant voltage approach could be used in which voltage is unvarying but current changes according to the degree of stimulus shunting as determined by impedance encountered at the stimulator tip. However, because nerve depolarization is a function of electric current, most applications use constant current stimulation that varies voltage as a function of impedance to maintain a constant current at the tip of the stimulator (Prass and Luders, 1985). Additionally, the use of a probe that incorporates insulation to the end of the probe helps to provide controlled stimulus delivery across most conditions.

A concern when directly stimulating an exposed cranial nerve particularly in its intracranial course is whether direct delivery of electrical current to the nerve is in fact safe. Few recent studies have critically evaluated the safety of electrical stimulation of surgically exposed cranial nerves. Love and Marchbanks (1978) described the case of facial paralysis after acoustic neuroma resection possibly related to the use of a disposable, handheld nerve stimulator. In their paper, the site of nerve stimulator application was characterized as "blanched," suggesting that a cautery-like effect may have been exerted by the nerve stimulator. Further experimental studies carried out by these investigators in the rabbit model suggested that direct-current nerve stimulation produced neural damage by inducing thermal injury. Hughes et al. (1980) demonstrated that repetitive, prolonged direct-current stimulation produced significant myelin and axonal degeneration in the rodent model. In a follow-up investigation, Chase et al. (1984) demonstrated that the damaging

effects of direct-current stimulation were avoidable if the nerve and stimulator were in contact for brief periods.

PRINCIPLES OF MOTOR CRANIAL NERVE MONITORING

The audiologist needs to clearly identify the specific muscles innervated by cranial motor nerves that will undergo monitoring. For example, when monitoring the function of peripheral branches of the facial nerve, recording electrodes must be placed in each muscle of interest associated with these peripheral branches, that is, frontalis, orbicularis oculi, nasolabial, and mentalis muscles. Choices in electrode design allow one to select the most appropriate type of electrode for the operative case. For example, paired sets of intramuscular monopolar needle electrodes could be positioned at interelectrode distances best suited to the size and bulk of the target muscle. Or for very specific recording from a small volume muscle (such as intrinsic laryngeal muscles), one may utilize a concentric needle electrode or paired hooked wire electrodes.

When multiple muscle monitoring is required (the muscles innervated by the same or by various cranial motor nerves), one needs to ascertain that the instrumentation selected for IOM has sufficient numbers of channels for free-run and triggered EMG as each monitored muscle requires a separate channel to allow the clinician carrying out the monitoring to distinguish between individual muscles/innervating nerve branches. Another important prerequisite is for the provider of IOM to have a working knowledge of the pharmacologic agents available to be administered to the patient either as premedication or during surgery. As mentioned elsewhere in this chapter, long-acting neuromuscular blocking agents should be avoided unless contraindicated by the patient's medical condition. Although a short-acting paralytic agent used for patient intubation is acceptable, close communication with the anesthesiologist is very important to avoid an anesthetic course that includes neuromuscular blockade throughout the operation. This is an extremely important aspect of intraoperative monitoring. In many surgical situations, patients are paralyzed throughout the procedure to modulate the overall concentration of anesthetics provided. Therefore, one should not assume that an anesthesiology team will refrain from using neuromuscular blockade. Even a subparalytic dose used throughout surgery has the potential to decrease the sensitivity of intraoperative monitoring.

As stated previously, one of the goals of IOM of motor cranial nerve function is to assist in the avoidance of unintended injury to a particular cranial nerve that may be at risk because of its specific anatomic location, relative to the lesion operated on. When surgery is performed by a skilled and experienced surgeon, IOM can significantly assist in nerve function preservation. Nerve preservation depends on the following variables. Identification is extremely important if the surgeon is to successfully dissect tumor away from nerve. Often, significant anatomical distortions are caused by expansive intracranial masses and as a result, cranial nerves may not be located in their normal anatomical position or condition. Nerve identification can be accomplished by discrete intracranial electrical stimulation of soft tissue structures near the cranial nerve of interest. This is especially important if the nerve has been compressed and attenuated by the expanding tumor. Electrical stimulation of the targeted cranial nerve(s) will produce a CMAP recorded by the intramuscular electrode(s) associated with a given cranial nerve. Stimulation of other structures should not result in the production of a CMAP. Figure 16.4A is a schematic representation of a situation involving a tumor (a), an attenuated nerve running along the tumor capsule (b), and the peripheral portion of the nerve trunk (c), within an innervated muscle (d). In the figure, paired intramuscular electrodes in muscle (d) display intramuscular activity on the neurodiagnostic monitor (e). When a nerve stimulator is used to deliver low-level electrical current pulses to the tumor capsule near the nerve but not quite touching the

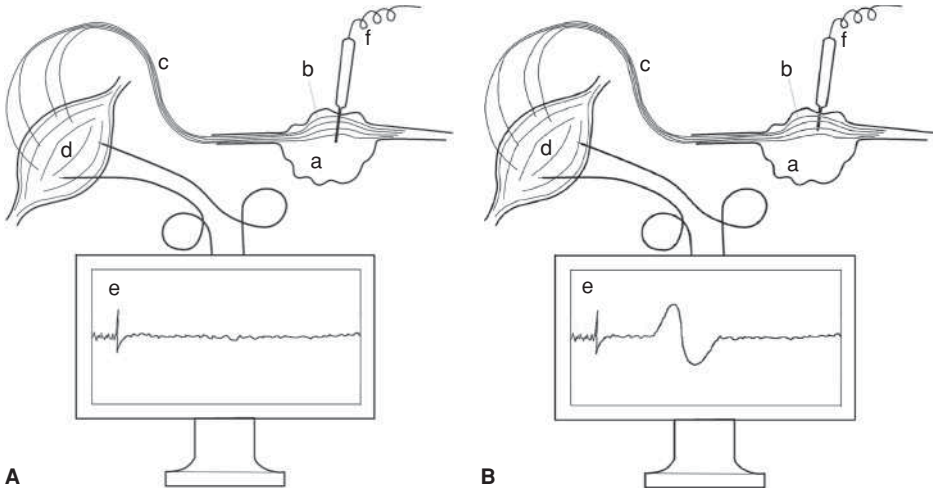


FIGURE 16.4 **A:** Schematic depicting no response to electrical stimulation. Note only the stimulus artifact on the monitor. **B:** Schematic depicting a response to electrical stimulation in the form of a compound muscle action potential that follows the stimulus artifact on the monitor.

nerve (f), the screen of the neurodiagnostic equipment continues to show baseline activity with the exception of the spike near the onset of the trace representing the stimulus artifact. No response to stimulation is present, and that specific interpretation made by the audiologist would be reported to the surgical team immediately. In Figure 16.4B, nerve stimulation via the probe (f) is applied directly to the nerve and elicits the CMAP as displayed on the screen of the neurodiagnostic equipment. The biphasic CMAP waveform occurs after the stimulus artifact spike and represents a positive response to stimulation which would be reported to the surgeon.

This simple example illustrates the principle of utilizing intraoperative monitoring to identify cranial motor nerves as distinguished from surrounding tissue. It is of note that with monopolar stimulation, in the setting of an operative field which contains fluid, especially with higher levels of current stimulation, the electrical current could spread to adjacent tissues away from the tip of the stimulus probe. A "false positive" could occur, that is, a surgeon could apply the stimulator to soft tissue that does not contain targeted nerve but is fairly close to the nerve and at higher intensity one could conceivably obtain a CMAP that would suggest that the stimulated site was in fact a cranial nerve. Therefore, caution is advised relative to current levels used for intracranial stimulation in particular if the stimulated site is anatomically closed to the presumed anatomical site of the target cranial nerve. With appropriate attention paid to stimulus delivery, excellent reliability can be accomplished. Specificity and reliability are important in IOM because an absent response to electrical stimulation is as significant as the presence of a response. An absent response indicates to the surgeon that this could be a safe area to dissect without risking the continuity or the function of the motor cranial nerve. Therefore, providing accurate, timely information to the surgical team is extremely important and will help to optimize patient outcomes.

Another important aspect of motor cranial nerve monitoring is providing feedback to the surgical team regarding inadvertent "mechanical" stimulation of the targeted cranial nerve that may result in temporary or permanent damage to the nerve and dysfunction for the patient. This can occur in situations where a cranial nerve has been identified and is within plain view in the surgical field or in a situation where the cranial nerve is not visible within the surgical field. As mentioned earlier, healthy muscle at rest is quiescent and both voluntary contraction and mechanical manipulation of the motor cranial nerve will give rise to motor unit potentials in a variety of patterns. Notably, a motor cranial nerve that has been compressed and/or attenuated can be more sensitive to mechanical manipulation than a cranial nerve in its normal condition. It is of note that completely harmless intraoperative events, such as cold irrigation in the vicinity of a cranial nerve, can also trigger a salvo of motor unit potentials. These motor unit "trains" are recorded on free-running EMG channels of the monitoring equipment. Thus, baseline or free-running EMG activity must be monitored continuously throughout the surgical procedure. This is different than triggered EMG stimulation used to identify or track the course of neural tissue in the operative field. In the latter case, one activates the stimulator when the surgeon requests to stimulate the nerve or adjacent tissue.

During continuous monitoring of free-running EMG, when a pattern of motor units appear in the course of surgery, this event needs to be communicated quickly to the surgical team. It may indicate possible proximity to the nerve of interest and/or potential damage to the monitored nerve. It is important to note that the appearance of these motor unit potentials do not necessarily indicate that the nerve has been injured but simply that it has been stretched or compressed mechanically in the course of dissection; continued surgical manipulation of this nature may result in temporary or permanent injury. This is particularly important in a situation in which the target cranial nerve has not yet been visually identified. Figure 16.5A,B schematically illustrates surgical dissection of a tumor while employing continuous IOM methods. In Figure 16.5A, the screen of the neuromonitoring equipment exhibits a quiet,

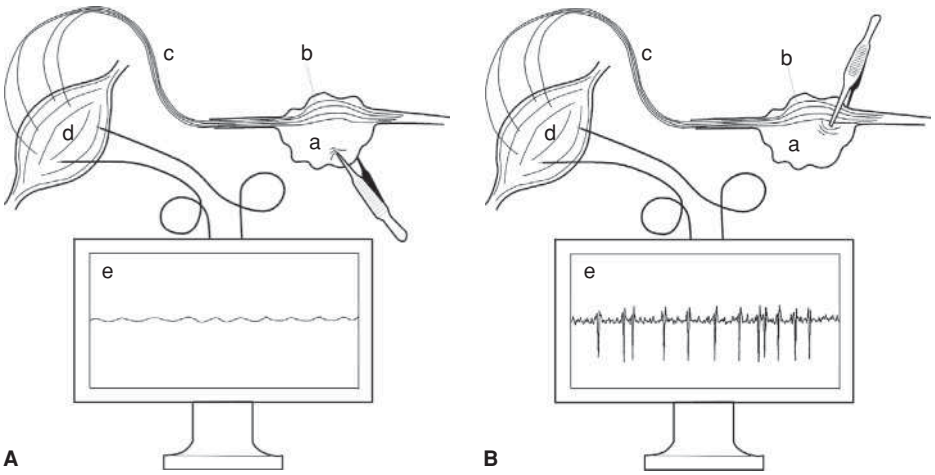


FIGURE 16.5 **A:** Baseline electromyography that illustrates a quiet free-running recording. **B:** Twelve motor units are observed in this depiction of an electromyographic change from the quiet baseline.

unchanged EMG baseline as tumor is manipulated without disturbing the cranial nerve with which the tumor may be in contact. Figure 16.5B demonstrates traction in the vicinity of the cranial nerve that results in the appearance of many motor units.

Importantly, after the audiologist observes and reports this acute change in activity, the surgeon may choose to reverse the maneuver that led to the decrement. For example, cessation of traction may produce a complete or an incomplete return to the previously observed quiet "motor-unit–free" baseline. Mechanically induced motor units may persist after cessation of the initiating maneuver and may even progress to a recurring train activity, consistent with muscle spasm. This may suggest that unintended injury has occurred or has the potential to occur. A brief pause in the surgical procedure may be recommended until a reduction in the spasm activity is observed.

Ideally, one should have the ability for direct electrophysiological recordings of facial nerve activation and responses to be used as indicators of direct or indirect mechanical manipulation of the nerve, to detect conditions that may lead to nerve injury, and finally to assist in the localization, identification, and mapping of the facial nerve. However, direct recording from cranial nerves during surgery is impractical presently and therefore, electromyographic recording of innervated muscle activity is the electrophysiological method used for intraoperative monitoring. Free-running facial muscle activity is recorded by means of intramuscular needle electrodes and multiple channels of electromyographic activity may typically be sampled. Depending on the desired sensitivity and specificity, one may record EMG differentially from a muscle using paired monopolar electrodes. Smaller interelectrode gaps (electrodes placed closer to one another) produce higher specificity of recorded activity from the target muscle. If the distance between the recording electrodes is relatively wide, the specificity may diminish but sensitivity may increase due the ability to detect activity within a broader muscle mass.

Importantly, a healthy, resting muscle is electrically quiet, that is, there is no spontaneous baseline activity present. Muscle contractions manifest in the appearance of motor unit potentials signaling mechanical manipulation of the target cranial nerve. Thus, nerve manipulation that is associated with certain mechanical forces such as blunt dissection, compression, or stretching may result in a burst of motor unit potentials. These surgeon-induced mechanical forces do not have to be exerted directly on the nerve. Indirect application of force such as with dissection of a tumor capsule adherent to a facial nerve may result in a pattern of activation similar to when a cranial motor nerve is directly manipulated.

Triggered electrical stimulation may also be used to map the course of a motor nerve or to determine the functional status of a nerve intraoperatively. Direct nerve stimulation produces a single CMAP that is synchronized with electrical stimulus pulses delivered by the surgeon using a handheld probe. The configuration of this response is typically biphasic and does not require signal averaging. For example, direct electrical stimulation of the facial nerve is often used to identify and map the course of the nerve and can provide information regarding the functional status of the nerve before, during, and after the dissection. Repeated measures obtained during surgery may reveal alterations in the amplitude of the CMAP, or the stimulus level required to elicit a response, and may be useful in determining the maintenance of the targeted motor nerve.

INTRAOPERATIVE MONITORING OF AUDITORY FUNCTION

The IOM plan developed for each patient begins well before surgery. For example, in the setting of neurotological surgery to treat mass lesions in the IAC and/or the CPA members of the surgical and intraoperative monitoring teams develop a surgical plan based on the size and appearance of the mass lesion and the patient's hearing and facial function. Puretone air and bone conduction thresholds, word recognition scores, immittance measures including stapedial reflexes, otoacoustic emissions, and ABR should be components of the preoperative study. For patients suspected to have facial nerve involvement, assessment of facial function may help to form the IOM plan. A clinical assessment using a measure such as the House–Brackmann Facial Nerve Grading Scale (House and Brackmann, 1985) coupled with electrophysiological measures such as ENOG and/or facial EMG is a useful measure to complete before formulating an IOM plan.

The compressive effects of mass lesions in the IAC or the CPA on neural or vascular components that supply sensory cranial nerves can include diminution or loss of function of the involved sensory system. For example, in the IAC an expanding lesion may compress the vestibulocochlear nerve, labyrinthine artery, and/or the facial nerve. The effect of compression of the eighth cranial nerve or cochlear blood supply may be evident in higher frequency hearing thresholds, word recognition scores, the strength or presence of otoacoustic emissions (Kim et al., 2006), and prolongation or diminution of ABR wave III or wave V components that have neural generators medial to the IAC. Those effects can be related to the source of the lesion, that is, inferior or superior vestibular nerve, facial nerve, meninges, and so on (Hirsch and Anderson, 1980). Because neither hearing function nor auditory-evoked responses are easily predicted by the size of a vestibular schwannoma (Badie et al., 2001; Sunderland, 1945) preoperative baseline studies of hearing and auditory-evoked potentials obtained just days prior to surgery help to identify the practicality of planned hearing preservation surgery and also help to modify the plan to perform intraoperative monitoring of eighth cranial nerve (Schwartz and Morris, 1991).

If the plan includes hearing preservation, AEPs obtained in the operating room before the opening incision take on a different role. In this case, the goal is to obtain and store a "baseline measure" against which subsequent measures are compared. To obtain this baseline measure recording and stimulating parameters should be manipulated to achieve the best recording possible prior to incision. For this reason, knowledge of the software associated with the equipment used to perform neurophysiological monitoring and ability to manipulate recording and stimulating parameters are critical skills of the monitoring audiologist.

Auditory nerve monitoring performed during surgery may include electrocochleography (ECOG), ABR, or direct nerve recordings. Each modality can be affected by either pharmacologic agents or physiological changes experienced by patients during operative procedures. Table 16.3 describes pharmacologic and physiological effects on IOM with which the audiologist should be familiar to be able to properly interpret IOM activity obtained over many hours of IOM. Properties such as temperature, hypoxia, hypotension, and hemodilution can impact evoked potential measures obtained in the operating room. For example, mild hypothermia, known to hamper synaptic transmission and nerve conduction as a result of reduced enzymatic reactions along the nerve pathway, may be demonstrated by variable response amplitudes and delays in conduction (Blair, 1965; Kileny et al., 1983). Conduction latency is less evident if the neural pathway has relatively fewer multiple synapses. This may explain why ABR peaks I, III, and V are delayed in the presence of relatively mild systemic cooling. Notably, effects of cooling can be cumulative; later components are delayed to a greater extent than the earlier ones. When extreme low core temperatures are encountered, averaged responses disappear (Banoub et al., 2003).

As mentioned previously, regional hypothermia may occur with prolonged exposure to a cold operating room or with use of cold irrigation fluids. Temperature management of room air and irrigation fluids can be effective in controlling their effects on auditory-evoked potentials. It is recommended that the monitoring audiologist periodically check with anesthesiology regarding a patient's vital signs including core temperature (Schwartz et al., 1988).

Mild hypoxemia, or a reduction of oxygen supply to tissue below physiological levels, is typically less apparent in the ABR. However, sudden, severe hypoxic or ischemic events can manifest in the loss of the ABR as a result of failure of the cochlear blood supply (Sohmer et al., 1982, 1986). This can occur because of encroachment of tumor on the auditory nerve or during surgery when cochlear blood supply is compromised during tumor removal.

Another common problem in IOM is masking of the auditory stimuli by the operating drill (Legatt, 2002). This predictable event is minimized or eliminated when the drilling stops or with reduced drilling periods. It is important to share this information with the surgeon to decrease the opportunity for unacknowledged injury. Although the auditory brainstem response is relatively resistant to anesthetic and anesthetic pharmacologic agents, it is not completely unaffected by these agents. Therefore, during intraoperative ABR monitoring, one must recognize the pharmacologic agents administered to a patient to sort out surgical versus nonsurgical changes in latency and amplitude. Brief, direct, and regular communication is key to avoiding unintended, potentially permanent change in monitored activity and associated hearing loss.

Intraoperative monitoring of auditory function examines the status of the cochlea, eighth cranial nerve, and at least several of the brainstem nuclei defining the auditory pathway and is typically used in otologic, neurotologic, and neurosurgical procedures when hearing preservation is a goal by detecting acute changes in auditory function and discussing such events with the surgical team before these changes become permanent. Typically, the surgical team takes some corrective action to avoid adverse effects.

As previously discussed, it is crucial to know the results of a patient's preoperative audiologic examination. To lessen the chance that a patient's auditory function will change significantly before surgery, these measures should be performed shortly before the surgical date. Results of the electrophysiological measures should be used both to determine the feasibility of monitoring the auditory pathway and to plan for the most effective way of carrying out eighth nerve monitoring. Notably, auditory electrophysiological activity can represent peripheral and central auditory function depending on the type of auditory-evoked potential that will be monitored. For instance, ABR combined with ECOG provides information about the cochlea, cochlear nerve function (peaks I and II), and brainstem activity via waveform peak III (cochlear nuclei), waveform peak IV (superior olivary complex), and wave V (nucleus of the lateral colliculus) if one can record each of those component parts during surgery.

The ABR is unquestionably the most frequently used method for monitoring eighth nerve function. The advantage of ABR monitoring is that it is more representative of activity generated from the auditory pathway than other methods, at least to the level of the midbrain. A disadvantage is that signal averaging required to record the ABR imposes a time delay between a potentially adverse intraoperative event and its detection. However, with the patient under general anesthesia, use of a faster stimulus rate (31 to 40 sweeps/second) produces an averaged response in less than 30 seconds. We routinely observe that during surgical manipulations in the vicinity of the eighth cranial nerve, response desynchronization or deterioration of the averaged response may occur with little notice, underscoring the importance of rapid averaging.

Auditory nerve function may also be monitored in actual time by directly measuring the compound action potential (CAP) utilizing a sterile electrode in the form of a saline-soaked, cotton wick attached to a thin wire electrode

placed on the proximal cochlear nerve by the surgeon and retained via careful positioning of the metallic portion of the electrode. Direct nerve monitoring requires access to an exposed cochlear nerve and successful recording of the CAP may be related to the size of the mass lesion. In cases of larger tumors, the electrode can be placed only after the mass in the CPA has been resected, exposing the cochlear nerve. In cases of smaller tumors, with less involvement of the cochlear nerve, access is simpler. Advantages of direct nerve monitoring include immediate averaging owing to the amplitude of the response related to the proximity of the electrode to the neural generator.

Further, real-time activity recorded from the cochlear nerve provides information regarding the distal auditory nerve and cochlear function. This is valuable in the setting of intracanalicular acoustic neuromas which are located further from the recording site. For instance, if blood flow to the cochlea decreases because of the intraoperative compression of the labyrinthine artery, a change in the directly recorded CAP could be seen and immediate feedback to the surgeon might lead to rapid corrective action prior to an irreversible ischemic change to the cochlea. A shortcoming of direct nerve recordings is that if not used in conjunction with ABR, changes in auditory pathway function beyond the cochlear nerve may not be detected. Another option open to the audiologist in the operating room is to record electrocochleographic activity using either a transtympanic needle electrode or a tympanic membrane surface electrode. This method provides an enhancement of the nerve action potential after brief periods of signal averaging. Fewer sweeps are required to obtain this response when compared to the ABR owing to its closer proximity to its neural generator and the associated improvement in signal-to-noise ratio. However, it too should be provided in combination with the ABR to observe the distal and the more proximal auditory nerve intraoperatively. Figure 16.6A,B displays portions of IOM results obtained during similar surgical procedures performed on two different patients with an aim in each case to preserve hearing function during resections of acoustic neuromas that measured approximately 1 centime in size. Each IOM setup included ECOG and ABR to monitor distal as well as proximal eighth nerve activity, respectively. In Figure 16.6A, observe the presence of responses throughout the monitoring period represented in the curve stack for both ECOG and ABR. The epoch includes the last portion of activity recorded during the hearing preservation for this case; the ABR and the ECOG were maintained throughout tumor removal, and hearing was successfully preserved. In contrast, Figure 16.6B demonstrates

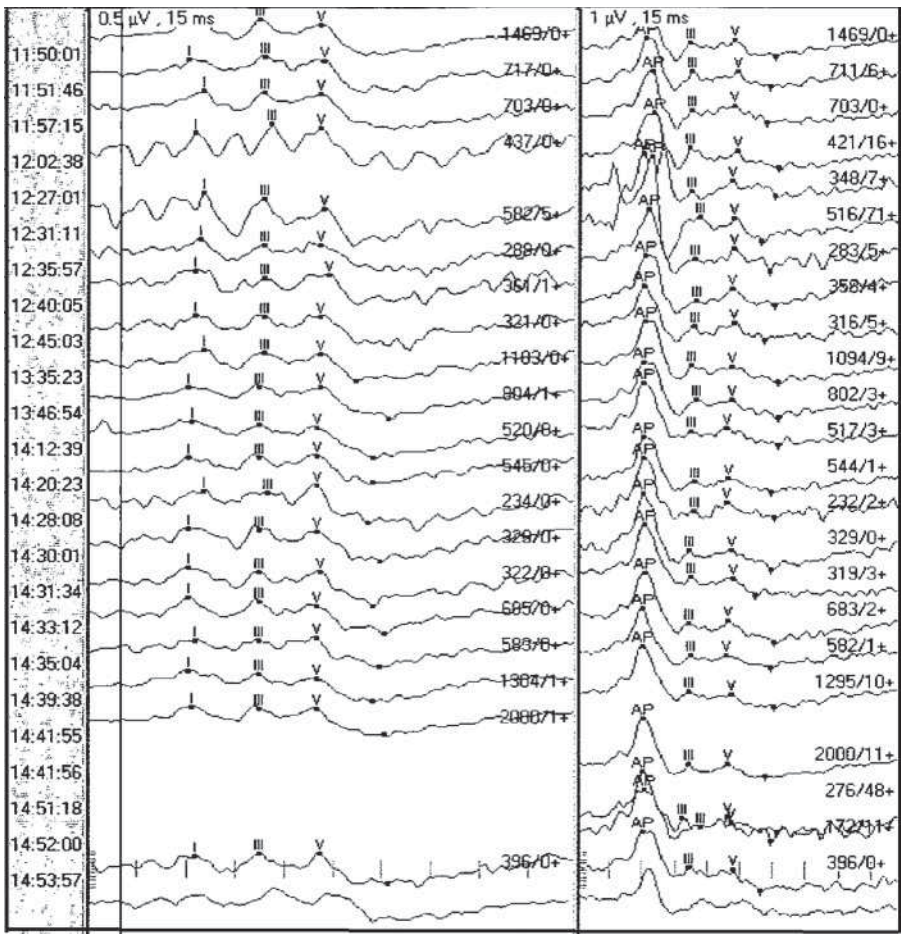
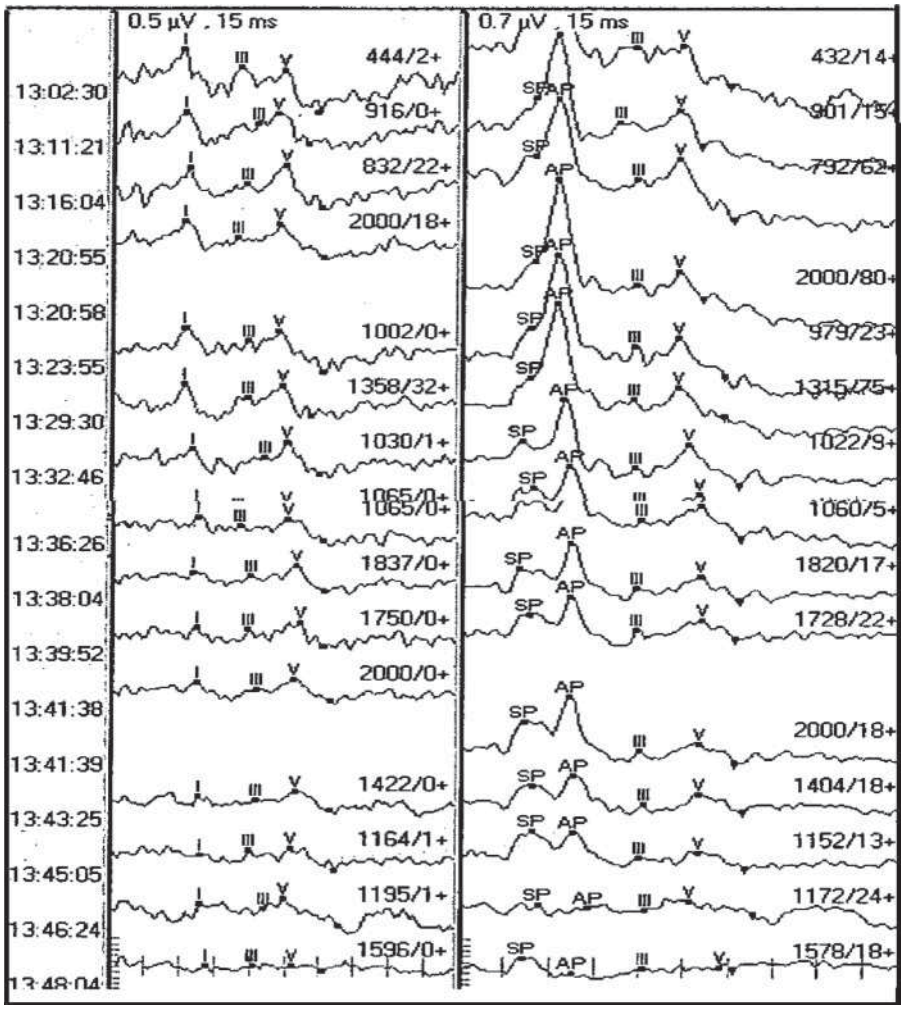


FIGURE 16.6 **A:** A stack panel of auditory-evoked potentials including the auditory brainstem response and electrocochleography. Individual waveform components are present at the conclusion of the case (*bottom of the panel*), suggesting preserved hearing function was achieved for this patient.



B

FIGURE 16.6 [*Continued*] **B:** A stack panel of auditory-evoked potentials includes the auditory brainstem response and electrocochleography. Individual waveform components are absent at the conclusion of the case [*bottom of the panel*], suggesting preserved hearing function was not realized in this case.

consecutive recordings of ECOG and ABR responses during a similar hearing preservation procedure that did not result in preserved hearing function postoperatively. Note in the left-hand column changes in the ABR wave I amplitude beginning at 13:32; in the right-hand column, changes in the ECOG's AP amplitude are observed as well. By the end of the surgical dissection, ABR responses are essentially absent whereas the ECOG AP response was absent as well. Unfortunately, the patient had no measurable hearing postoperatively, presumably related to an acute interruption of cochlear blood supply during the very last portions of the surgical intervention.

THE DEVELOPMENT AND MANAGEMENT OF AN INTRAOPERATIVE MONITORING PROGRAM

Intraoperative monitoring requires a team approach with the surgeon, anesthesiologist, operating room nurse or technician, and audiologist contributing their respective skills while working toward a goal of excellent patient outcomes. Although members of this team have different roles and training, each must understand the others' set of skill and role. For instance, the audiologist who may be responsible for performing the necessary preoperative audiological evaluations, planning and arranging neurophysiological monitoring, and recording, monitoring and interpreting the pertinent events must also have a thorough understanding of the surgical procedure. The audiologist must know the surgical anatomy and surgical indications for taking corrective action when trauma is imminent. The audiologist must be inquisitive and have a good working knowledge of the effects of pharmacologic agents on the electrophysiological events to be monitored in the operating room and must collaborate closely with the anesthesiologist or nurse anesthetists to properly decode response changes associated with alterations in anesthetic regimen. It is crucial to have adequate knowledge and preparation to be able to differentiate between changes in intraoperative monitoring events related to anesthetic intervention and those related to surgical events.

Differences exist across institutions regarding the specific models of intraoperative monitoring that the institution will

support financially and administratively. Therefore, before initiating such a program there should be a clear understanding among all stakeholders as to what the program will require, what will it consist of, financial implications, staffing, and staff preparation. All members of the operating room team need to be well informed about plans for this program and be aware that introducing IOM into the operating room may change the routine of a number of people to some degree. The unannounced appearance of an IOM work station in an already-crowded operating room may create problems. Similarly, a last-minute request to the anesthesia team about avoiding the use of neuromuscular blockade might also be a problem if, for example, a patient's medical condition imposes limitations in terms of the type and dosage of anesthetic agents used. Thus, the value of an intraoperative monitoring program lies in the preparation of all of the individuals involved in or affected by this practice. This also requires an understanding of the needs of the particular patient and the limitations of other professionals involved. Therefore, specific protocols for specific types of surgical procedures requiring intraoperative monitoring are necessary for a given institution. The standards and criteria associated with each protocol will assist the clinician carrying out intraoperative monitoring to provide a level of service that will maximize surgical outcomes.

Clearly, program differences in the IOM model exist across institutions. The "continuously attended" model involves someone other than the surgeon to prepare the patient, record pertinent neurophysiological activity and interpret activity within the context of the surgical procedure, and, importantly, provide specific feedback regarding changes in the monitored activity that might result in specific changes in surgical technique or approach. The intraoperative neuromonitoring clinician needs to have the ability to observe the surgical procedure (typically on a monitor connected to a microscope mounted camera in most microsurgical procedures) and correlate that activity with neurophysiological events. For instance, in the case of motor cranial nerve monitoring, continuous observation of spontaneous EMG and the immediate interpretation of any changes associated with mechanical manipulation of the motor component of the cranial nerve are important, because those events may be relevant to the prevention of postoperative paralysis or paresis. Additionally, one should follow established criteria that quantitatively assess the electrically activated CMAP as a predictor of postoperative functional status. Similar principles apply to sensory-evoked potential monitoring that may utilize ABR, ECOG, or direct cochlear nerve measurements.

Another model of intraoperative monitoring, arguably the most frequently used method, is the so-called "black box" or "remote" technology. This approach is utilized exclusively for monitoring the function of motor cranial nerves and also involves the recording of EMG events by means of intramuscular electrodes. The EMG activity is routed to an instrument that is intended to provide acoustic alarms upon mechanical manipulation of the target nerve. A similar acoustic alert signals the presence of a CMAP response associated with electrical nerve stimulation. In this model, no audiologist observes or interprets the various events occurring during surgery. Instead, the surgeon is required to attend to the alerting sounds and must learn to discriminate between acoustic alarms that signal either a baseline change in the free-running EMG associated with surgical events or spurious noise emanating from contact between noninsulated instruments touching in the operative field. The surgeon also needs to distinguish between acoustic signals that represent just stimuli being delivered about three times per second and the sound of effective nerve stimulation resulting in a CMAP. In this paradigm, the onus for proper and immediate interpretation of monitoring events belongs to the surgeon. The recognition of a particularly adverse situation is typically based solely on the perception of a change in acoustic output from the monitoring equipment's speaker. Some devices provide a screen that displays monitored activity or responses to stimulation in various ways. However, with attention properly focused on the operative field, the visual representation of activity on a screen is typically disregarded.

In addition to a decision regarding the type of monitoring employed, that is, continuously attended versus remote or nonattended, another decision that needs to be made beforehand concerns the type of procedures that will be monitored. In other words, what surgical service(s) will be supported by the IOM program? Among other things, this decision will impact the size of the monitoring staff, the type and level of training of that staff, and the type of neuromonitoring equipment purchased to support the service. For instance, if major skull base cases will be monitored requiring multiple channels available for cranial nerve monitoring, devices that allow for that level of activity must be sought out, purchased, and importantly supported for the life of the equipment. Decisions about surgical monitoring also impact the type of disposable supplies used including acoustic transducers, types of electrodes, and special EMG endotracheal tubes. There are multiple types of surgeries within the broader specialty of otolaryngology-head and neck surgery that benefit from monitoring. They include pediatric, otologic, and head and neck procedures involving the parotid gland or thyroid gland, and/or neck dissections.

Importantly, in 1992 and again in 2004, the National Institutes of Health published consensus statements based on expert testimony summarizing management options including facial nerve monitoring during surgical treatments for vestibular schwannoma (Edwards and Kileny, 2005). The American Academy of Otolaryngology-Head and Neck Surgery (1998) concurred with the value of facial nerve monitoring and noted that competently performed IOM of the facial nerve is effective and minimizes the risk of injury. Greenberg et al. (2002) sampled 500 members of the American Academy of Otolaryngology-Head and Neck Surgery regarding the value of facial nerve monitoring. Two

TABLE 16.4

Selected Elements of a Standardized Intraoperative Cranial Nerve Monitoring Service

I. Intended procedure
  A. Surgical service
    1. Otology
    2. Head and neck
    3. Skull base
    4. Pediatric otolaryngology
    5. Neurosurgery
    6. Thoracic/endocrine/general
  B. Estimated procedure duration
  C. Operating room characteristics
    1. Physical dimensions
    2. Access to single-use supplies
    3. Location of monitoring and surgical teams
      a. Sight lines
      b. Communication obstacles
II. Recording Issues
  A. Electrodes
    1. Surface, subdermal, tympanic membrane surface, promontory needle, EMG endotracheal, hookwire
    2. Matched impedances
    3. Patient ground
  B. Monitoring system components and parameters
    1. Amplifiers
    2. Filters
    3. Averaging mode
  C. Artifact issues
    1. Isolating source
    2. Comprehending presence/magnitude in ongoing activity
III. Stimulation issues
  A. Trigger source
    1. Internal versus
    2. External
      a. Synchronization with sweep cycle
  B. Electrodes
    1. Exposed tip, flush tip
    2. Lead extensions
  C. Electrical stimulation
    1. Monopolar versus bipolar
    2. Constant current versus constant voltage
    3. Stimulus increment, intensity range, duration rate
  D. Auditory stimuli
    1. Transducers and fixation
    2. Stimulus type: Click, tone, filtered noise
    3. Envelope: Blackman, Gaussian, Hanning, etc.
    4. Stimulus increment, intensity range, polarity, duration, ramping, plateau
IV. Anesthesia issues
  A. Use of agents before and during surgical procedure
  B. Ability/willingness to use reversing agents to improve ability to perform monitoring
  C. Communication methodologies to avoid unintended/suboptimal patient outcomes
V. Monitoring hardware
  A. Averager
    1. Multiple, independent time bases to support multimodality recording
    2. Variable display sensitivity
    3. Flexible mode designation to estimate noise
  B. Monitor display
    1. Multiple waveform display necessary
    2. Advanced information environment
      a. Waveforms, cursors, text, graphics, remote communication supported
  C. Grounding, safety procedures
    1. System ground connection via line power cord
    2. Leakage current-to-ground measures
    3. Estimate leakage current at patient isolation (amplifier)
    4. Electrosurgical unit(s) to return electrode positioned correctly
  D. Physical location in room
    1. Acoustic (speaker) electromyographic activity available to room
    2. Ability to connect monitoring equipment to microscope or to see feed from operating microscope on room monitor(s)
    3. Room noise controlled to facilitate communication with surgical, anesthesia, nursing teams
VI. Interpreting monitored activity
  A. Quantifying reproducibility to distinguish acceptable degrees/characteristics of variability
  B. Recognizing and reporting significant changes in activity compared to baseline reference
  C. Recognizing false-negative, false-positive events
  D. Consistency in marking waveforms
VII. Data management
  A. Storage medium: Hard drive, disk, tape, etc.
  B. Printed copy of events, reports, logs
    1. Deadline for report entry into electronic medical record
    2. Individualized versus canned formats
      a. Supplemental material inclusion
    3. Monitoring report components
      a. Preoperative patient history and/or study results
        1. Physical examination
        2. Tests of function (audiologic, facial, vestibular, etc.)
        3. Tests of structure (computerized tomography, magnetic resonance imaging, radioisotope injection, etc.

After Edwards BM, Kileny PR. (2000) Intraoperative monitoring of cranial nerves. In: Canalis RF, Lambert PR, eds. *The Ear: Comprehensive Otology*. Philadelphia, PA: Lippincott Williams & Wilkins, used with permission.

hundred and twenty-three of these respondents were private practitioners. Within that group, 82% of self-described otologists use facial nerve monitoring as compared with 50% of self-reported general otolaryngologists. Seventy-six percent of all respondents reported that they had access to facial nerve monitoring when desired. The more frequent users of facial nerve monitoring were employed in academic medical centers and were more recently trained or were subspecialized otologists. Most did not advocate mandatory monitoring for chronic ear surgery. Wilson et al. (2003) examined the cost-effectiveness of facial nerve monitoring in middle ear and mastoid surgery. The primary outcome measure selected by these authors was the increased cost per incremental quality adjusted live year (QALY) saved. The decision matrix examined cost-effectiveness of facial nerve monitoring in three cohorts of patients: Those who receive facial nerve monitoring for all middle ear mastoid surgery, patients who had selective facial nerve monitoring, and patients who received no monitoring at all during their surgery. QALYs were obtained by multiplying life expectancy with estimated utility of patients living with a facial paralysis. Calculations were made using a self-evaluation tool for patients with facial paralysis (Kahn et al., 2001). Results strongly favored facial nerve monitoring for any patients undergoing middle ear and mastoid surgery. A cost range of approximately $223 to $528 associated with the provision of IOM was reported. Rank-ordering of QALY supported a strategy to monitor primary and revision surgery versus either revision only or no monitoring scenarios. Associated costs of facial paralysis increased with decreasing use of monitoring, that is, higher costs were associated with no provision for monitoring.

Importantly, audiology membership groups support IOM in their various policy documents and scopes of practice. For example, the American Speech Language-Hearing Association recognized audiologists' growing interest and involvement in IOM. In 1991, the Legislative Council approved an official policy document entitled "Neurophysiologic Intraoperative Monitoring." The intent of the document was to provide audiologists with guidelines that considered primarily the quality of care offered to patients for whom IOM was an option (American Speech Language-Hearing Association, 1992). Their scope of practice was revised in 1996 to include IOM while promoting the continued development of professional and technical standards (American Speech Language-Hearing Association, 1996). Similarly, the American Academy of Audiology included in its scope of practice the provision of IOM by audiologists (1996), strengthening that statement several years later (American Academy of Audiology, 2004).

FOOD FOR THOUGHT

1. Please discuss the difference between automated, non-attended intraoperative monitoring versus monitoring provided by a qualified clinician. Please consider the surgeon's point of view, as well as the audiologist's/neurophysiologist's point of view. Who takes responsibility? Who is liable if something goes wrong? Who is responsible for setting up the automated, non-attended device, and does that person have any responsibility?
2. How do you interact with the surgical and anesthesia team members in the operating room; what information do you share with either or both specialties in the course of a surgical procedure? What do you do when you identify a significant change in the neural function of one or more of the structures you are monitoring for a given surgical procedure?
3. Consider the electrically hostile OR environment and discuss ways and means to combat those issues. Is there one solution for all; would the same solution work every time?

DEDICATION TO A FRIEND AND COLLEAGUE

The authors recognize the contributions of the late Dr. Roger Ruth in the development of IOM in the field of audiology and for his contributions to the material in this chapter. He is greatly missed by his family, friends, colleagues, and students.

REFERENCES

American Academy of Audiology. (1996) Audiology: scope of practice. *Audiol Today.* 9, 12–13.

American Academy of Audiology. (2004) Scope of practice. Available online at: http://www.audiology.org/resources/documentlibrary/Pages/ScopeofPractice.aspx. Accessed November, 2013.

American Academy of Otolaryngology-Head and Neck Surgery_ (AAO-HNS). (1998) *Facial Nerve Monitoring. Position Statements.* Alexandria, VA: AAO-HNS. Available online at: http://www.entlink.net/practice/rules/facial_nerve_monitoring.cfm.

American Speech-Language-Hearing Association. (1992) Neurophysiologic intraoperative monitoring [Position Statement]. Available online at: www.asha.org/policy.

American Speech-Language-Hearing Association. (1996) Scope of practice in audiology. *ASHA.* 38 (suppl 16), 12–15.

Badie B, Pyle GM, Nguyen PH, Hadar EJ. (2001) Elevation of internal auditory canal pressure by vestibular schwannomas. *Otol Neurotol.* 22, 696–700.

Banoub M, Tetzlaff JE, Schubert A. (2003) Pharmacologic and physiologic influences affecting sensory evoked potentials. Implications for perioperative monitoring. *Anesthesiology.* 99, 716–737.

Bergmans JA. (1983) Neurophysiological features of experimental and human neuropathies. In: Battistin L, et al., eds. *Clinical and Biological Aspects of Peripheral Nerve Disorders.* New York, NY: Alan R Liss.

Blair E. (1965) A physiologic classification of clinical hypothermia. *Surgery.* 38, 607–618.

Chase SG, Hughes GB, Dudley AW. (1984) Neuropathologic changes following direct current stimulation of the rat sciatic nerve. *Otolaryngol Head Neck Surg.* 92, 615–617.

Edwards BM, Kileny PR. (2000) Intraoperative monitoring of cranial nerves. In: Canalis RF, Lambert PR, eds. *The Ear: Comprehensive Otology.* Philadelphia, PA: Lippincott Williams & Wilkins.

Edwards BM, Kileny PR. (2005) IOM: Indications and techniques for common procedures in otolaryngology-head and neck surgery. *Otolaryngol Clin North Am.* 38, 631–642.

Greenberg JS, Manolidis S, Stewart MG, Kahn JB. (2002) Facial nerve monitoring in chronic ear surgery: US practice patterns. *Otolaryngol Head Neck Surg.* 126, 108–114.

Hirsch A, Anderson H. (1980) Audiologic test results in 96 patients with tumors affecting the eighth nerve. *Acta Otolaryngol (Stockh).* S369, 1–26.

House JW, Brackmann DE. (1985) Facial nerve grading system. *Otolaryngol Head Neck Surg.* 93, 146–147.

Hughes GB, Bottomy MB, Dickins JR, Jackson CG, Sismanis A, Glasscock ME III. (1980) A comparative study of neuropathologic changes following pulsed and direct current stimulation of the mouse sciatic nerve. *Am J Otolaryngol.* 1, 378–384.

Jewett DL, Romano MN, Williston JS (1970) Human auditory evoked potentials: Possible brain stem components detected on the scalp. *Science.* 167, 1517–1518.

Kahn JB, Gliklich RE, Boyev KP, Stewart MG, Metson RB, McKenna MJ. (2001) Validation of a patient-graded instrument for facial paralysis: The facial clinimetric evaluation scale. *Laryngoscope.* 111, 387–398.

Kileny PR, Disher MJ, El-Kashlan HK. (1999) Facial paralysis: Diagnosis and management. *Semin Hear.* 20, 77–91.

Kileny PR, Dobson D, Gelfand ET. (1983) Middle latency auditory evoked responses during open-heart surgery with hypothermia. *Electroencephalogr Clin Neurophysiol.* 55, 268–276.

Kim AH, Edwards BM, Telian SA, Kileny PR, Arts HA. (2006). Transient evoked otoacoustic emissions pattern as a prognostic indicator for hearing preservation in acoustic neuroma surgery. *Otol Neurotol.* 27, 372–379.

Legatt AD. (2002) Mechanisms of intraoperative brainstem auditory evoked potential changes. *J Clin Neurophysiol.* 19, 396–408.

Love JT, Marchbanks JR. (1978) Injury to the facial nerve associated with the use of a disposable nerve stimulator. *Otolaryngology.* 86, 61–64.

Moller AR, Jannetta PJ. (1984) Preservation of facial function during removal of acoustic neuromas: Use of monopolar constant-voltage stimulation and EMG. *J Neurosurg.* 61, 757–760.

National Institutes of Health: Acoustic Neuroma. NIH Consensus Statement. December, 1991. Available online at: http://consensus.nih.gov/1991/1991AcousticNeuroma087html.htm.

Niparko JK, Kileny PR, Kemink JL, Lee HM, Graham MD. (1989) Neurophysiologic intraoperative monitoring: II. Facial nerve function. *Am J Otol.* 10, 55–61.

Nuwer MR. (2002) Regulatory and medical-legal aspects of intraoperative monitoring. *J Clin Neurophysiol.* 19 (5), 387–395.

Prass R, Luders H. (1985) Constant-current versus constant-voltage stimulation. *J Neurosurg.* 62, 622–623.

Prass RL, Luders H. (1986) Acoustic (loudspeaker) facial electromyographic monitoring: Part I. Evoked electromyographic activity during acoustic neuroma resection. *Neurosurgery.* 19, 392–400.

Sala F, Krzan MJ, Deletis V. (2002) Intraoperative neurophysiological monitoring in pediatric neurosurgery: Why, when, how? *Child Nerv Syst.* 18, 264–287.

Schwartz DM, Bloom MJ, Pratt RE, Costello JA. (1988) Anesthetic effects on neuroelectric events. *Semin Hear.* 8, 99–111.

Schwartz DM, Morris MD. (1991) Strategies for optimizing the detection of neuropathology from the auditory brainstem response. In: Jacobson JR, Northern JL, eds. *Diagnostic Audiology.* Austin, TX: Pro-ed.

Sohmer H, Freeman S, Gafni M, Goitein K. (1986) The depression of the auditory nerve brainstem evoked response in hypoxemia: Mechanism and site of effect. *Electroencehpalogr Clin Neurophysiol.* 64, 334–338.

Sohmer H, Gafni M, Chisin R. (1982) Auditory nerve-brainstem potentials in man and cat under hypnoxic and hypercapnic conditions. *Electroencehpalogr Clin Neurophysiol.* 53, 505–512.

Sunderland S. (1945) The arterial relations in the internal auditory meatus. *Brain.* 68, 23–27.

Wilson L, Lin E, Lalwani E. (2003) Cost-effectiveness of intraoperative facial nerve monitoring in middle ear surgery. *Laryngoscope.* 113, 1736–1745.

CHAPTER 17

Middle-Latency Auditory-Evoked Potentials

Anthony T. Cacace and Dennis J. McFarland

INTRODUCTION

Middle-latency auditory-evoked potentials (MLAEPs) occur from approximately 15 to 70 ms and may be of value in estimating auditory thresholds as a function of frequency and in assessing higher-order auditory processes. This chapter focuses on anatomy and generator sites, theoretical and practical considerations including recording parameters and state and subject variables, and other topics relevant to clinical concerns. Some applications, such as those related to sensory gating and depth of anesthesia, have emerged with increased frequency in recent years. Thus, this chapter provides a comprehensive overview of MLAEPs that would be valuable to students and informative to seasoned professionals.

ANATOMIC FRAME OF REFERENCE

Knowledge of thalamic and cortical anatomy is necessary to understand MLAEPs. The auditory thalamus (medial geniculate nucleus, MGN) is an integral component of the subcortical afferent auditory pathways; descending reciprocal pathways from cortex to thalamus and other areas are also noteworthy (Winer and Lee, 2007). Ascending thalamocortical fibers on route to the cerebral cortex course through the sublenticular portion of the internal capsule (Truex and Carpenter, 1964). The ventral division of MGN projects to the core areas of the auditory cortex (AC) located on the superior aspect of the temporal lobe; the dorsal division of MGN projects more diffusely (Rauschecker et al., 1997). The ventral MGN sends direct projections in parallel to primary and rostral areas of the AC, which are highly responsive to puretone stimuli. The dorsal-medial and other aspects of the MGN send direct inputs to caudal-medial aspects of AC. These caudal-medial areas respond preferentially to more complex broadband stimuli. Auditory cortical regions also project back to the medial geniculate regions from which they receive projections (Pandya et al., 1994). Thus, both feedforward and feedback neural projections coexist. These reciprocal relationships allow thalamocortical circuits to be interactive with the environment, thus allowing spectral and temporal transformations of stimulus representations to dynamically modify perception and behavior. In this regard, the classical view of the thalamus as being merely a "passive relay area" requires modification and updating (Winer et al., 2005).

The AC consists of a core area on the superior temporal plane (including Heschl's gyrus) (Figure 17.1A). The core contains three cochleotopically organized fields (Kaas et al., 1999), surrounded by a belt of association areas, which in turn are surrounded by parabelt association areas, extending to the lateral surface of the superior temporal gyrus. The belt also receives afferent input from the core area and dorsal divisions of the medial geniculate complex, with minor projections from ventral and medial geniculate areas. The parabelt has strong connections with the belt area, has minimal connections with the core, and receives thalamic inputs in parallel with belt inputs across its subdivisions. Additionally, the belt and parabelt zones consist of several regions that are distinct in terms of cyto-architecture and connections (Hackett et al., 1999). A block diagram from Kaas et al. (1999) summarizes known neuroanatomical relationships relevant to higher level thalamocortical processing (Figure 17.1B) described above. There is support for the view that these various auditory association areas may be specialized for processing two distinct classes of auditory features (i.e., sound identity information and temporal and spatial information) (Rauschecker et al., 1997) in a manner analogous to what has been described in visual cortical areas (Mishkin et al., 1983). Polysensory regions of the temporal, parietal, and frontal cortices also receive input from AC (Pandya, 1995). Thus, there are both feedforward and feedback connections within and between subregions, including connections with multimodal insula, the superior temporal sulcus, and long association connections with amygdala and prefrontal cortex. Contemporary neuroanatomical staining methods and various imaging-related activation studies suggest that human AC can be subdivided into at least eight different putative regions (Rivier and Clarke, 1997). Human investigations continue to be elaborated on by the increased use of functional neuroimaging methodology (e.g., Saenz and Langer, 2014).

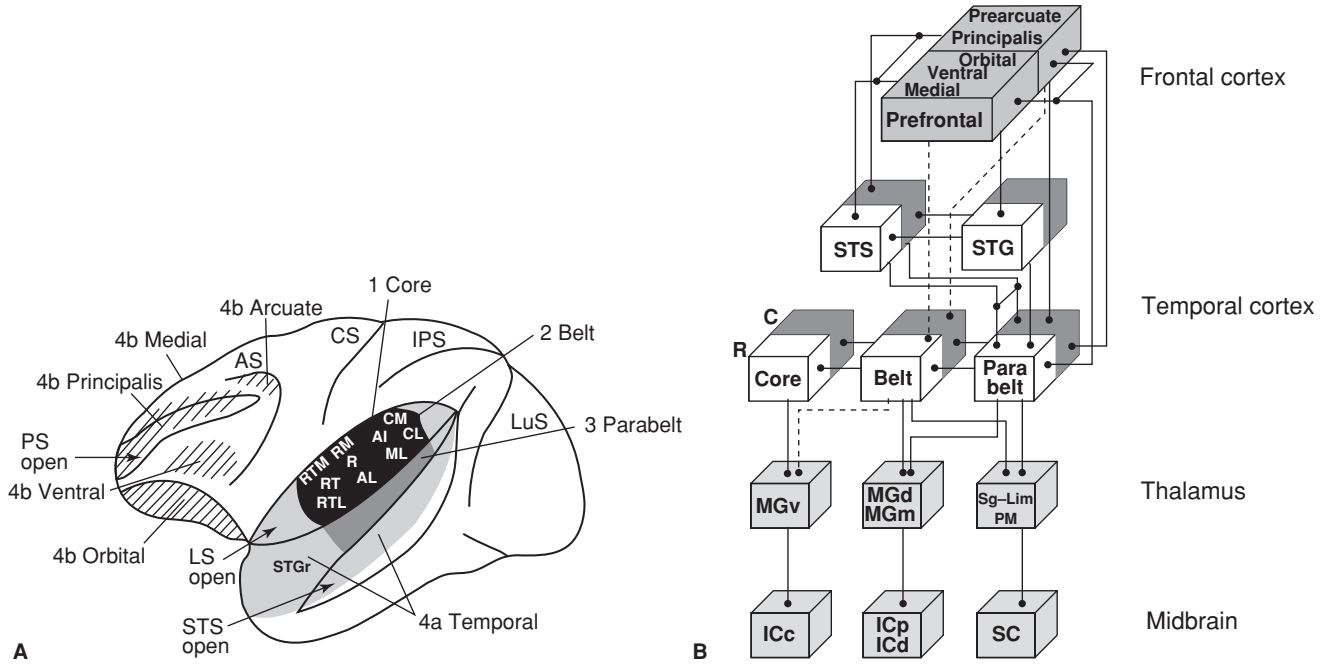


FIGURE 17.1 **A:** Schemata of contemporary auditory cortical neuroanatomy based on levels and regions of processing. In this representation, the lateral sulcus (LS) is opened to show auditory areas of the lower bank and the superior temporal sulcus (STS) has been opened to show the extension of auditory-related cortex in this sulcus. *Level 1* represents the core (*darkest shading*); *level 2* represents the belt (*moderate shading*); *level 3* represents the parabelt (*light shading*); *level 4a* represents the temporal region (*dense hatching*); and *level 4b* represents the frontal region (*sparse hatching*). In this diagram, the following abbreviations are used: AL, anterolateral; AS, arcuate sulcus; CL, caudolateral; CS, central sulcus; IPS, intraparietal sulcus; LuS, lunate sulcus; ML, middle lateral; PS, principle sulcus; RM, rostromedial; RTL, lateral rostrotemporal; RTM, medial rostrotemporal, STGr, rostral superior temporal gyrus. **B:** Block diagram providing details of known connections and levels of processing to the primate auditory cortex. The *solid lines* represent major connections and the *dashed lines* represent minor connections. According to this framework, the main stream of processing involves the central nucleus of the inferior colliculus (ICc), the ventral nucleus of the medial geniculate complex (MGv), and the core areas of the auditory cortex. A parallel stream involves the dorsal (ICd) and pericentral (Icp) divisions of the inferior colliculus, the dorsal (MGd) and medial (MGm) divisions of the medial geniculate complex, and the belt cortex. The superior colliculus (SC) projects to parts of the medial pulvinar (PM), suprageniculate (Sg), and limitans (Lim) nuclei, as a possible third source of input to the parabelt cortex. Additional levels of processing include cortex of the superior temporal gyrus (STG), adjoining belt and parabelt regions, STS, and prefrontal cortex. The preferential connections of rostral (R) and caudal (C) sectors of cortex are indicated. (Both the anatomical representation and block diagram are taken from Kaas JH, Hackett TA, Tramo MJ. (1999) Auditory processing in primate cerebral cortex. *Curr Opin Neurosci.* 9, 164–170, with permission).

GENERAL CONCEPTUAL FRAMEWORK OF AUDITORY-EVOKED POTENTIALS

Electrical activity measured from scalp electrodes in response to sensory stimulation represents the sum (superposition) of the electrical fields projected by all active sites at any given point in time. These include thalamic nuclei, the ascending thalamocortical fibers, primary auditory and auditory association areas, polysensory association areas, and inter- and intracortical fiber tracts. The polarity, spatial distribution, and whether or not electrical potentials generated from brainstem or from within the brain can even be detected by surface electrodes on the scalp depend in large part on the underlying geometry of active cell populations. Idealized and highly schematic examples of the spatial organization of different populations of cells and their resultant electric field distributions are shown in Figure 17.2A to D. The work of Lorente de Nó (1947), updated by Buzsáki et al. (2012), forms the basis for understanding the transformation of electric activity from near-field (intracranial) to far-field potentials detectable at the scalp. Populations of neurons can be regarded as sources of electrical current with a positive charge at one location and negative charge at another (so-called stationary dipoles). The electric field distributions of different populations of neurons

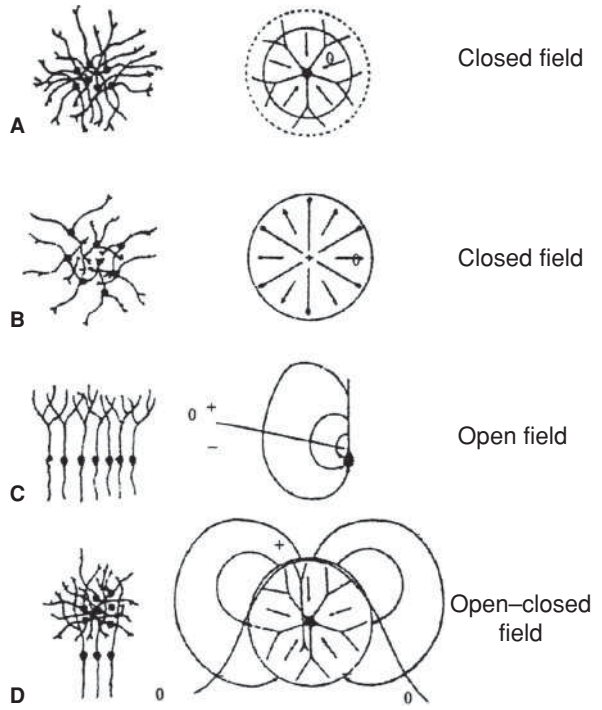


FIGURE 17.2 Examples of closed [**A, B**], open [**C**], and open–closed fields [**D**], for different populations of neurons in the CNS. The *left column* represents different populations of neurons; the *right column* represents activated neuronal populations together with *arrows* representing the lines of current flow at the instant when the impulse has invaded the cell bodies. The *zero* [0] indicates the isopotential line. In [**A**], the schematic depicts the oculomotor nucleus with dendrites oriented radially outward. The isopotential lines are circles with current flowing entirely within the nucleus. This results in a closed field with all points outside the nucleus remaining at zero potential. In [**B**], the schematic depicts the neurons of the superior olive having dendrites oriented radially inward. Here also, the currents result in a closed field. In [**C**], the accessory olive is represented by single neurons and associated long dendritic processes. In this arrangement, the sources and sinks permit the spread of current in the volume of the brain and result in an open-field configuration. In [**D**], two structures mixed together generate an open–closed field. [Adapted from Lopes da Silva F, Van Rotterdam A. [1999] Biophysical aspects of EEG and magnetoencephalogram generation. In: Niedermeyer E, Lopes da Silva F, eds. *Electroencephalography: Basic Principles, Clinical Applications and Related Fields.* 4th ed. Baltimore, MD: Williams & Wilkins; pp 93–109, based on the work of de No [1947], with permission].

have been classified as closed (Figure 17.2A and B), open (Figure 17.2C), and open–closed fields (Figure 17.2D). In an open-field configuration, if dendrites from these populations of cells are all orientated in the same general direction, then in theory, the far-field potentials will be large and easily detectable at the scalp. Indeed, the morphologic polarization of pyramidal cells with long and vertically directed apical dendrites that terminate in the most superficial layers of cortex (gyral crests) can be characterized in this manner. These cell populations, which line the walls of sulci, can generate strong electric and extracranial magnetic fields (Lewine and Orrison, 1999). In neocortical tissue, which is composed of characteristic cell layers, the polarity of the sources and resultant fields are thought to be at a right angle to the plane of the surface and are consistent with this notion of cell distribution (Nunez, 1995). However, in populations of neurons in which the cell bodies are positioned in the center with dendrites projecting in all directions (so-called closed-field or open–closed-field configurations (Figure 17.2A, B, and D), the amplitudes of the resultant far-field electric potentials will be much smaller and may not be detectable at the scalp at all. This observation results from the fact that various components of these electric fields can cancel each other over time. Further complications arise for surface recordings from human AC based on the convoluted nature and complex three-dimensional geometry of the superior temporal plane. Galaburda and Sanides (1980) report that the superior temporal plane is "one of the most highly folded in the human brain" (p 603). Furthermore, individual differences complicate matters further by introducing additional variability between hemispheres and brains.

Given the spatial geometry of active cell populations in both the thalamus and on the superior temporal plane, the resultant electric fields in response to acoustic stimulation should project toward the top of the head and be largest at medial central and/or anterior-central locations. Moreover, these potentials, which are localized at central scalp locations, should be diffuse, since in theory their sources are at a distance. In these circumstances, electric field projections should be broader with greater distance from the generator source. This is in contrast to the projections from auditory association or belt regions on the lateral surface of the temporal lobe, which in theory should produce more focal and lateralized scalp distributions, since the generators are closer to the surface of the scalp. Recent evidence and other supporting data show that there is considerable divergence in the auditory thalamocortical pathway (Winer et al., 2005) and, consequently, results in more complex *radial* and *tangential* cell orientations (Cetas et al., 1999).

Yvert et al. (2005) examined intracranial responses to tone-evoked potentials in subjects being evaluated for epilepsy surgery and found a sequential spread of evoked activity from Heschl's gyrus to surrounding auditory association areas. They modeled the spread of this activity on the scalp and concluded that potentials at the surface represent the activation of multiple sources that are simultaneously active. Furthermore, Boutros et al. (2013) observed activity in the P50 time frame evoked by the first of a pair of tones in temporal, parietal, and cingulate areas. These studies show that there are multiple diverse sources of the P50. Moreover, in a modeling study, Ahlfors et al. (2010) showed that even a small number of simultaneously active dipoles could

produce cancellation of activity at the scalp. Thus, based on empirical evidence and results from modeling experiments, it is evident that multiple sources are simultaneously active in the middle (~15 to 70 ms) and longer (>70 to <256 ms) latency ranges and that there is considerable overlap in both temporal and spatial distributions on the scalp. Thus, adequate spatial sampling with electrodes is necessary.

RECORDING CONSIDERATIONS

The most common type of AEP study is accomplished via surface electrodes. Electrode placement on the surface of the scalp is based on specific conventions, allowing for comparisons between different laboratories and countries (see Jasper, 1958). Depending on the specific context, recordings in the middle-latency range have been derived from single-channel recordings (Thornton et al., 1977), from two channels (Schochat et al., 2010), from linear arrays (Scherg and von Cramon, 1986a), or from more comprehensive matrix designs where data are sampled from a wide range of locations over the entire scalp (Cacace et al., 1990).

A restrictive set of MLAEPs may be recorded from a single channel, with electrodes placed, for example, at the vertex (noninverting electrode), a known reference site (inverting electrode), and ground (common). Evoked potentials recorded from one channel (i.e., two electrodes; active and reference) can be compared to sampling the waveform with only two points in time (Lopes da Silva, 1999). Whereas auditory brainstem responses (ABRs) are far-field electric potentials that reflect broadly distributed responses from fiber tracts and nuclei in peripheral and brainstem auditory pathways (Møller, 1988), MLAEPs originate from sources closer to the surface of the scalp. As a result, MLAEPs show more variation with electrode position and consequently some degree of spatial sampling appears necessary. The electrical potential recorded at any particular electrode, at any point in time, can be regarded as the signal of interest corrupted with unwanted noise. If the noise is random and uncorrelated with the signal, then averaging of multiple samples can improve the signal-to-noise ratio proportional to the square root of "*N*", where *N* is the number of samples in the average (Lopes da Silva, 1999). In theory, by averaging many poststimulus epochs of EEG, the noise component decreases toward zero and the AEP waveform can be extracted from the noise. This approach is the most common analytical strategy for capturing the neural reactivity of "synchronized" EEG to a sensory stimulus (Dawson, 1951). This "additive model" assumes that data contained within individual trials are composed of a linear combination of stimulus (or time-locked activity) plus background noise. However, a fundamental shortcoming of the additive model concerns the way in which nonstimulus locked activity is handled. For example, nonstimulus locked activity is also known as induced, emergent, or "unlocked activity" (see Ba ar and Bullock, 1992; McFarland and Cacace, 2004). The distinction between "stimulus locked" and "unlocked" activity requires consideration because it recognizes that in addition to synchronized activity reflected in the time-domain average, two additional types of signals are embedded within ongoing background EEG. These signals include (1) EEG that is *reactive* to the stimulus but *not* time-locked to the event and (2) unwanted noise. Unlocked activity is largely rhythmic or oscillatory in nature and therefore, it does not have a fixed waveform. In this context, the unlocked component represents those frequency-dependent changes in EEG rhythmicities that are modulated by sensory stimulation. The key point here is that these oscillatory dynamics thought to underlie EEG reactivity *cannot* be captured by typical signal averaging techniques in the time domain and therefore alternative methodologies such as frequency-domain analysis are needed (see Cacace and McFarland, 2006). Accumulating research shows that unlocked oscillatory activity is a rich source of information about brain function which can provide a unique perspective when incorporated within perceptual, cognitive, and motor paradigms (Başar and Bullock, 1992). With more complex tasks used to elicit longer latency AEPs (i.e., oddball or other cognitive-related paradigms), research has shown that unlocked EEG spectral power is associated with attended targets during a frequency discrimination task (McFarland and Cacace, 2004). Precisely how important the spectral dynamics of oscillatory EEG is for MLAEPs is just beginning to be explored; however, recent work has shed light on the fact that assessment of EEG oscillations has implications for P50 in the context of sensory gating endeavors used in psychiatry (Smucny et al., 2013). Additionally, Kruglikov and Schiff (2003) have shown that MLAEPs vary with the phase of the background EEG and suggest that a new conceptualization is required to account for such findings. Phase resetting may also have important implications related to perception, but a more cohesive account of these types of data will be needed for this body of research to advance (Ross et al., 2005).

Ultimately, the more effective the clinician/researcher is in isolating and removing sources of noise contamination during data acquisition, the better the recordings will be. Noise sources which electrodes can pick up by induction include extracranial electrical fields such as the 60-Hz noise from nearby power lines (50 Hz in Europe) and electrical potentials resulting from the movement of the wires connecting the electrodes to the amplifying system. Other intracranial nonauditory noise sources include low-frequency potentials induced by lateral eye movements, higher frequency fields resulting from vertical eye movements (blinks), and broadband signals resulting from electromyographic (EMG) activity. As noted in a previous review on this topic (Cacace and McFarland, 2002), distinguishing true "neurogenic" from "myogenic" activity had complicated the interpretation of MLAEPs for some time. Sources of EMG contamination are provided in Table 17.1. Moreover, if the underlying noise (lateral eye movements, eye blinks, EMG) is in-phase

TABLE 17.1

Time-averaged Electromyographic Potentials from Post-auricular, Temporalis, and Neck Muscles that Can Be Recorded from the Scalp in the Middle-latency Time Frame

Reflex	Description
Post-auricular muscle	Variable from subject to subject and even with subjects. Large negative peak at 11.8 ± 0.8 ms and positive at 16.4 ± 0.7 ms
Temporalis muscle	Very easily recordable from subjects with clenched teeth. Large negative peak at 17.2 ± 1.9 ms and positive peak at 22.8 ± 2.8 ms
Neck muscles	Recordable from the inion. Begins as early as 7.4 ms. Has multiple components: Negative waves at 11.3 + 0.2 and 24.6 ± 1.5 ms and positive waves at 16.8 + 2.4 and 33.8 ± 0.5 ms
Frontalis muscle	Highly variable response. There is usually a distinct positive component at approximately 30 ms

Adapted from Picton TW, Hillyard SA, Krausz HI, Galamabos R. (1974) Human auditory evoked potentials: I. Evaluation of components. *Electroencephalogr Clin Neurophysiol*. 36, 179–190.

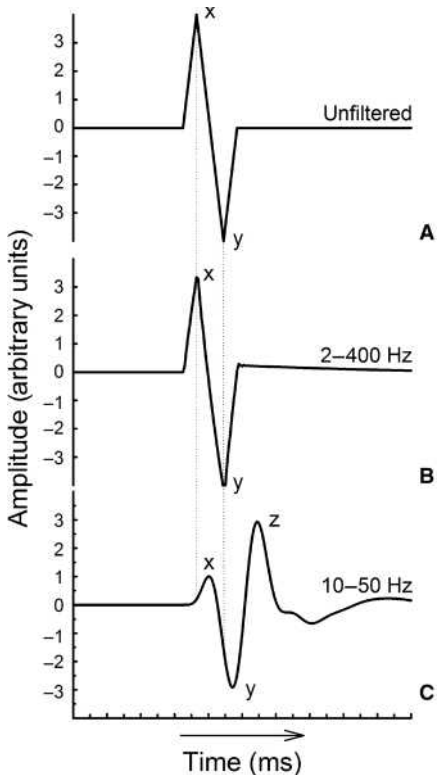


FIGURE 17.3 A simulated waveform is shown under three filtering conditions: (1) Unfiltered, (2) filtered between 2 and 400 Hz, and (3) filtered between 10 and 50 Hz. The first two conditions compare the unfiltered and 2 to 400 Hz filtered condition (**A, B**). They show that the simulated waveform is relatively unchanged (*dropped dashed line*) with respect to latency and amplitude. However, when too narrow a filter setting is used (**C;** 10 to 50 Hz), the waveform is significantly altered; latencies of peak x and y are prolonged, amplitude is clearly diminished, and an additional component (peak z), not seen in the unfiltered or filtered waveform between 2 and 400 Hz (**A, B**) was added. This simulation illustrates that too narrow a filter setting can induce significant artifacts in the recordings including the addition of new peaks that were not part of the original unfiltered AEP.

(i.e., time-locked) with stimulus presentation rate (e.g., 50 to 60 Hz or a harmonic thereof), then the signal averaging strategy discussed above will be unsuccessful. In summary, to improve the quality of the recorded potentials, it is advisable to minimize and/or control exogenous and endogenous artifacts and sources of contamination as best as possible.

Bandwidth is another important consideration in recording MLAEPs. Scherg (1982) has reported that MLAEPs are readily detectable with averaging over a relatively wide bandwidth (1.0 to 5,000 Hz). Campbell and Leandri (1984) have demonstrated that filtering can introduce temporal and amplitude distortions that may not be distinguishable from true potentials. Based on the work of Chang et al. (2012), it can be seen how a narrow recording bandwidth could negatively influence waveform morphology in the middle-latency range; four passbands were compared (0.23 to 75, 10 to 50, 10 to 75, 10 to 200 Hz). To illustrate temporal and amplitude distortions, we present a simulated waveform under three filtering conditions: (1) unfiltered, (2) filtered between 2 and 400 Hz, and (3) filtered between 10 and 50 Hz (Figure 17.3). As can be seen, comparing the unfiltered and 2 to 400 Hz filter (Figure 17.3A and B) shows that the waveform is relatively unchanged. However, when too narrow a filter setting is used (Figure 17.3C; 10 to 50 Hz filter), the waveform is significantly altered; latency of peak x and y is prolonged and amplitude is diminished. Most significant, however, is the observation that an additional component (peak z) was added to the waveform complex. This filter setting was used by Chang et al. (2012) to record the P50 during sensory gating and demonstrates that too narrow of a filter setting can induce significant artifacts in the recordings including the addition of new peaks that were not part of the unfiltered evoked potential. Litvan et al. (2002) used a finite impulse response (FIR) 170th order band-pass filter (25 to 65 Hz) to record MLAEPs in the operating room to identify depth of propofol anesthesia along with a rapid signal averaging method. Limited waveforms are provided and

their technique shows that the amplitudes are significantly blunted. In sum, too broad a filter may retain too much noise in the recording and too narrow a filter can introduce unwanted artifacts that should be avoided. Nevertheless, with available technology, it is possible to design an optimal filter for detecting middle-latency components.

Alternative signal-processing methods, such as correcting for latency jitter (Lopes da Silva, 1999), may also prove useful for improving signal-to-noise ratio. In the audiologic literature, technical considerations related to temporal and amplitude distortions have been discussed (Kraus et al., 1994). The issue of filter bandwidth is also related to the context in which MLAEPs are recorded. For example, it has been suggested that combining ABR and MLAEP recording can enhance threshold estimation for frequencies <1,000 Hz (Scherg, 1982). In this context, the filter's passband needs to be relatively broad to accommodate both ABR and MLAEP spectra. The digital signal-processing strategy of Scherg (1982), including dual artifact rejection algorithms and broad bandwidth (1.0 to 5,000 Hz), appears optimal for these types of recordings. However, the variability of MLAEP detection during different stages of sleep complicates broad bandwidth recordings, particularly when estimating frequency-specific thresholds in infants and young children. In contrast to combining ABR and MLR responses, others have focused on the simultaneous recording of middle and longer latency AEPs (Cacace et al., 1990; Wood and Wolpaw, 1982). In this context, the low-pass cutoff frequency can be more restrictive and the passband of 1.0 to 300 Hz is adequate.

The middle-latency waveform has been characterized by the amplitude and latency of individual components, by means of area under the curve, or by spectral analysis. Various authors describe different numbers of middle-latency potentials. Musiek et al. (1984) identify four positive and three negative waves at the vertex, which they label as Po, Na, Pa, Nb, Pb, Nc, and Pc, similar to the nomenclature used by Thornton et al. (1977). Celesia and Brigell (1999) describe three negative and two positive middle-latency waves, whereas Scherg (1982) identified a single negative and positive peak. Pynchon et al. (1998) describe a procedure involving baseline correction, rectification, and integration of the vertex waveform across an interval determined by the latency of Na and Nb. The rationale for this method is based on the premise that it "is believed to represent the total amount of neural energy contributing to the evoked response" (Pynchon et al., 1998, p 1). As noted earlier, because the potential recorded at the scalp evoked by acoustic stimulation is the superposition of electrical fields projected by all active underlying neural sources, and since sources may cancel, the net projection will vary with the point on the scalp being considered. Thus, it is unlikely that an integration of the scalp waveform at a single point on the scalp represents the total amount of neural energy contributing to the evoked potential.

Reference Electrode

An electrical potential on the scalp, or any other place for that matter, is always recorded between two points. Although some recording montages are referred to as monopolar, they are in fact recordings between two electrodes: An electrode of interest and a shared reference. The potential waveform recorded at any noninverting electrode varies with the reference electrode site used (Gencer et al., 1996). If sources are modeled as dipoles, then a given component will be larger when it is close to the electrode in question and parallel in orientation to a line drawn between the electrode and the reference.

One current view holds that the optimal site for a reference electrode is the placement on the head or body where the potential field is most stable (Wolpaw and Wood, 1982). This consideration ensures that changes in the evoked potential field over time reflect changes in the vicinity of the recording electrode(s) and not a complex combination (sum) of changes at both reference and recording electrode locations. Based on available empirical information, the balanced noncephalic sternovertebral reference of Stephenson and Gibbs (1951) is an optimal choice for use in recording auditory-evoked potentials. Chen et al. (1997) have shown that MLAEPs are larger with a balanced noncephalic reference versus linked earlobes. It is also important to realize that if hemispheric asymmetries exist, as is the case in longer latency AEPs (Cacace et al., 1988; Wolpaw and Penry, 1975), then the balanced noncephalic reference is preferred over other sites.

If clinicians are sampling from many electrode sites on the scalp, then so-called "reference-free derivations" can be used. The common average reference assumes that if one samples the time/voltage waveform at electrode locations all around the skull, the average voltage value of all points would sum to zero. In theory, this approach would provide a reference that would not favor any one electrode over another and presumably would not distort the *true* response. However, this assumption depends on the number of sites sampled. Rarely could one sample all locations around the skull, and as a result, significant biases can occur with this method. The Laplacian method (McFarland et al., 1997) may be applicable to enhance the topographic distinctiveness of the various evoked potential components. This relatively underused but powerful methodology has utility to help delineate evoked potentials in the middle- and long-latency time domains (Hjorth and Rodin, 1988; Law et al., 1993).

Defining AEP Components: Analysis Strategy

Electrode placements for clinical studies need to be based on detailed topographic studies in the time frame of interest. Figure 17.4 provides the waveforms at several recording sites (central, Cz, and temporal, T3 and T4) and detailed topographic maps in the middle-latency range for two individual subjects, which are representative of average data (Cacace et al., 1990).

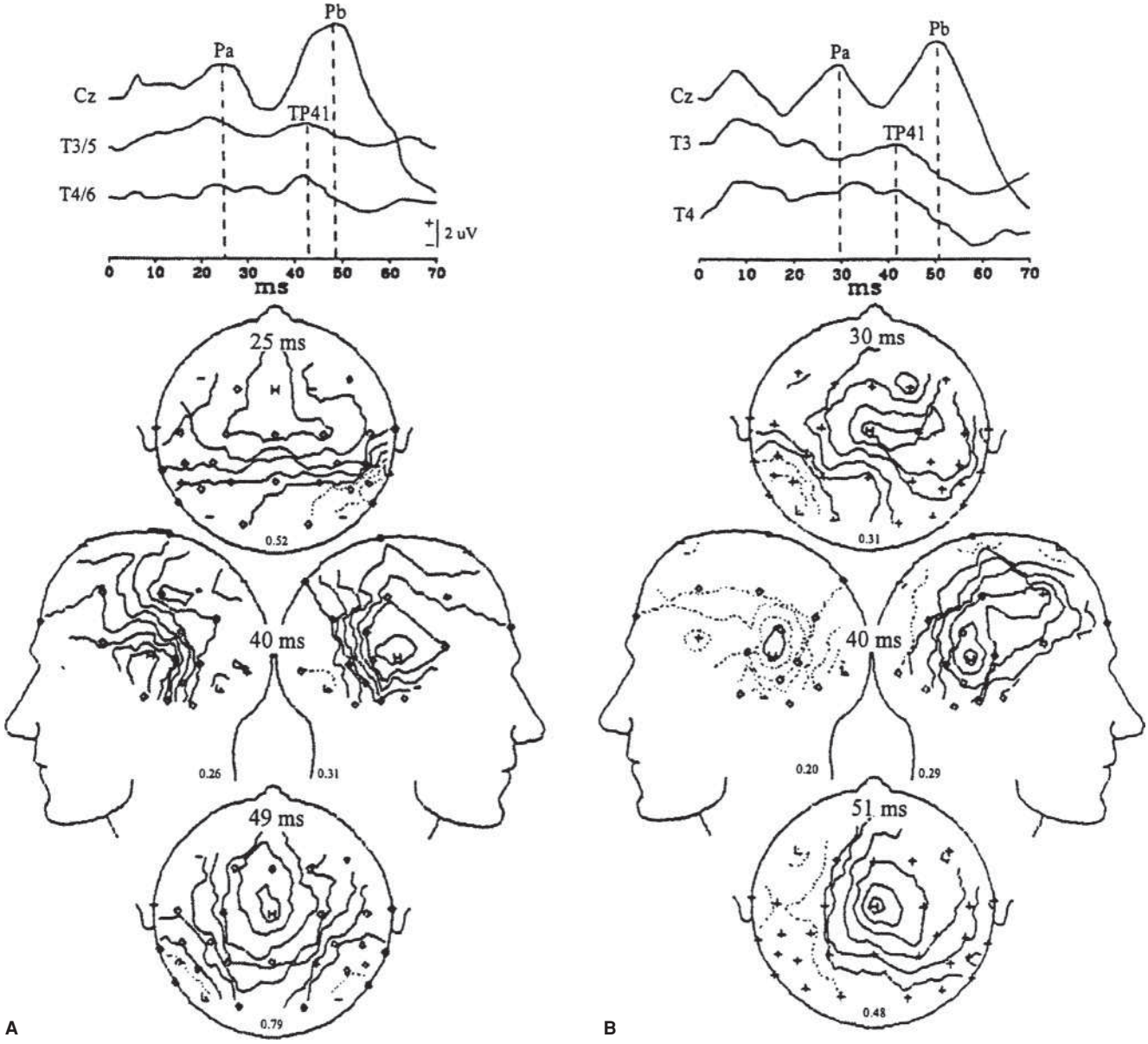


FIGURE 17.4 Middle-latency waveforms and corresponding topographies for two individuals [**A, B**] at central scalp locations [Cz] and over auditory cortex on the lateral surfaces of the temporal lobes [T3/5 or T3 and T4/6 or T4]. Data represented are in response to binaural clicks. Middle-latency components Pa and Pb are seen in the vertex waveforms and TP41 is seen in the temporal waveforms. The central and lateral view topographies are designated at times when components Pa, Pb, and TP41 are maximum. *Solid lines* represent positive voltages, *dashed lines* represent negative voltages; H, high point; L, low point. [Reprinted from Cacace AT, Satya-Murti S, Wolpaw JR. [1990]. Middle latency auditory evoked potentials: vertex and temporal components. *Electroencephalogr Clin Neurophysiol. 77*, 6–18, with permission].

In the middle-latency range (~15 to 70 ms), three components were dominant: Two centered at or near the vertex (Pa, P30; Pb, P50 or P1) and one centered over posterior lateral surface of each temporal areas (TP41). Data provided by Law et al. (1993) show convincingly that the Laplacian derivation, in contrast to the standard potential recordings, enhances the temporal components in the middle- and long-latency domains (Figure 17.5). The spatiotemporal dipole model of Scherg and von Cramon (1986a) also identifies a radial source potential in the middle-latency range that is consistent with TP41. The lateral scalp topography is TP41 is shown convincingly in Figure 17.6. This montage is contrasted with C3 and C4 recording sites used by Schochat et al. (2010), where the rationale for these particular scalp locations is not justified, given available topographic data and known information derived from theoretical models.

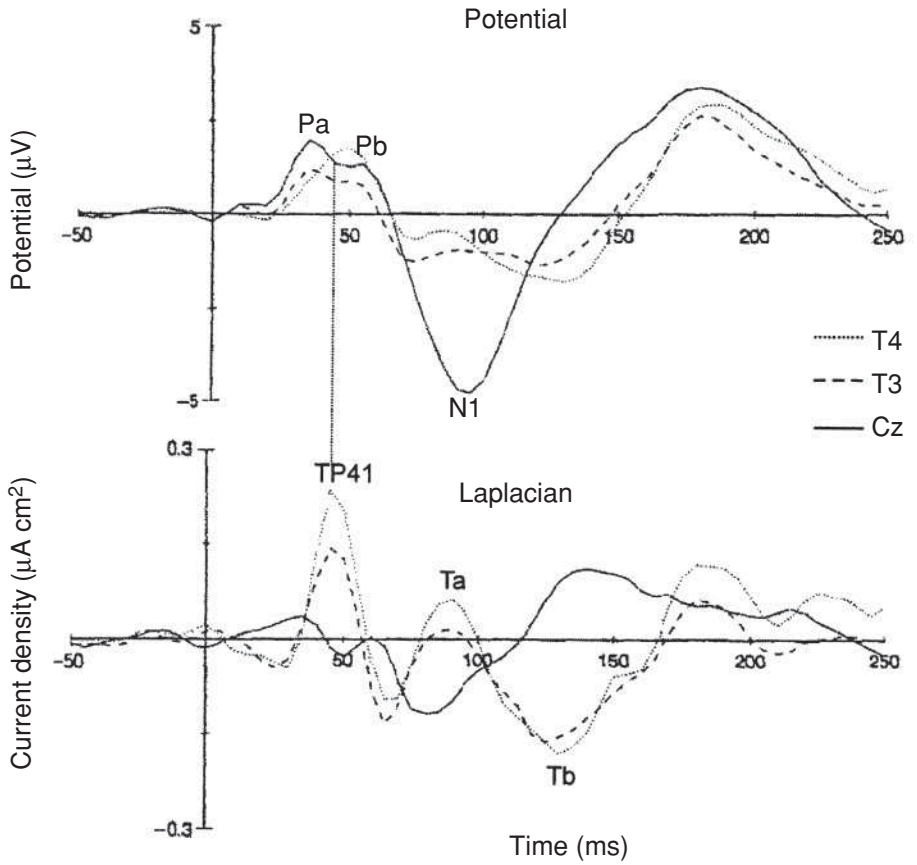


FIGURE 17.5 Middle- and long-latency AEP waveforms are compared in the standard (*top*) potential versus Laplacian derivation (*bottom*), at central (Cz) and right and left temporal lobe sites (T4, T3). Data are in response to mid-frequency tone pips presented at 50-dB sensation level (SL). The electrodes were based on 19 recording sites, referenced to a balanced sternovertebral lead. The top waveform designated as potential shows at Cz well-known middle- and long-latency AEP components (Pa, Pb, N1, P2). The temporal MLAEP component TP41 is not well observed and T-complex components (Ta and Tb) are obscured. However, with the Laplacian derivation (*bottom*), the MLAEP temporal component TP41 and T-complex are clearly enhanced by this transformation. (Adapted from Law SK, Rohrbaugh JW, Adams CM, Eckardt MJ. (1993) Improving spatial and temporal resolution in evoked EEG responses using surface Laplacians. *Electroencephalogr Clin Neurophysiol.* 88, 309–322, with permission).

As noted by Møller (1994), coincidence in latencies does not necessarily constitute proof that near-field potentials are the sources of far-field potentials recorded at the scalp. For example, early cortical potentials in the middle-latency range (approximating 13 ms) are small and can only be recorded from a limited area localized to the posterior aspect of Heschl's gyrus. Subsequent potentials recorded at 30 ms are much larger. Liegeos-Chauvel et al. (1994) note that the response of the planum temporale is more diffuse than that observed in Heschl's gyrus and as such is physically more capable of being propagated to the surface. Hashimoto (1982) used intraventricular electrodes and found that the No–Po–Na complex appeared largest in the vicinity of the inferior colliculus. In addition, it was argued that thalamic contributions to surface recordings are minimal because of the closed-field structure of these cell populations. Thus, the initial response of the primary auditory area is not clearly associated with a reliable scalp potential, a view that has been noted by others (Goff et al., 1977).

Middle-Latency Component Pa: Evidence for Neural Origin

Human pial surface recordings demonstrate a positive peak of Pa latency over temporal and parietal lobes (Chatrian et al., 1960; Lee et al., 1984). Human neuromagnetic recordings show a positive peak at approximately 30 ms (Pelizzone et al., 1987). A positive peak of approximately 30 ms was reported from within the brain (Goff et al., 1977). Furthermore, neuromuscular blockers do not eliminate Pa (Kileny et al., 1983). Multielectrode probe measures at depths above and below Heschl's gyrus failed to show a Pa phase reversal (Goff et al.,

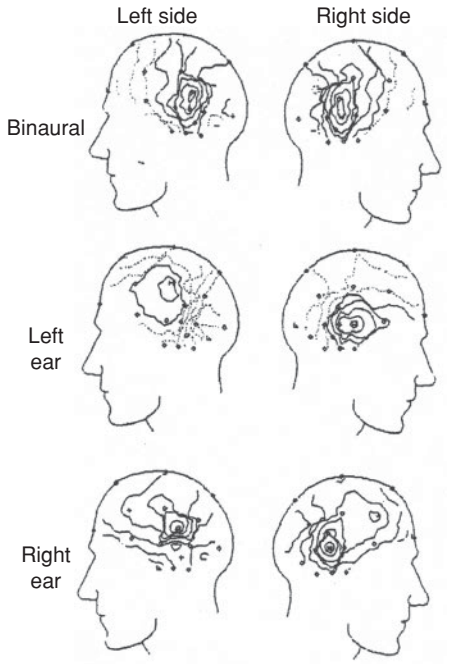


FIGURE 17.6 Middle-latency TP41 is represented by distinct topographic distributions over posterior temporal regions (lateral scalp views; left and right hemispheres) in responses to left, right, and binaural click stimuli. (Reprinted from Cacace AT, Satya-Murti S, Wolpaw JR. (1990). Middle latency auditory evoked potentials: vertex and temporal components. *Electroencephalogr Clin Neurophysiol.* 77, 6–18, with permission).

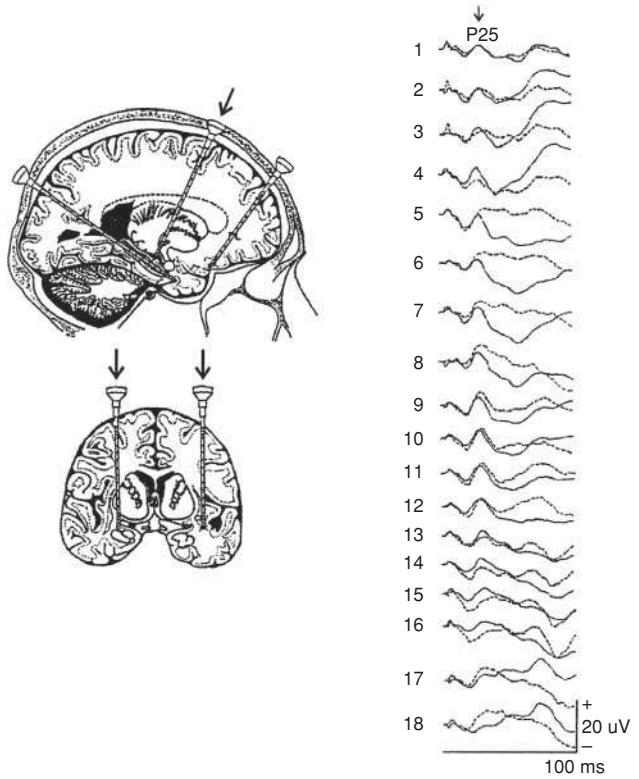


FIGURE 17.7 Middle-latency AEPs recorded from bilateral fronto-temporal probes from within the human brain. *Arrows* indicate the approximate location of the probe. Each probe has 18 contact points (electrodes), with electrode 1 at the most superficial location and electrode 18 at the deepest location. Individual waveforms are grand averages, shown at the right side of the figure. Waveforms represented by *solid lines* are from the right-sided probe; *dashed lines* are from the left-sided probe. Middle-latency component Pa is clearly seen at a latency approximating 25 ms. As shown in the graph (*lower left panel*), Pa amplitude remains relatively constant across recording locations. No phase reversal for Pa is noted at depths above and below Heschl's gyrus. [Adapted from Goff WR, Allison T, Lyons W, Fisher TC, Conte R. (1977) Origins of short latency auditory evoked potentials in man. In: Desmedt JE, ed. *Auditory Evoked Potentials in Man. Psychopharmacology Correlates of Evoked Potentials. Prog Clin Neurophysiol.* Vol 2. Basel: Karger; pp 30–44, with permission].

1977) (Figure 17.7), as would occur if Pa were generated at this site. Neuromagnetic recordings show that the supratemporal AC is active during Pa and a change in waveform morphology occurs in the anterior–posterior plane (Pelizzone, 1987). Pial surface recordings show a similarly oriented change (Lee et al., 1984). However, lesion studies do not provide unambiguous evidence of Pa's origin.

Furthermore, Pa is unaffected by sleep apnea (Mosko et al., 1984) (i.e., ancillary evidence of a noncortical origin). Whereas recent modeling studies suggest Pa is produced by tangentially oriented dipole sources in AC (Scherg and von Cramon, 1986a), data are also consistent with similarly oriented subcortical sources.

Middle-Latency Component Pb: Evidence for a Neural Origin

Recordings from the pial surface in humans demonstrate a positive peak at Pb latency over temporal and parietal lobes (Chatrian et al., 1960). Human neuromagnetic recordings show a positive peak at approximately 50 ms (Pelizzone et al., 1987). A positive peak at approximately 50 ms was reported from within the brain, but less prominent than Pa (Goff et al., 1977). Pb is altered by stage of sleep (Erwin and Buchwald, 1986) and in patients with Alzheimer's disease (AD) (Buchwald et al., 1989).

MLAEP Pb Source

Multielectrode probe measures from within the human brain at depths above and below Heschl's gyrus failed to show a Pb phase reversal (Goff, 1978; Goff et al., 1977). Neuromagnetic recordings show that the supratemporal AC is also active

during Pb and that a change in waveform morphology occurs in the anterior–posterior plane (Pellizone et al., 1987). Reite et al. (1988) suggests that the source is the planum temporale. Goff (1978) reports a positive peak comparable in latency at a depth and location corresponding to the hippocampus and it has been shown that Pb disappears with large lesions to the hippocampus (Woods et al., 1987). Because depth recordings do not show a phase reversal across Heschl's gyrus and since Pb depends on stage of sleep, Pb may not originate in AC but in subcortical structures.

Recent mapping and more invasive studies on the pial surface with grid electrodes in epilepsy patients have been performed based on continuing interest in the area of sensory gating of the P50 paired-stimulus paradigm (Boutros et al., 2013; Korzyukov et al., 2007). Based on this methodology and stimulus paradigm, including source reconstruction and mapping data, the general consensus holds that P50 has prominent generator sites localized in the temporal, parietal, and cingulate regions of the brain. This analysis also suggested that neuronal activity contributing to the amplitude reduction in the P50 time range was localized to the frontal and parietal lobes and the cingulate area.

MLAEP TP41: A New Component

Because early studies have used relatively few electrodes and concentrated on central and not temporal scalp areas, the temporal middle-latency component was not identified. In addition, most studies used ear or mastoid reference electrodes which do not obscure Pa and Pb but markedly obscure and even eliminate TP41. However, it is noteworthy that the appearance of TP41 may require long interstimulus intervals (ISIs) (e.g., 1 second: Knight et al., 1988; 3.3 seconds: Cacace et al., 1990) or pseudo-random ISIs (Scherg and von Cramon, 1986a, 1986b). Furthermore, TP41 may habituate to large numbers of stimuli. Nevertheless, detailed topographic analysis shows that TP41 is highly localized over lateral temporal scalp locations of each hemisphere in response to left, right, and binaural click stimulation.

TP41: Evidence for a Neural Origin

TP41 is undoubtedly neural in origin. In our experience, subjects were relaxed and online monitoring of temporal channels did not show overt EMG activity. The post-auricular muscle reflex is seen more anteriorly with induced muscle tension and this component has a much shorter latency (Picton et al., 1974). A positive peak of comparable latency has been seen in human pial surface recordings over lateral temporal and perisylvian regions (Celesia, 1976). Also, TP41 matches the P39 radial dipole source potentials in the left and right hemispheres of the spatiotemporal dipole model proposed by Scherg and von Cramon (1986a, 1986b).

SUBJECT AND STATE VARIABLES

Age and Gender Effects

McGee and Kraus (1996) have reviewed many relevant issues, which are important toward understanding the complexity of maturational changes in infants and young children that continue through the first decade of life. In adults, Kelly-Ballweber and Dobie (1984) and Woods and Clayworth (1986) found that Pa latency and amplitude increased with age throughout the life span. Gender effects were not found in the Woods and Clayworth (1986) study and Kelly-Ballweber and Dobie (1984) only studied males. In a study limited to females, Chambers and Griffiths (1991) showed that Pa amplitude grows linearly with age, although interpretation can be complicated by changes in hearing sensitivity. In partial contrast, Erwin and Buchwald (1986) found a significant increase in Pa amplitude but not latency with age. Data of Newman and Moushegian (1989) also report larger MLAEP amplitudes in older subjects at higher stimulation rates. Amenedo and Diaz (1998) showed a positive relationship between Na–Pa amplitude and age for individuals between 20 and 86 years.

Pfeifferbaum et al. (1979) reported that Pb (P1 or P50) amplitudes increased with age in women. However, their data are difficult to compare with other studies because they combined Pa and Pb in their data analysis. Chambers (1992) also found larger Pb amplitudes in older individuals (51 to 71 years vs. 20 to 24 years). Using a paired-stimulus paradigm and binaural stimulation, Papanicolaou et al. (1984) showed differential recovery cycle effects on P1–N1 amplitude, characterized by a more rapid recovery in young versus older individuals. Recovery cycle effects for P1 latency, however, were not significant and gender effects were not evaluated. With binaural stimulation, Erwin and Buchwald (1986) found longer Pb latencies in older women. They speculated that menopausal-related hormonal instability might have contributed to this finding. Spink et al. (1979) also failed to find age-related changes in Pb latency, although their sample size was small. Amenedo and Diaz (1998) showed a positive relationship between Nb–Pb amplitude and age for individuals between 20 and 86 years.

Prior use of MLAEPs in certain types of neurologic disease or their early states (minimal cognitive impairment [MCI], AD, Parkinson's disease, Rett syndrome) has been reported but research is limited to a small number of studies with mixed results. Additional details regarding these issues are reviewed in the web version of this chapter.

Extremely large amplitude MLAEP components have been reported in individuals with chronic tinnitus (Gerken et al., 2001), an effect that maybe related to alterations in inhibitory mechanisms (Gerken, 1996). Enhancement of steady-state magnetic fields has also been reported in patients with problem tinnitus (Diesch et al., 2004). In

their study, variations in component amplitude were not observed in the normal-hearing and hearing loss groups. Whereas MLAEP amplitudes reportedly increase with age, very large amplitudes were less common in the elderly group than in the younger group with problem tinnitus. Because increased amplitude MLAEPs have been associated with elderly individuals with MCI, and in individuals with problem tinnitus, larger MLAEPs may not be pathognomonic of any particular dysfunction and may result from different underlying mechanisms.

Handedness

Hood et al. (1990) found that Pb varies with handedness, being 4 ms longer in left-handed adults. Stewart et al. (1993) also found an increase in the latency of the MLAEP components in left-handed individuals, with the greatest effect being on Pb.

State Variables

Whereas MLAEPs are generally unaffected by attention (Picton and Hillyard, 1974), attention-related effects have been shown for amplitude-modulated steady-state responses (Ross et al., 2004). Short latency AEPs (i.e., electrocochleography and ABRs) have been used in the operating room for purposes of monitoring auditory function during otologic or otoneurologic surgery (Møller, 1988). Middle-latency AEPs have also been applied successfully to issues of importance to anesthesiologists or emergency room physicians (i.e., Thornton and Sharpe, 1998), such as monitoring depth of anesthesia (Litvan et al., 2002), in the development of adaptive controllers to deliver anesthesia (Nayak and Roy, 1999), and in the assessment of different hypnotic states in infants and young children (Weber et al., 2004). Interestingly, a device is being marketed to ascertain the degree of consciousness in comatose patients using the MLAEPs, most notably in emergency departments of hospitals (Tsurukiri et al., 2013), but there is little data on this topic. Lastly, knowledge of anesthesia-related issues is important when assessing electrically evoked potentials during cochlear-implant surgery. Kileny et al. (1983) showed that MLAEPs are not affected by nitrous oxide and narcotic analgesics.

MLAEPS AND THE SPEECH-EVOKED ABR: A CHALLENGING TOPIC WITH AN UNCERTAIN INTERPRETATION

First described by Kraus and colleagues (Song et al., 2006) and recently reviewed by Banai and Kraus (2009), the speech-evoked ABR (sABR) is pertinent to MLAEPs because this response is measured over a time interval which captures middle-latency responses (i.e., 70 to 80 ms). It is typically recorded in response to a repetitive synthesized speech stimulus (typically /da/; ~40 ms in duration) that reflects the onset, offset, and periodicity of the stimulus. Some studies have recorded over a longer time frame (−40 to 190 ms) (Anderson et al., 2012). Nevertheless, the presumed transient component elicited by the stimulus includes ABR Waves I, III, and V (with the most prominent Wave V positive peak and negative trough A) and is thought to represent speech encoding in the brainstem. The so-called "sustained" portion of the response, which follows the ABR, is much less clear and somewhat ambiguous in terms of where and how the speech signal is being processed. In some reports where repetitive waves are observed, investigators invoke the so-called "frequency-following response" (FFR) (Galbraith and Arroyo, 1993). However, Kraus and colleagues never provide a clear explanation why conventional MLAEP components (Pa and Pb) are not observed in this time frame. Furthermore, it is also important to understand whether the response obtained from the speech token is reliable (Hornickel et al., 2011; Song et al., 2011). There is reason to believe that it is not; it is also reasonable to suggest that statistics used to evaluate reliability were not appropriate (see McFarland and Cacace, 2011, 2012). Thus, a wide range of metrics has been proposed (Song et al., 2011) and it is unclear which of these components produces consistent results in a clinical setting.

Perhaps most important is a consideration of the evidence for the proposed brainstem origin of these potentials (Chandrasekaran and Kraus, 2010). It is well known that early components of the auditory-evoked potential can be associated with brainstem sources (Møller and Jannetta, 1983). However, potentials recorded at these later intervals could have contributions from sources located more rostrally. This issue is glossed over in discussions of the significance of these potentials, but as illustrated by our earlier discussion of conventional middle-latency components, source identification can be a complex issue. Song et al. (2008) invoke the "corticofugal system" as influencing the sABR. However, they only provide speculative scenarios and no direct evidence to support their position.

STIMULUS CONSIDERATIONS IN MIDDLE-LATENCY RESPONSES

Middle-latency responses can be affected by changes in various stimulus parameters, including frequency, level, duration, rise/fall time, monaural versus binaural presentation, spectral complexity, and recency (e.g., McPherson and Starr, 1993; Thornton et al., 1977). For example, increases in stimulus rise time have the general effect of significantly increasing latency and decreasing peak amplitudes of Na, Pa, and P1 or Pb (Kodera et al., 1979). Vivion et al. (1980) similarly found that the latencies of five middle-latency components (Pa, Nb, Pb, Nc, Pc) increased and their amplitudes

decreased as rise time increased from 3 to 10 ms, or as the equivalent duration increased from 10 to 30 ms. Thornton et al. (1977) performed parametric studies on MLAEPs in normal-hearing adults using linearly gated tone bursts varying in frequency (250, 1,000, and 4,000 Hz) and stimulus level (no stimulus, 10 to 80 dB HL, in 10-dB steps) and evaluated amplitude and latency components Na, Pa, Nb, and Nc. An inverse relationship was found between latency and frequency but latency was only slightly affected by changes in stimulus level. Input/output functions varied with frequency and depended on the specific MLAEP studied. Audiogram reconstruction is an ambitious goal using evoked potentials, because the degree of frequency specificity is influenced by temporal constraints. Therefore, the type of windowing function used (Harris, 1978) is thought to be important, because it influences how energy is distributed in the frequency domain. When stimuli are presented in isolation, one accepts the same limitations inherent in the behavioral puretone audiogram; depending on stimulus level, and based on basilar membrane traveling-wave characteristics, low-frequency stimuli have poorer frequency selectivity and poorer synchrony. Therefore, one is less confident in the precision of frequency-dependent thresholds, particularly if hearing loss exists. Alternatively, stimuli can be presented in the presence of various types of masking noise, with the intent of restricting spread of excitation in the cochlea and improving the place specificity of the response. The first option is relatively straightforward and simple to apply with current computer technology. The second option is much more complex and more difficult to implement, particularly in the clinical setting, and in some instances, results can have more complicated rather than simpler interpretations (Margolis et al., 1981).

Several recent investigations have addressed these issues and their findings can help clinicians to select the appropriate stimuli for AEP studies. In normal-hearing subjects, the degree of frequency and place specificity was studied by comparing brainstem and MLAEPs in response to short-duration, linear rise/fall, and Blackman-windowed 500- and 3,000-Hz stimuli presented at a moderate level, ~52 to 53 dB nHL (Oates and Stapells, 1997a, 1997b). In these investigations, stimuli with linear rise/fall times were constructed based on a 2–1–2 design (two cycles of rise time, two cycles of fall time, and one cycle of plateau). The Blackman-windowed stimuli were five cycles in total duration, with no plateau (50% rise/50% fall times). Stimuli were either presented alone or in the presence of high-pass noise at various high-pass cutoff frequencies (Oates and Stapells, 1997a). Subtracting the response obtained at one high-pass masker cutoff frequency from the response obtained at a higher frequency high-pass cutoff, Oates and Stapells (1997b) evaluated the place specificity of these derived responses. The results from both studies showed that at moderate input levels, few or no differences exist in the place specificity under these various stimulus conditions and that either linear- or Blackman-windowed stimuli presented in isolation were appropriate.

We also emphasize that the above-mentioned findings must be tempered in patients with steeply sloping high-frequency hearing loss in whom higher stimulus levels are needed to elicit a response. Here, side-lobe energy contamination to lower frequencies becomes more of an issue and the windowing function takes on greater importance, particularly if masking is not used. Consequently, the result of such side-band energy contamination would be to underestimate the magnitude of high-frequency hearing loss. However, in the case of low-frequency hearing loss, spread of excitation to higher stimulus frequencies, as stimulus level is increased, is potentially a much greater concern. In this instance, stimulus shaping alone may be insufficient to guarantee frequency and place specificity. Based on thresholds derived from psychoacoustic studies, special masking procedures in conjunction with click or tone-burst stimuli may be needed to ensure that more accurate thresholds are ascertained (Halpin et al., 1994; Turner et al., 1983).

Erwin and Buchwald (1986) studied recovery cycle effects for middle-latency components Pa and Pb (P1), in part to assess if these components may arise from different generator systems. They showed that increasing stimulus presentation rate (0.5, 1, 5, 8, and 10/second) differentially affected peak-to-peak amplitudes of Pb, but not Pa. They suggest that this evidence supports the existence of separate neural generators for Pa and Pb. Using a noncephalic reference, Nelson et al. (1997) reported a rate effect for Pb, showing that this component is largest at slower rates and for lower frequencies (500 vs. 4,000 Hz). Using MEG, Onitsuka et al. (2003) also found a rate effect for P50m but not for P30m. These findings are in partial contrast to those of McFarland et al. (1975), in which rates as high as 8/second had little effect on the middle-latency waveform or its identifiability.

Although mismatch responses are usually associated with later potentials, MLAEPs have been found to be enhanced to the oddball in a mismatch paradigm (Boutros et al., 1995). Althen et al. (2013) provide evidence that acoustic regularities are encoded at different levels of the auditory system as MLAEPs are sensitive to simpler stimulus features as compared to later potentials which respond to a wider range of mismatch features. Their results suggest that auditory change detection involves a distributed system that is not confined to later time periods.

There has been considerable recent interest in stimulus-evoked and induced oscillatory activity, particularly in the gamma (40-Hz) range (Tallon-Baudry and Bertrand, 1999). This response may be related to the 40-Hz steady-state potential identified by Galambos et al. (1981), as discussed below. Evoked oscillatory activity is phase-locked to the stimulus and can be seen in the averaged waveforms. Induced activity is not phase-locked and requires spectral analysis or some related technique. There has been

speculation that gamma rhythms are neural correlates of various perceptual processes (e.g., Tallon-Baudry and Bertrand, 1999).

CLINICAL USE OF MIDDLE-LATENCY POTENTIALS

Threshold Estimation

Effects of Hearing Loss on MLAEPs

Initially, it was felt that MLAEPs could serve as a means of threshold estimation in the lower frequency range for audiometric purposes. This view came about because MLAEPs are less dependent on temporal synchrony than ABRs and because frequency-specific ABRs are less reliable below 1,000 Hz than above this frequency (Gorga et al., 1988). Clearly, this type of application is important in the assessment of hearing sensitivity in infants and young children. Xu et al. (1995) used the cross-correlation function of two MLAEP waveforms to ensure response identification and found good agreement between evoked potential and behavioral thresholds in individuals with moderate hearing loss. Hausler et al. (1991) also reported good agreement between MLAEPs and behavioral responses in infants and developmentally delayed children. However, others have expressed caution in estimating low-frequency (500 Hz) thresholds with MLAEPs, particularly in children (Barajas et al., 1988a, 1988b).

Galambos et al. (1981) described a steady-state auditory potential elicited by a continuous 40-Hz stream of acoustic stimuli. Initial studies in normal-hearing and hearing-impaired adults showed promise at threshold estimation in both the low- and high-frequency range with this technique (Dauman et al., 1984; Galambos et al., 1981; Stapells et al., 1988). Presently, there is a resurgence of human steady-state responses, and this area is reviewed by Dimitrijevic and Cone (Chapter 15 of this book). However, interest in steady-state or transient MLAEPs waned when they were found to be absent in infants and were affected by sleep, sedation, and anesthesia (Kraus et al., 1989; Plourde and Picton, 1990; Small and Stapells, 2004; Stapells et al., 1988).

Because MLAEPs are less dependent on neural synchrony than ABRs, suggested applications include threshold assessment in the low-frequency range (<1,000 Hz), particularly in those instances where neurologic damage may affect neural synchrony, making threshold detection with ABRs difficult or impossible (McGee and Kraus, 1996). However, those clinical instances in which use of AEPs are most important, such as early identification of peripheral hearing loss in infants and young children, are also those settings in which MLAEP variability is highest. As noted above, sleep and anesthesia affect MLAEPs and as a result, they may not be useful in patients who cannot or will not cooperate with the examiner. Based on a series of experiments (Oates and Stapells, 1997a, 1997b), the frequency specificity of MLAEPs and ABRs at low (500 Hz) and high frequencies (2,000 Hz) were found to be relatively similar. Therefore, because of the general robustness of the ABR response and its independence from state variables, the need to use MLAEPs for low-frequency threshold detection on a routine basis, at least at 500 Hz, is questionable.

Site-of-Lesion Testing

An analysis of the effects of cerebral lesions on MLAEPs can provide information about underlying neural generators. In addition, correlation of evoked potential results with perceptual disturbances provides a means of establishing the validity of various components as indices of central auditory processing. Woods et al. (1984) examined a case of bitemporal lesions associated with cortical deafness, in which they observed a positive wave at 57 ms and a negative wave at 98 ms with normal topographies, latencies, and amplitudes. They suggested that one possible explanation of these results is that middle-latency vertex potentials are produced by polysensory cortex in the vicinity of auditory areas. In a subsequent study, Knight et al. (1988) reported reduced amplitudes of temporal and vertex middle-latency peaks in a group of patients with lesions of the superior temporal gyrus. In contrast, lesions of the inferior parietal lobe produced minimal effects. They concluded that the superior temporal gyrus played a critical role in the generation of these potentials. Scherg and von Cramon (1986b, 1990) suggest that there are three types of AEP alterations resulting from unilateral lesions to central auditory pathways: (1) An "acoustic radiation" type, with unilateral reduction in middle-latency dipole source potentials and preserved tangential and radial dipole source potentials; (2) a "primary AC" type with unilateral reduction of both middle- and long-latency radial and tangential dipole source potentials; and (3) an "auditory association" type, showing normal MLAEPs with a localized reduction of long-latency radial N150 and preserved tangential long-latency dipole source potentials.

As we noted above, the issue of number of electrodes (i.e., spatial sampling) becomes crucial in attempting to determine the effects of CNS lesions on these potentials. Initial studies have limited electrode placements to central scalp locations, and therefore are limited in scope. Many studies evaluating the effect of brain lesions in the middle-latency range have not even placed recording electrodes over temporal lobe sites or have used reference electrode sites that make such obtained data uninterpretable. Parving et al. (1980) recorded MLAEPs in a patient with auditory agnosia and found a normal Pa response. Although they conclude that Pa was neurogenic in origin (an important issue at that time), they also argued that MLAEPs cannot be regarded as being generated exclusively, if at all, in primary AC. Özdamar et al. (1982) report a case of cortical deafness

with preserved BAEPs but absent middle-latency peak Pa. Based on CT studies, Özdamar and colleagues concluded that the absence of the MLAEP component Pa was due to hematomas and infarcts of the left and right temporal lobes. However, this interpretation has been challenged, suggesting that secondary changes because of retrograde degeneration of subcortical structures were not ruled out as a cause for this abnormality. In 19 patients with temporal lobe lesions studied by Kraus et al. (1982), Na–Pa was reduced over the involved hemisphere. According to Kraus and colleagues, normal intersubject variability of conventional amplitude measures and occasional myogenic contamination set limits in establishing reliable criteria that could be applied clinically for the diagnosis of patients with temporal lobe lesions. In two patients with central deafness resulting from bilateral localized vascular lesions at the level of the putamen, Tanaka et al. (1991) reported auditory thresholds in the moderately severe-to-profound range, intact ABRs (Waves I to VI), and complete absence of middle-latency component Pa bilaterally. Interesting, the long-latency AEP (LLAEP) (N1P2 complex) was preserved in both cases. In a review of other cases with bitemporal lesions of the AC, which did not produce cortical deafness, Woods et al. (1984) were unable to support the view that the primary generator sources of MLAEPs and LLAEPs reside exclusively in AC. They suggest that abnormalities found in MLAEPs are associated with subcortical lesions or cortical lesions extensive enough to denervate thalamic projection nuclei. Woods et al. (1987) reported a case of cortical deafness in an 82-year-old woman, resulting from successive strokes of the right and left temporal lobes secondary to bilateral occlusion of the posterior temporal branch of the middle cerebral arteries. MLAEPs and LLAEPs (P1, N1, P2) were preserved, despite the fact that puretone behavioral thresholds were in the moderate-to-profound range and no auditory discriminations could be made. According to the authors, this case is an example of dissociated perception from MLAEPs and LLAEPs. Based on a three- or four-electrode array in the coronal plane (Cz, C6, C5, T3, T4), Kileny et al. (1987) showed that Pa was reduced over the involved hemisphere, but remained intact over the contralateral hemisphere, in individuals with unilateral lesions of the temporal lobe. In this study, the ABRs, particularly Wave V latency, were normal regardless of the site of lesion. No mention was made of whether TP41 was recorded. Comparing conventional MLAEPs with those obtained using maximum-length-sequence technique in controls and individuals with CNS lesions, no advantage was observed using the more sophisticated technique (Musiek and Lee, 1997). In a study that used both magnetic and electric AEPs, Leinonen and Joutsiniemi (1989) examined four patients with temporal lobe infarcts and recorded AEPs in the 40- to 200-ms range. Responses were abnormal in all four patients, and missing in two. In one individual, responses were of abnormally high amplitude, and in another, parts of the response sequence were missing. Electric AEPs were in accordance with the magnetic field measures, although magnetic recordings are *insensitive* in evaluating radially oriented dipoles (Lewine and Orrison, 1999), and therefore cannot delineate the TP41 or later T-complex waveforms. In 12 patients with intractable seizures, Jacobson et al. (1990) found that Pa was unaffected by anterior temporal lobectomy, whereas Na latency and Na/Pa amplitude showed significant increases after surgery. They suggest that changes in Na and Na/Pa amplitude reflect a loss of the modulating influence of the cortex on the subcortical generators of Na. In 24 patients with cortical lesions affecting primarily the temporal lobes (i.e., by CT documentation), Na and Pa obtained over vertex were normal, whereas MLAEPs over the coronal plane showed Pa amplitude to be attenuated or absent over the damaged temporal lobe, relative to the vertex or intact hemisphere (Shehata-Dieler et al., 1991). Again, no mention was made of abnormalities to the temporal component, TP41. Vizioli et al. (1993) suggested that Na and Pa have different generator sites, based on findings from three individuals with brain tumors. Toyoda et al. (1998) suggest that large vessel vascular disease, including those with Moyamoya-like vasculopathy, significantly affects auditory-evoked MEG fields and dipoles in the middle-latency range. Toyoda and colleagues also correlated MEG abnormalities with changes in blood flow using positron emission tomography (PET) and showed that reduced perfusion in areas encompassing both the auditory radiations and AC correlated with the deficits in auditory-evoked magnetic fields. Setzen et al. (1999) found absent MLAEPs and LLAEPs but normal transient-evoked and distortion product otoacoustic emissions and ABRs in a child with Moyamoya disease with central deafness. In this case, digital subtraction angiography showed that the middle cerebral artery was absent on the right side and almost completely occluded on the left side, except for a prominent angular/parietal branch. However, the lesions in this case were more diffuse, including subcortical and cortical ischemic damage and focal lesions in frontal, parietal, and temporal lobes. The absence and occlusion of the vessels noted above is significant, because they supply blood to the lateral two-thirds of each hemisphere (motor, tactile and auditory areas) as well as to adjacent subcortical sites (Taveras, 1996). Additionally, unilateral lesions of the superior temporal gyrus abolish TP41 over the lesioned hemisphere. No change in either latency or amplitude of TP41 is observed with inferior parietal lobe lesions (Knight et al., 1988).

Cochlear Implants

The use of MLAEPs has been suggested for the evaluation of cochlear-implant candidates (Kileny and Kemink, 1987). These authors have generally found that MLAEPs evoked by electrical pulses are similar to those evoked by acoustic stimulation. In adults, electrically evoked MLAEP

thresholds correlated positively with acoustic thresholds obtained using the implanted device. Furthermore, in postlingually deafened adults, MLAEP variations in amplitude and latency were related to specific speech-perception abilities. In contrast to electrically evoked ABRs, studies using MLAEPs have additional advantages: They are not influenced by electrically induced stimulus artifacts, longer stimulus pulse trains can be used, which results in lower levels of stimulation, and a greater proportion of the auditory pathway is activated, which may correlate better with outcome. When P1 (P50 or Pb) is used as a marker of auditory system maturation, its latency becomes adult-like by 15 years of age (Eggermont et al., 1997). The P1 peak latency was found to mature at the same rate in normal-hearing and implanted children, and it was found that the time to maturity in implanted subjects is delayed by an amount approximately equal to the duration of deafness. Kelly et al. (2005) studied MLAEPs and LLAEPs in experienced adult cochlear-implant users and compared them to speech-perception measures. Electrode sites were limited to central scalp locations. For middle-latency component Pa, similar latencies and amplitudes were found in normal-hearing and cochlear-implant groups. The authors noted considerable intersubject variability and found no relation between MLAEPs and CI performance for sentences and word lists. Gordon et al. (2005) evaluated the electrically evoked MLAEPs in children receiving cochlear implants. Except for testing that was performed at the time of surgery, recordings were made when individuals were awake using a standard central recording location and recording (epoch 80 ms). Middle-latency AEP detection increased dramatically over a 5-year period (from poor detection at the time of surgery in the operating room to near 100% detection after 5 years of use). These results suggest that electrically evoked MLAEPs are not useful clinically for predicting optimum stimulation levels or assessing CI function at early stages of device use. Whereas MLAEPs were not found to be particularly useful during early stages of CI usage, they may be a valuable tool to track activity in thalamocortical pathways in CI use over time.

SENSORY GATING AND THE DUAL-STIMULUS PARADIGM USED IN PSYCHIATRIC RESEARCH

Other applications of MLAEPs related to sensory processing have been in the area of psychiatric research concerned specifically with the neurobiology and theoretical underpinnings of schizophrenia, focusing specifically on P50 or Pb. Available data suggest that when comparing the responses of normal healthy subjects to paired identical acoustic stimuli (with an ISI approximating 500 ms), the second stimulus in the pair usually elicits a much smaller P50 amplitude (Dolu et al., 2001). The decrement in response has been interpreted to mean that when a continuous stream of incoming (repetitive) auditory information is presented, this stream is gated or screened, preventing an overload of higher-order stages of auditory information processing. It has been hypothesized that individuals with schizophrenia have a disturbance in this gating phenomenon that results in the "inability" to filter out extraneous noise stimuli from meaningful sensory inputs (Boutros and Belger, 1999; Freedman et al., 1987). Individuals with schizophrenia do not show the normal attenuation of the P50 response to the second stimulus in a pair of acoustic stimuli. This has been interpreted as an inability of the schizophrenic brain to inhibit or gate its response to specific stimuli. In fact, the P50 response to the second stimulus is often larger than normal and this has been interpreted as a deficit in P50-response suppression. As a potential marker for sensory gating, a considerable literature now exists linking a disturbance in this phenomenon to brain neurobiochemical abnormalities and other developmental and genetic influences (Adler et al., 1998; Freedman et al., 1995; Light, 1999). Impaired suppression of P50 to the second stimulus in a pair has also been found in individuals with post-traumatic stress disorder (Karl et al., 2006), antisocial personality disorder (Lijffijt et al., 2009a), bipolar disorder (Lijffijt et al., 2009b), post-traumatic stress disorder with a history of torture (Gjini et al., 2013), psychosis proneness and cocaine dependence (Gooding et al., 2013); the use of P50 as an outcome measure for drug treatment in schizophrenia has also been explored (Oranje and Glenthøj, 2013). Other areas which fall under the heading of learning disabilities have shown mixed results; abnormal MLAEPs have been reported in selected groups (Arehole et al., 1995) and where others have failed to find abnormalities in children with a wide range of cognitive, neurologic, speech, and language disorders (Kraus et al., 1985; Mason and Mellor, 1984).

The novel application of correlating P50 latency with the diffusion tensor imaging (DTI)[1] metric fractional anisotropy (FA) was explored in a neurodevelopmental study comparing groups of typically developing normal children with children along the autistic spectrum (Roberts et al., 2013). FA was measured using a region-of-interest analysis that was localized to the thalamocortical radiations; P50 was measured using

[1]Diffusion tensor imaging measures the displacement of water molecules (diffusion) within white matter tracts, providing information on the microstructure of cerebral white matter of the brain, thus serving as a biomarker of tissue integrity. For each voxel, DTI estimates diffusion in three orthogonal axes (eigenvectors) of an ellipsoid, defining the principal (major), intermediate, and minor axes. The most commonly used metric to quantify the relationship between eigenvalues is FA, a normalized scalar that represents the fraction of the diffusion tensor which is anisotropic. The FA metric ranges between 0 and 1, where 0 represents perfectly "isotropic" diffusion, such as is found in the cerebrospinal fluid where diffusion is equivalent in all directions, and where 1 is the extrema for "anisotropic" diffusion, indicating maximum difference between directional components, such as is found in coherent white matter tracts which consist of long tubes.

magnetoencephalography. The authors found that FA values increased with age in the typically developing group but not in the autistic spectrum group. In typically developing children, P50 latency decreased as FA increased with age. In the autistic spectrum group, no such relationship was observed. These correlations underscore the importance of white-matter development in auditory thalamocortical electrophysiological measures in normal development whereas the lack of association between P50 and FA observed in the autistic spectrum group implies an "uncoupling" between structure and function in the thalamocortical radiations. Clearly, the introduction and utilization of clinically based electrophysiology and magnetic resonance imaging should result in further advancements in this area as more researchers apply these power methods in future investigations.

Are MLAEPs Modality Specific?

It has been suggested that MLAEPs may be valuable indices of central auditory processing disorders (e.g., Musiek and Baran, 1987; Pasman et al., 1997). As noted in our discussion of the literature, several authors have suggested that polysensory areas contribute to the generation of AEPs. Of primary concern here is the extent to which alterations in auditory-evoked potentials are influenced by pathology in polysensory or supramodal brain areas. This topic has been the concern of investigations using other AEP components, such as the mismatch negativity. For example, Alho et al. (1994) report that lesions to the frontal cortex can impair the generation of this response.

CONCLUSIONS

Based on this overview, it is evident that MLAEPs have been applied to virtually all areas of auditory research and one cannot escape the conclusion that these potentials seem to have more theoretical than practical value; however, as we have shown, there are areas where this may change, such as in the P50 gating literature and in the context of MRI and electrophysiological correlations. As an audiometric tool for threshold determination in infants, MLAEPs have not been particularly reliable. Nevertheless, although routine clinical applications are limited, sophisticated research studies continue to add to the literature on this topic (Rupp et al., 2004) which is encouraging for future developments.

FOOD FOR THOUGHT

1. How can one determine whether or not an evoked potential component is modality specific?
2. Would it be a good idea to use an adaptive filter that adaptively varies the bandpass in order to enhance EEG signals?
3. Why are the well known MLAEP components (e.g., Pa and Pb) not observed in speech-evoked ABRs given the typical time epoch being recorded?

REFERENCES

Adler LE, Olincy A, Waldo M, Harris JG, Griffith J, Stevens K, et al. (1998) Schizophrenia, sensory gating, and nicotinic receptors. *Schizophr Bull.* 24, 189–202.

Ahlfors SP, Han J, Lin F-H, Witzel T, Belliveau JW, Hamalainen MS, et al. (2010) Cancellation of EEG and MEG signals generated by extended and distributed sources. *Hum Brain Mapp.* 31, 140–149.

Alho K, Woods DL, Algaza A, Knight RT, Näätänan R. (1994) Lesions of frontal cortex diminish the auditory mismatch negativity. *Electroencephalogr Clin Neurophysiol.* 91, 353–362.

Althen H, Grimm S, Escera C. (2013) Simple and complex acoustic regularities are encoded at different levels of the auditory hierarchy. *Eur J Neurosci*. 38, 3448–3455.

Amenedo E, Diaz F. (1998) Effects of aging in middle latency evoked potentials: a cross-sectional study. *Biol Psychiatry.* 43, 210–219.

Anderson S, Parbery-Clark A, White-Schwoch T, Kraus N. (2012) Aging affects neural precision of speech encoding. *J Neurosci.* 32, 14156–14164.

Arehole S, Augustine LE, Simhadri R. (1995) Middle latency response in children with learning disabilities: preliminary findings. *J Commun Disord.* 28, 21–38.

Banai K, Kraus N. (2009) The dynamic brainstem: implications for auditory processing disorder. In: Cacace AT, McFarland DJ, eds. *Controversies in Central Auditory Processing Disorder.* San Diego, CA: Plural; pp 269–289.

Barajas JJ, Exposito M, Fernandez R, Marin LJ. (1988a) Middle latency response to a 400 Hz tone pip in normal-hearing and hearing impaired subjects. *Scand Audiol.* 17, 21–26.

Barajas JJ, Fernandez R, Bernal MR. (1988b) Middle latency and 40 Hz auditory evoked responses in normal hearing children: 500 Hz thresholds. *Scand Audiol Suppl.* 30, 99–104.

Başar E, Bullock TH. (1992) *Induced Rhythms in the Brain.* Boston, MA: Birkhauser.

Boutros NN, Belger A. (1999) Midlatency evoked potentials attenuation and augmentation reflect different aspects of sensory gating. *Biol Psychiatry.* 45, 917–922.

Boutros NN, Gjini K, Eickhoff SB, Urbach H, Pflieger ME. (2013) Mapping repetition suppression of the P50 evoked response to the human cerebral cortex. *Clin Neurophysiol.* 124, 675–685.

Boutros NN, Torello MW, Barker BA, Teuting PA, Wu S-C, Nasrallah HA. (1995) The P50 evoked potential component and mismatch detection in normal volunteers: implications for the study of sensory gating. *Psychiatr Res*. 57, 83–88.

Buchwald JS, Erwin RJ, Read S, Van Lancker D, Cummings JL. (1989) Midlatency auditory evoked responses: differential abnormality of P1 in Alzheimer's disease. *Electroencephalogr Clin Neurophysiol.* 74, 378–384.

Buzsáki G, Anastassiou CA, Koch C. (2012) The origin of extracellular fields and currents – EEG, ECoG, LFP and spikes. *Nat Rev Neurosc*i. 13, 407–420.

Cacace AT, Dowman R, Wolpaw JR. (1988) T complex hemispheric asymmetries: effects of stimulus intensity. *Hear Res.* 34, 225–232.

Cacace AT, McFarland DJ. (2002) Middle-latency auditory evoked potentials: basic issues and potential applications. In: Katz J, ed. *Handbook of Clinical Audiology.* 5th ed. Philadelphia, PA: Lippincott, Williams & Wilkins; pp 349–377.

Cacace AT, McFarland DJ. (2006) Frequency domain analysis of event related potentials and oscillations. In: Burkard RF, Don M, Eggermont JJ, eds. *Auditory Evoked Potentials: Basic Principles and Clinical Application.* Baltimore, MD: Lippincott, Williams & Wilkins; pp 124–137.

Cacace AT, McFarland DJ. (2013) Factors influencing tests of auditory processing: a perspective on current issues and relevant concerns. *J Am Acad Audiol.* 24, 572–589.

Cacace AT, Satya-Murti S, Wolpaw JR. (1990) Middle latency auditory evoked potentials: vertex and temporal components. *Electroencephalogr Clin Neurophysiol.* 77, 6–18.

Campbell JA, Leandri M. (1984) The effects of high pass filters on computer-reconstructed evoked potentials. *Electroencephalogr Clin Neurophysiol.* 57. 99–101.

Celesia GG. (1976) Organization of auditory cortical areas in man. *Brain*. 99, 403–414.

Celesia GG, Brigell M. (1999) Auditory evoked potentials. In: Niedermeyer E, Lopes da Silva F, eds. *Electroencephalography: Basic Principals, Clinical Applications and Related Fields.* 3rd ed. Baltimore, MD: Williams and Wilkins; pp 994–1013.

Cetas JS, de Venecia RK, McMullen NT. (1999) Thalamocortical afferents of Lorente de Nó: medical geniculate axons that project to primary auditory cortex have collateral branches to layer. *Brain Res.* 830, 203–208.

Chambers RD. (1992) Differential age effects for components of the adult auditory middle latency response. *Hear Res.* 58, 123–131.

Chambers RD, Griffiths SK. (1991) Effects of age on the adult auditory middle latency response. *Hear Res.* 51, 1–10.

Chandrasekaran B, Kraus N. (2010) The scalp-recorded brainstem response to speech: neural origins and plasticity. *Psychophysiology.* 47, 236–246.

Chang W-P, Gavin WJ, Davies PL. (2012) Bandpass filter settings differentially affect measurement of P50 sensory gating in children and adults. *Clin Neurophysiol.* 123, 2264–2272.

Chatrian GE, Petersen MC, Lazarte JA. (1960) Responses to clicks from human brain: some depth electrographic observations. *Electroencephalogr Clin Neurophysiol.* 12, 479–489.

Chen C, Ninomiya H, Onitsuka T. (1997) Influence of reference electrodes, stimulation characteristics and task paradigms on auditory P50. *Psychiatry Clin Neurosci.* 51, 139–143.

Dauman R, Szyfter W, Charlet de Sauvage R, Cazals Y. (1984) Low frequency thresholds assessed with 40 Hz MLR in adults with impaired hearing. *Arch Otorhinolaryngol*. 240, 85–89.

Dawson GD. (1951) A summating technique for detecting small signals in a large irregular background. *J Neurophysiol.* 115, 2–3.

Diesch E, Struve M, Rupp A, Ritter S, Hulse M, Flor H. (2004) Enhancement of steady-state auditory evoked magnetic fields in tinnitus. *Eur J Neurosci.* 19, 1093–1104.

Dolu N, Süer C, Ozesmi C. (2001) A comparison of the different interpair intervals in the conditioning-testing P50 paradigms. *Int J Psychophysiol.* 41, 265–270.

Eggermont JJ, Ponton CW, Don M, Waring MD, Kwong B. (1997) Maturational delays in cortical evoked potentials in cochlear implant users. *Acta Otolaryngol.* 117, 161–163.

Erwin R, Buchwald J. (1986) Midlatency auditory evoked responses: differential recovery cycle characteristics of sleep in humans. *Electroencephalogr Clin Neurophysiol.* 65, 383–392.

Freedman R, Adler LE, Gerhardt GA, Waldo M, Baker N, Rose GM, et al. (1987) Neurobiological studies of sensory gating in schizophrenia. *Schizophr Bull.* 13, 669–678.

Freedman R, Hall M, Adler LE, Leonard S. (1995) Evidence in postmortem brain tissue for decreased numbers of hippocampal nicotinic receptors in schizophrenia. *Biol Psychiatry.* 38, 22–33.

Galaburda A, Sanides F. (1980) Cytoarchitectonic organization of the human auditory cortex. *J Comp Neurol.* 190, 597–610.

Galambos R, Makeig S, Talmachoff PJ. (1981) A 40-Hz auditory potential recorded from the human scalp. *Proc Natl Acad Sci U S A.* 78, 2643–2647.

Galbraith GC, Arroyo C. (1993) Selective attention and the brainstem frequency following response. *Biol Psychol.* 37, 3–22.

Gencer NG, Williamson SJ, Gueziec A, Hummel R. (1996) Optimal reference electrode selection for electrical source imaging. *Electroencephalogr Clin Neurophysiol.* 99, 163–173.

Gerken GM. (1996) Central tinnitus and lateral inhibition: an auditory brainstem model. *Hear Res.* 97, 75–83.

Gerken GM, Hesse PS, Wiorkowski JJ. (2001) Auditory evoked responses in control subjects and in patients with problem tinnitus. *Hear Res.* 157, 52–64.

Gjini K, Boutros NN, Haddad L, Aikins D, Javanbakht A, Amirsadri A, et al. (2013) Evoked potential correlates of post-traumatic stress disorder in refugees with history of exposure to torture. *J Psychiatr Res.* 47, 1492–1498.

Goff WR. (1978) The scalp distribution of auditory evoked potentials. In: Naunton RF, Fernandez C, eds. *Evoked Electrical Activity in the Auditory Nervous System.* New York: Academic Press; pp 505–524.

Goff WR, Allison T, Lyons W, Fisher TC, Conte R. (1977) Origins of short latency auditory evoked potentials in man. In: Desmedt JE, ed. *Auditory Evoked potentials in Man. Psychopharmacology Correlates of Evoked Potentials. Prog Clin Neurophysiol.* Vol 2, Karger, Basel; 7, 30–44.

Gooding DC, Gjini K, Burroughs SA, Boutros NN. (2013) The association between psychosis proneness and sensory gating in cocaine-dependent patients and healthy controls. *Psychiatry Res.* 210 (3), 1092–1100.

Gordon KA, Papsin BC, Harrison RV. (2005) Effects of cochlear implant use on the electrically evoked middle latency response in children. *Hear Res.* 204, 78–89.

Gorga MP, Kaminski JR, Beauchaine KA, Jesteadt W. (1988) Auditory brainstem responses to tone bursts in normally hearing subjects. *J Speech Hear Res.* 31, 87–97.

Hackett TA, Stepniewska I, Kaas JH. (1999) Callosal connections of the parabelt auditory cortex in macaque monkeys. *Eur J Neurosci.* 11, 856–866.

Halpin C, Thornton A, Hasso M. (1994) Low-frequency sensorineural loss: clinical evaluation and implication for hearing aid fitting. *Ear Hear.* 15, 71–81.

Harris FJ. (1978) On the use of windows for harmonic analysis with the discrete Fourier transform. *Proc IEEE.* 66, 51–83.

Hashimoto I. (1982) Auditory evoked potentials from the human midbrain: slow brain stem responses. *Electroencephalogr Clin Neurophysiol.* 53, 652–657.

Hausler R, Cao M, Magnin C, Mulette O. (1991) Low frequency hearing threshold determination in newborns, infants, and

mentally retarded children by middle latency responses. *Acta Otolaryngol Suppl.* 482, 58–71.

Hjorth B, Rodin E. (1988) An eigenfunction approach to the inverse problem of EEG. *Brain Topogr.* 1, 79–86.

Hood LJ, Martin DA, Berlin CI. (1990) Auditory evoked potentials differ at 50 milliseconds in right- and left-handed listeners. *Hear Res.* 45, 115–122.

Hornickel J, Knowles E, Kraus N. (2011) Test-retest consistency of speech-evoked auditory brainstem responses in typically-developing children. *Hear Res.* 284, 52–58.

Jacobson GP, Privitera M, Neils JR, Grayson AS, Yeh HS. (1990) The effects of anterior temporal lobectomy (ATL) on the middle-latency auditory evoked potential (MLAEP). *Electroencephalogr Clin Neurophysiol.* 75, 230–241.

Jasper HH. (1958) The ten-twenty electrode system of the International Federation. *Electroencephalogr Clin Neurophysiol.* 10, 371–375.

Kaas JH, Hackett TA, Tramo MJ. (1999) Auditory processing in primate cerebral cortex. *Curr Opin Neurobiol.* 9, 164–170.

Karl A, Malta LS, Maercker A. (2006) Meta-analytic review of event-related potential studies in post-traumatic stress disorder. *Biol Psychol.* 71, 123–147.

Kelly AS, Purdy SC, Thorne PR. (2005) Electrophysiological and speech perception measures of auditory processing in experienced adult cochlear implant users. *Clin Neurophysiol.* 116, 1235–1246.

Kelly-Ballweber D, Dobie RA. (1984) Binaural interaction measured behaviorally and electrophysiologically in young and old adults. *Audiology.* 23, 181–194.

Kileny PR, Dobson D, Gelfand ET. (1983) Middle latency auditory evoked responses during open heart surgery with hypothermia. *Electroencephalogr Clin Neurophysiol.* 55, 268–276.

Kileny PR, Kemink JL. (1987) Electrically evoked middle-latency auditory evoked potentials in cochlear implant candidates. *Arch Otolaryngol.* 113, 1072–1077.

Kileny PR, Paccioretti D, Wilson AF. (1987) Effects of cortical lesions on middle-latency auditory evoked responses (MLR). *Electroencephalogr Clin Neurophysiol.* 66, 108–120.

Knight RT, Scabini D, Woods DL, Clayworth C. (1988) The effects of lesions of the superior temporal gyrus and inferior parietal lobe on temporal and vertex components of the human AEP. *Electroencephalogr Clin Neurophysiol.* 70, 499–509.

Kodera K, Hink RF, Yamada O, Suzuki JL. (1979) Effects of rise time on simultaneous recorded auditor-evoked potentials from the early, middle and late ranges. *Audiol.* 18, 395–402.

Korzyukov O, Pflieger ME, Wagner M, Bowyer SM, Rosburg T, Sundaresan K, et al. (2007) Generators of the intracranial P50 response in auditory sensory gating. *Neuroimage.* 35, 814–826.

Kraus N, Kileny P, McGee T. (1994) Middle latency auditory evoked potentials. In: Katz J, ed. *Handbook of Clinical Audiology.* 4th ed. Baltimore, MD: Williams & Wilkins; pp 387–405.

Kraus N, McGee T, Comperatore C. (1989) MLRs in children are consistently present during wakefulness, Stage I, and REM sleep. *Ear Hear.* 10, 339–345.

Kraus N, Özdamar O, Hier D, Stein L. (1982) Auditory middle latency responses (MLRs) in patients with cortical lesions. *Electroencephalogr Clin Neurophysiol.* 54, 275–287.

Kraus N, Smith D, Reed N, Stein L, Cartee C. (1985) Auditory middle latency responses in children: effect of age and diagnostic category. *Electroencephalogr Clin Neurophysiol.* 62, 343–351.

Kruglikov SY, Schiff SJ. (2003) Interplay of electroencephalogram phase and auditory-evoked neural activity. *J Neurosci.* 23, 10122–10127.

Law SK, Rohrbaugh JW, Adams CM, Eckardt MJ. (1993) Improving spatial and temporal resolution in evoked EEG responses using surface Laplacians. *Electroencephalogr Clin Neurophysiol.* 88, 309–322.

Lee YS, Leuders DS, Dinner RP, Lesser J, Hahn J, Klem G. (1984) Recording of auditory evoked potentials in man using chronic subdural electrodes. *Brain.* 107, 115–131.

Leinonen L, Joutsiniemi SL. (1989) Auditory evoked potentials and magnetic fields in patients with lesions of the auditory cortex. *Acta Neurol Scand.* 79, 316–325.

Lewine JD, Orrison WW. (1999) Magnetic source imaging: integration of magnetoencephalography and magnetic resonance imaging. In: Stark DD, Bradley WG, eds. *Magnetic Resonance Imaging.* Chapter 71, Vol. III, 3rd ed. St. Louis, MO: Mosby; pp 1575–1593.

Liegeos-Chauvel C, Musolino A, Badier JM, Marquis P, Chauvel P. (1994) Evoked potentials recorded from the auditory cortex in man: evaluation and topography of the middle latency components. *Electroencephalogr Clin Neurophysiol.* 92, 204–214.

Light GA, Malaspina D, Geyer MA, Luber BM, Coleman EA, Sackeim HA, et al. (1999) Amphetamine disrupts P50 suppression in normal subjects. *Biol Psychiatry.* 46, 990–996.

Lijffijt M, Moeller FG, Boutros NN, Burroughs S, Steinberg JL, Lane SD, et al. (2009a) A pilot study revealing impaired P50 gating in antisocial personality disorder. *J Neuropsychiatry Clin Neurosci.* 21, 328–331.

Lijffijt M, Moeller FG, Boutros NN, Burroughs S, Steinberg JL, Meier SL, et al. (2009b) Diminished P50, N100 and P200 auditory sensory gating in bipolar I disorder. *Psychiatry Res.* 167, 191–201.

Litvan H, Jensen EW, Galan J, Lund J, Rodriguez BE, Henneberg SW, et al. (2002) Comparison of conventional averaged and rapid averaged, autoregressive-based extracted auditory evoked potentials for monitoring the hypnotic level during propofol induction. *Anesthesiology.* 97, 351–358.

Lopes da Silva F. (1999) Event-related potentials: methodology and quantification. In: Niedermeyer E, Lopes da Silva F, eds. *Electroencephalography: Basic Principals, Clinical Applications and Related Fields.* 4th ed. Baltimore, MD: Williams and Wilkins; pp 947–957.

Lopes da Silva F, Van Rotterdam A. (1999) Biophysical aspects of EEG and magnetoencephalogram generation. In: Niedermeyer E, Lopes da Silva F, eds. *Electroencephalography: Basic Principals, Clinical Applications and Related Fields.* 4th ed. Baltimore, MD: Williams and Wilkins; pp 93–109.

Lorente de No R. (1947) Analysis of the distribution of action currents of nerve in volume conductors. *Stud Rockefeller Inst Med Res Repr.* 132, 384–482.

Margolis RH, Dubno JR, Hunt SM. (1981) Detection of tones in band-reject noise. *J Speech Hear Res.* 24, 336–344.

Mason SM, Mellor DH. (1984) Brain-stem, idle latency and late cortical evoked potentials in children with speech and language disorders. *Electroenceph Clin Neurophysiol.* 59, 297–309.

McFarland DJ, Cacace AT. (2004) Separating stimulus-locked and unlocked components of the auditory event related potential. *Hear Res.* 193, 111–120.

McFarland DJ, Cacace AT. (2011) Covariance is the proper measure of test-retest reliability. *Clin Neurophysiol.* 122, 1893–1894.

McFarland DJ, Cacace AT. (2012) Questionable reliability of the speech-evoked auditory brainstem response (sABR) in typically-developing children. *Hear Res.* 287, 1–2.

McFarland DJ, McCane LM, David SV, Wolpaw JR. (1997) Spatial filter selection for EEG-based communication. *Electroencephalogr Clin Neurophysiol.* 103, 386–394.

McFarland WH, Vivion MC, Wolf KE, Goldstein R. (1975) Reexamination of the effects of stimulus rate and number on middle components of the averaged electroencephalic response. *Audiology.* 14, 456–465.

McGee T, Kraus N. (1996) Auditory development reflected by middle latency response. *Ear Hear.* 17, 419–429.

McPherson DL, Starr A. (1993) Binaural interaction in auditory evoked potentials: brainstem middle- and long-latency components. *Hear Res*. 66, 91–98.

Mishkin M, Ungerleider LG, Macko KA. (1983) Object vision and spatial vision: two cortical pathways. *Trends Neurosci.* 6, 414–417.

Møller AR. (1988) *Evoked Potentials in Intraoperative Monitoring.* Baltimore, MD: Williams & Wilkins.

Møller AR. (1994) Neural generators of auditory evoked potentials. In: Jacobson JT, ed. *Principles and Applications in Auditory Evoked Potentials.* Boston, MA: Allyn and Bacon.

Møller AR, Jannetta PJ. (1983) Interpretation of brainstem auditory evoked potentials: results from intracranial recordings in humans. *Scand Audiol.* 12, 125–133.

Mosko SS, Knipher KF, Sassin JF, Donnelly J. (1984) Middle latency evoked potential in sleep apneics during waking and as a function of arterial oxygen saturation during apneas. *Sleep.* 7, 239–246.

Musiek FE, Baran J. (1987) Central auditory assessment: thirty years of challenge and change. *Ear Hear*. 8, 22–35.

Musiek FE, Geurkink NA, Weider DJ, Donnelly K. (1984) Past, present, and future applications of the auditory middle latency response. *Laryngoscope*. 94, 1545–1553.

Musiek FE, Lee WW. (1997) Conventional and maximum length sequences middle latency response in patients with central nervous system lesions. *J Am Acad Audiol.* 8, 173–180.

Nayak A, Roy RJ. (1999) Anesthesia control using midlatency auditory evoked potentials. *IEEE Trans Biomed Eng.* 45, 409–421.

Nelson MD, Jall JW III, Jacobson GP. (1997) Factors affecting the recordability of auditory evoked response components Pb (P1). *J Am Acad Audiol.* 8, 89–99.

Newman J, Moushegian G. (1989) Comparison of rate effects on the auditory middle latency response (MLR) in young and elderly adults. *J Acoust Soc Am.* 85, S38.

Ninomiya H, Onitsuka T, Chen C, Kinukawa N. (1997) Possible overlapping potentials of the auditory P50 in humans: factor analysis of middle latency auditory evoked potentials. *Electroencephalogr Clin Neurophysiol.* 104, 23–30.

Nunez PL. (1995) *Neocortical Dynamics and Human EEG Rhythms.* New York: Oxford University Press.

Nunez PL, Silbersrein RB, Cadusch PJ, Wijesinghe RS, Westdorp AF, Srinivasan RA. (1994) A theoretical and experimental study of high resolution EEG based on surface Laplacians and cortical imaging. *Electroencephalogr Clin Neurophysiol.* 90, 40–57.

Oates P, Stapells DR. (1997a) Frequency specificity of the human auditory brainstem and middle latency responses to brief tones. I. High-pass noise masking. *J Acoust Soc Am.* 102, 3597–3608.

Oates P, Stapells DR. (1997b) Frequency specificity of the human auditory brainstem and middle latency responses to brief tones. II. Derived response analyses. *J Acoust Soc Am.* 102, 3609–3619.

Onitsuka T, Ninomiya H, Sato E, Yamamoto T, Tashiro N. (2003) Differential characteristics of the middle latency auditory evoked magnetic responses to interstimulus intervals. *Clin Neurophysiol.* 114, 1513–1520.

Oranje B, Glenthøj BY. (2013) Clonidine normalizes levels of P50 gating in patients with schizophrenia on stable medication. *Schizophr Bull.* Epub ahead of print.

Özdamar Ö, Kraus N, Curry F. (1982) Auditory brain stem and middle latency responses in a patient with cortical deafness. *Electroencephalogr Clin Neurophysiol.* 53, 224–230.

Pandya DN. (1995) Anatomy of the auditory cortex. *Rev Neurol (Paris).* 151, 486–494.

Pandya DN, Rosene DL, Doolittle AM. (1994) Corticothalamic connections of auditory-related areas of the temporal lobe in the rhesus monkey. *J Comp Neurol.* 345, 447–471.

Papanicolaou AC, Loring DW, Eisenberg HM. (1984) Age-related differences in recovery cycle of auditory evoked potentials. *Neurobiol Aging.* 5, 291–295.

Parving A, Salomon G, Elberling C, Larsen B, Lassen NA. (1980) Middle components of the auditory evoked response in bilateral temporal lobe lesions. Report on a patient with auditory agnosia. *Scand Audiol.* 9, 161–170.

Pasman J, Rotteveel J, Maasen B, Graaf R, Visco Y. (1997) Diagnostic and predictive value of auditory evoked responses in preterm infants: II. Auditory evoked responses. *Pediatr Res*. 42, 670–677.

Pellizone M, Hari R, Makela JP, Huttunen J, Ahlfors S, Hamalainen M. (1987) Cortical origin of middle-latency auditory evoked responses in man. *Neurosci Lett.* 82, 303–307.

Perrault N, Picton TW. (1984) Event related potentials recorded from the scalp and nasopharynx. I. N1 and P2. *Electroencephalogr Clin Neurophysiol.* 59, 177–194.

Pfeifferbaum A, Ford JM, Roth Wt, Hopkins WF, Kopell BS. (1979) Event-related potential changes in healthy aged females. *Electroencephalogr Clin Neurophysiol.* 46, 81–86.

Picton TW, Dimitrijevic A, John MS. (2002) Multiple auditory steady-state responses. *Ann Otol Rhinol Laryngol Suppl.* 189, 16–21.

Picton TW, Hillyard SA. (1974) Human auditory evoked potentials. II. Effects of attention. *Electroencephalogr Clin Neurophysiol.* 36, 191–199.

Picton TW, Hillyard SA, Krausz HI, Galamabos R. (1974) Human auditory evoked potentials: I. Evaluation of components. *Electroencephalogr Clin Neurophysiol.* 36, 179–190.

Plourde G, Picton TW. (1990) Human auditory steady-state responses during general anesthesia. *Anesth Analg.* 71, 460–468.

Pool KD, Finitzo T, Hong CT, Rogers J, Pickett RB. (1989) Infarction of the superior temporal gyrus: a description of auditory evoked potential latency and amplitude topology. *Ear Hear.* 10, 144–152.

Pynchon KA, Tucker DA, Ruth RA, Barrett KA, Herr DG. (1998) Area-under-the-curve measure of the auditory middle latency response (AMLR) from birth to early adulthood. *Am J Audiol.* 7, 1–5.

Rauschecker JP, Tian B, Pons T, Mishkin M. (1997) Serial and parallel processing in rhesus monkey auditory cortex. *J Comp Neurol.* 382, 89–103.

Reite M, Teale P, Zimmerman J, Davis K, Whalen J. (1988) Source location of a 50 msec latency auditory evoked field component. *Electroencephalogr Clin Neurophysiol.* 70, 490–498.

Rivier F, Clarke S. (1997) Cytochrome oxidase, acetylcholinesterase, and NADPH-diaphorase staining for human supratemporal and insular cortex: evidence for multiple auditory areas. *Neuroimage.* 6, 288–304.

Roberts TP, Lanza MR, Dell J, Qasmieh S, Hines K, Blaskey L, et al. (2013) Maturational differences in thalamocortical white matter microstructure and auditory evoked response latencies in autism spectrum disorders. *Brain Res.* 1537, 79–85.

Ross B, Herdman AT, Pantev C. (2005) Stimulus induced desynchronization of human auditory 40-Hz steady-state responses. *J Neurophysiol.* 94, 4082–4093.

Ross B, Picton TW, Herdman AT, Hillyard SA, Pantev C. (2004) The effect of attention on the auditory steady state response. *Neurol Clin Neurophysiol.* 22, 1–4.

Rupp A, Gutschalk A, Uppencamp S, Scherg S. (2004) Middle latency auditory-evoked fields reflect psychoacoustic gap detection thresholds in human listeners. *J Neurophysiol.* 92, 2239–2247.

Saenz M, Langers DR. (2014) Tonotopic mapping of human auditory cortex. *Hear Res.* 307, 42–52.

Scherg M. (1982) Simultaneous recording and separation of early and middle latency auditory evoked potentials. *Electroencephalogr Clin Neurophysiol.* 54, 339–341.

Scherg M, von Cramon D. (1986a) Evoked dipole source potentials of the human auditory cortex. *Electroencephalogr Clin Neurophysiol.* 65, 344–360.

Scherg M, von Cramon D. (1986b) Psychoacoustic and electrophysiologic correlates of central hearing disorders in man. *Eur Arch Psychiatry Neurol Sci.* 236, 56–60.

Scherg M, von Cramon D. (1990) Dipole source potentials of the auditory cortex in normal subjects and in patients with temporal lobe lesions. In: Grandori F, Hoke M, Romani GL, eds. *Auditory Evoked Magnetic Fields and Electric Potentials.* Basel: Karger, pp 165–193.

Schochat E, Musiek FE, Alonso R, Ogata J. (2010) Effect of auditory training on the middle latency response in children with (central) auditory processing disorder. *Braz J Med Biol Res.* 43, 777–785.

Setzen G, Cacace AT, Eames F, Riback P, Lava N, McFarland DJ, et al. (1999) Central deafness in a young child with Moyamoya disease: paternal linkage in a Caucasian family. Two case reports and a review of the literature. *Int J Pediatr Otorhinolaryngol.* 48, 53–76.

Shehata-Dieler W, Shimizu H, Soliman SM, Tusa RJ. (1991) Middle latency auditory evoked potentials in temporal lobe disorders. *Ear Hear.* 12, 377–388.

Small SA, Stapells DR. (2004) Artifactual responses when recording auditory steady-state responses. *Ear Hear*. 25, 611–623.

Smucny J, Wylie K, Rojas D, Stevens K, Olincy A, Kronberg E, et al. (2013) Evidence for gamma and beta sensory gating deficits as translational endophenotypes for schizophrenia. *Psychiatry Res.* 214, 169–174.

Song JH, Banai K, Kraus N. (2008) Brainstem timing deficits in children with learning impairment may result from corticofugal origins. *Audiol Neurotol.* 13, 335–344.

Song JH, Banai K, Russo NM, Kraus N. (2006) On the relationship between speech- and nonspeech-evoked auditory brainstem responses. *Audiol Neurootol.* 11, 233–241.

Song JH, Nicol T, Kraus N. (2011) Test-retest reliability of the speech-evoked auditory brainstem response. *Clin Neurophysiol.* 122, 346–355.

Spink U, Johannsen HS, Pirsig W. (1979) Acoustically evoked potential: dependence on age. *Scand Audiol.* 8, 11–14.

Stapells DR, Galambos R, Costello JA, Makeig S. (1988) Inconsistency of auditory middle latency and steady-state responses in infants. *Electroencephalogr Clin Neurophysiol.* 71, 289–295.

Stephenson WA, Gibbs FA. (1951) A balanced noncephalic reference electrode. *Electroencephalogr Clin Neurophysiol.* 3, 237–240.

Stewart MG, Jerger J, Lew HL. (1993) Effect of handedness on the middle latency auditory evoked potential. *Am J Otol.* 14, 595–600.

Tallon-Baudry C, Bertrand O. (1999) Oscillatory gamma activity in humans and its role in object representation. *Trends Cogn Sci.* 4, 151–162.

Tanaka Y, Kamo T, Yoshida M, Yamadori A. (1991) "So-called" cortical deafness. *Brain.* 114, 2385–2410.

Taveras JM. (1996) *Neuroradiology.* 3rd ed. Baltimore, MD: Williams & Wilkins.

Thornton AR, Mendel MI, Anderson CV. (1977) Effects of stimulus frequency and intensity on the middle components of the averaged auditory electroencephalic response. *J Speech Hear Res.* 20, 81–94.

Thornton C, Sharpe RM. (1998) Evoked responses in anaesthesia. *Br J Anaesth.* 81, 771–781.

Toyoda K, Ibayashi S, Yamamoto T, Kuwabara Y, Fujishima M. (1998) Auditory evoked magnetic response in cerebrovascular disease: a preliminary study. *J Neurol Neurosurg Psychiatry.* 64, 777–784.

Truex RC, Carpenter MB. (1964) *Human Neuroanatomy.* Baltimore, MD: Williams and Wilkins.

Tsurukiri J, Nagata K, Okita T, Oomura T. (2013) Middle latency auditory-evoked potential index for predicting the degree of consciousness of comatose patients in EDs. *Am J Emerg Med.* 31 (11), 1556–1559.

Turner C, Burns EM, Nelson DA. (1983) Pure tone pitch perception and low-frequency hearing loss. *J Acoust Soc Am.* 73, 966–975.

Vivion MC, Hirsch JE, Frye-Osier JL, Goldstein R. (1980) Effects of rise-fall time and equivalent duration on middle components of AER. *Scand Audiol.* 9, 223–232.

Vizioli L, Bucciero A, Quaglietta P, Mosca F. (1993) Auditory middle latency responses in patients with intracranial space-occupying lesions. *J Neurosurg Sci.* 37, 77–81.

Weber F, Bein T, Hobbhahn J, Taeger K. (2004) Evaluation of the Alaris auditory evoked potential index as an indicator of anesthetic depth in preschool children during induction of anesthesia with sevoflurane and remifentanil. *Anesthesiology.* 101, 294–298.

Winer JA, Lee CC. (2007) The distributed auditory cortex. *Hear Res.* 229, 3–13.

Winer JA, Miller LM, Lee CC, Schreiner CE. (2005) Auditory thalamocortical transformation: structure and function. *Trends Neurosci.* 28, 255–263.

Wolpaw JR, Penry K. (1975) A temporal component of the auditory evoked potential. *Electroencephalogr Clin Neurophysiol.* 39, 609–620.

Wolpaw JR, Wood CC. (1982) Scalp distribution of human auditory evoked potentials: I. Evaluation of reference electrode sites. *Electroenceph Clin Neurophysiol.* 54, 15–24.

Wood CC, Wolpaw JR. (1982) Scalp distribution of human auditory evoked potentials. II. Evidence for overlapping sources and involvement of auditory cortex. *Electroencephalogr Clin Neurol.* 54, 25–38.

Woods DL, Clayworth CC. (1986) Age-related changes in human middle latency auditory evoked potentials. *Electroencephalogr Clin Neurophysiol.* 65, 297–303.

Woods DL, Clayworth CC, Knight RT, Simpson GV, Naeser MA. (1987) Generators of middle- and long-latency auditory evoked potentials: implications from studies of patients with bitemporal lesions. *Electroencephalogr Clin Neurophysiol.* 68, 132–148.

Woods DL, Knight RT, Neville HJ. (1984) Bitemporal lesions dissociate auditory evoked potentials and perception. *Electroencephalogr Clin Neurophysiol.* 57, 208–220.

Xu Z-M, De Vel E, Vinck B, Van Cauwenberge P. (1995) Selecting the best tone-pip stimulus-envelope time for estimating an objective middle-latency response threshold for low- and middle-tone sensorineural hearing losses. *Eur Arch Otorhinolaryngol.* 252, 275–279.

Yvert B, Fischer C, Bertrand O, Pernier J. (2005) Localization of human auditory areas from intracerebral auditory evoked potentials using distributed source models. *Neuroimage.* 28, 140–151.

CHAPTER 18

Cortical Auditory-Evoked Potentials

Kelly Tremblay and Christopher Clinard

INTRODUCTION, INSTRUMENTATION, AND IMPLEMENTATION

Over the past few decades, there has been a great deal of research aimed at identifying the neural mechanisms of central sound processing using various approaches including single-cell recordings in nonhuman primates, functional magnetic resonance imaging, and scalp recording of neuroelectric or neuromagnetic brain activity. We provide a review of the literature that is relevant to students, audiologists, and clinician scientists by focusing on cortical auditory-evoked potentials (CAEPs) and how they are used to (1) characterize the neural detection and/or discrimination of sound(s) and (2) assist with the assessment and rehabilitation of people with auditory-based communication disorders.

These potentials are sometimes called event-related potentials (ERPs, AERPs) or long-latency responses (LLRs) by psychologists. Time-locked evoked cortical activity provides information regarding the timing (through latency measurement) and salience (through amplitude measurement) of sound processing. To some extent, information regarding the location of processing may be determined by means of scalp topography, brain source modeling, and statistical inferences (Hillyard and Picton, 1987). Many ERPs are not sensory, specific, meaning they can be evoked by more than one type of stimulus modality and therefore can be used to study multisensory processing (e.g., auditory visual integration). Because they are used to measure sensory, cognitive, and motor events, neurologists and psychologists also use CAEPs to study brain and behavior relationships underlying human communication as well as related disorders.

In the sequence of auditory-evoked potentials (AEPs), LLRs occur 50 ms or later after the stimulus; each deflection is identified according to its polarity, order of occurrence, and latency. For example, the P1-N1-P2 cortical response consists of a small positive wave (P1), a large negative component (N1), followed by a positive peak (P2). The N1 peak, also referred to as the N100, is a negative peak that occurs approximately 100 ms following sound onset (Figure 18.1). Neuroscientists sometimes display polarities in the reverse direction, with positive polarities down and negative peaks up. The reason for this difference has more to do with historical and cultural differences between hearing and cognitive scientists and less to do with the science. Therefore, the interpretation of the CAEPs remains the same, regardless of how polarity is displayed.

What Do the Latencies and Amplitudes of an AEP Imply?

CAEPs are said to result from stimulus-locked postsynaptic potentials within apical dendrites of pyramidal neurons in the cerebral cortex. As described by Eggermont (2001), and shown in Figure 18.2, extracellular electric currents spread through the conductive brain tissue, the cerebrospinal fluid, the skull, and the skin, resulting in voltage differences at the scalp surface, which are recorded using electrodes placed on the scalp. The number of activated neurons, extent of neuronal activation, and synchrony of the neural response all contribute to the resulting CAEP pattern. The amplitude of each CAEP component quantifies the strength of the response and is measured in microvolts (μV). Latency

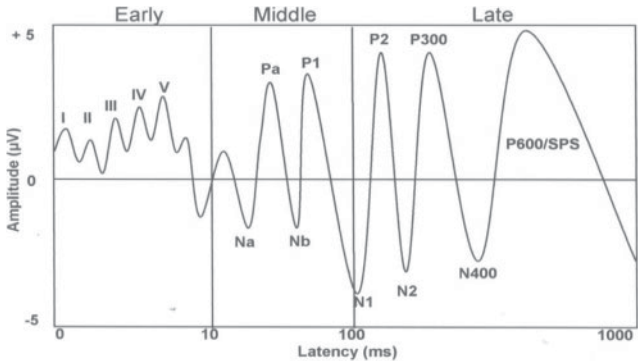


FIGURE 18.1 A hypothetical illustration of auditory-evoked potentials, from early responses occurring at the level of the auditory nerve and brainstem to the later cortical responses. [Reprinted with permission from Friesen L, Tremblay K. (2003) Electrophysiologic measures of speech, language and hearing. *Perspect Neurophysiol Neurogenic Speech Lang Disord.* 13 (1), 3–10.]

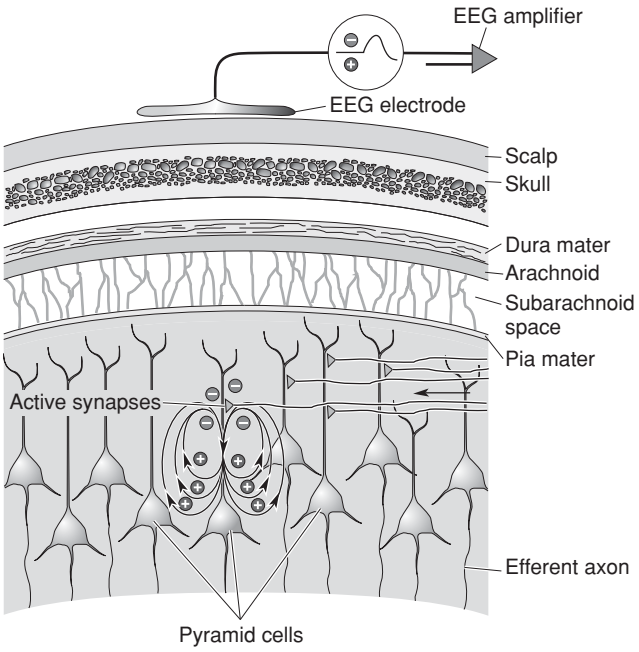


FIGURE 18.2 An illustration of a neural dipole, representing the neural activity that scalp electrodes are able to detect. [Reprinted with permission from Bear M, Connors B, Pardiso M. (2007) *Neuroscience: Exploring the Brain.* 3rd ed. Philadelphia, PA: Lippincott Williams and Wilkins.]

refers to the amount of time, in milliseconds (ms), that it takes to generate the bioelectrical response following stimulus onset. Latency is therefore related to neural conduction time and the location of the neural generator: The time it takes for the sound to travel through the peripheral auditory system to the place of excitation in the central auditory system. Even though they are not routinely utilized in clinical settings, long-latency ERPs are regularly used in research to determine how physical acoustic energy translates into patterns of brain activity and contributes to perception in normal and hearing-impaired listeners (see Burkard et al., 2007, Chapter 11 for more about recording and analysis of auditory CAEPs).

Whereas the auditory brainstem response (ABR) has been used to examine synaptic events related to more peripheral sensory function (bottom up—afferent), longer latency cortical CAEPs have been used to examine more central processes related to how the brain makes use of the sound (top down—efferent). The boundaries between bottom-up and top-down cortical contributions are becoming increasingly blurred as increasing numbers of integrated neural networks, involving afferent and efferent processes, are defined.

Exogenous and Endogenous Contributions

CAEPs can be described as being sensitive to exogenous (acoustic representation) or endogenous (attention and learning) aspects of sound processing. Exogenous can be defined as brain activity that is influenced by external sources, like the decibel level of a signal. Endogenous refers to brain activity modulated by internal events like motivation and alertness. In this respect, earlier latency responses like the ABR are described as exogenous because they are sensitive to the intensity level and other acoustic characteristics (e.g., rise time) of the incoming signal and are relatively insensitive to subject state of the individual. For example, the ABR can be used to estimate hearing sensitivity thresholds in infants during sleep. Despite these exogenous aspects of the ABR, and the recording parameters used to capitalize on these strengths for the purpose of estimating hearing sensitivity, there is also evidence to show that efferent contributions (descending cortical to brainstem neural networks) modulate brainstem activity. When studying brainstem activity contributing to the frequency-following response (FFR), sometimes referred to as the complex auditory brainstem response (cABR), there is evidence that short- (over days/weeks) and long-term (over years) listening experience influences activity of the auditory brainstem (for a review, see Chandrasekaran and Kraus, 2010). So, it can be said that evoked potentials have endogenous and exogenous aspects, but the resultant AEPs that are recorded depend on the methods used to acquire them.

If the purpose is to use CAEPs to estimate perceptual abilities in difficult-to-test clinical populations that cannot provide a reliable behavioral response, then recording CAEPs in a "passive" paradigm is typically used. Here, the term "passive" refers to the collection of brain activity without an active response of the subject. Passive recordings are used for many different reasons, especially when behavioral measures might not be possible (e.g., coma and dementia) or when there is suspicion of malingering. An ABR protocol for estimating hearing sensitivity in infants is one example of a passive paradigm because the infant is not expected to respond in any way. Even if perception was possible to measure, it would be difficult to determine if impaired behavioral responses were the result of cognitive deficits, fatigue, malingering, or other potential contributing factors. Therefore, "passive" electroencephalography (EEG) recordings can be seen as an opportunity to measure the physiological capacity of the auditory system, independent of a perceptual task, to determine if there is biologic evidence of abnormal physiological processing that might be contributing to impaired perception. If there is evidence of abnormal physiological encoding of sound, then rehabilitation efforts could center on improving the neural detection and discrimination of sound. However, if the neural detection/discrimination of sound appears normal, then rehabilitation efforts can focus on making better use of this physiological capacity.

When there is no active participation on the part of the test subject, these "passively" recorded CAEPs are often called "preattentive"; however, the use of this term is not entirely accurate because one cannot control or quantify all attentive

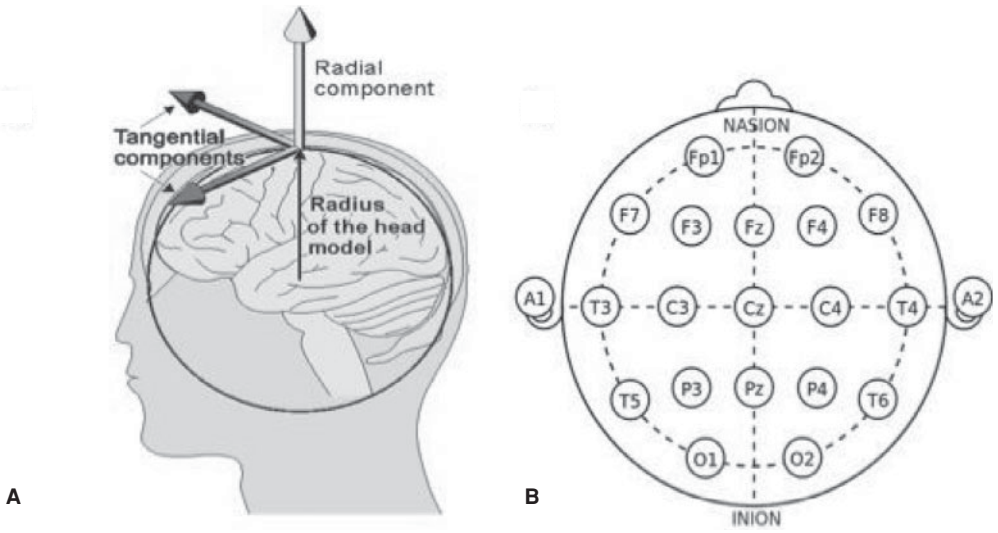


FIGURE 18.3 **A:** Illustration showing radial and tangential components. **B:** The 10–20 electrode placement system. [Reprinted with permission from Malmivuo J, Suihko V, Eskola H. [1997] Sensitivity distributions of EEG and MEG measurements. *IEEE Trans Biomed Eng.* 44 [3], 196–208.]

processes. For example, the mismatch negativity (MMN), described later in this chapter, involves a "passive" paradigm where oddball (deviant) stimuli are presented within a series of similar (standard) stimuli (e.g., da, da, da, da, ga, da, da, da, …). The deviant sound can differ from the standards in one or more perceptual features such as pitch, duration, loudness, or phonemic content. The purpose of the MMN is to determine if a person's auditory system is able to detect the change in stimuli, evoked by the oddball /ga/ stimulus in this example. The presence of an MMN, signaling the neural detection of the deviant stimuli, is described as a noninvasive, preattentive, task-independent, physiological correlate of stimulus discrimination and auditory sensory memory. Because its acquisition does not require an active task, the MMN has been used to study "preattentive" auditory processing in clinical populations (e.g., children with specific language impairment (SLI)) suspected of abnormal auditory physiological discrimination capabilities (Naatanen et al., 1978, 2007). The same oddball paradigm can evoke what is called a P300 if the participant is asked to signal the presence of the deviant stimulus, either through a motor task or by counting, during "active" EEG recording. An argument in favor of using "active", rather than "passive", EEG recordings is that one should record neural mechanisms that are activated during the perceptual task to truly understand how the brain is contributing to perception. Therefore, the type of recording paradigm and CAEP that is measured depend on the user's intended purpose.

EEG versus MEG

When reviewing the literature, readers will note that the CAEPs described in this chapter can be recorded using EEG or magnetoencephalography (MEG). When the N1 CAEP is recorded using MEG, for example, scientists sometimes report it as the N1m. In many ways, the interpretation and functional significance of the EEG and MEG CAEP waveforms are similar; but the methods used to obtain them as well as the neural mechanisms that contribute to them differ (Malmivuo et al., 1997). Whereas scalp EEG is sensitive to both tangential and radial components of a current source (Figure 18.3), MEG detects only its tangential components. This means scalp EEG is sensitive to activity in the sulci and cortical gyri, but MEG is most sensitive to activity originating in sulci. EEG is therefore described as being sensitive to activity in more brain areas, but activity that is visible in MEG can be localized with more accuracy. Because magnetic fields are less distorted than electric fields by the skull and scalp, MEG is said to provide better spatial resolution than EEG. The typical needs of the audiologist involve EEG because it is far more feasible and cost-effective than MEG. Thus, the remaining focus of this chapter will be on acquisition of CAEPs involving EEG.

Electrode Configurations and Acquisition Parameters

Clinical EEG systems are more limited in functionality than research EEG systems. Clinic devices feature ease of use, but flexibility is often sacrificed. For example, some clinical systems only have a small number of channels, do not provide the opportunity to present sounds in oddball paradigms, and do not permit data analysis offline. Clinical EEG systems often do not permit the use of customized sounds and so the user is limited to the type of stimuli they can use.

Research systems, although more expensive, have greater flexibility and permit the ability to present all types of stimuli and allow offline processing so data can be analyzed in a multitude of ways.

CAEPs are typically recorded using the 10–20 system or International 10–20 system. It is an internationally recognized method that is used to describe the placement of electrodes on the scalp. A standardized application method assures reproducibility across multiple test sessions as well as across different clinics and laboratories. Each electrode site has a letter to identify the brain region over which it is placed, as well as a number to identify the hemisphere location (odd numbers = left hemisphere). The letters F, T, C, P, and O represent frontal, temporal, central, parietal, and occipital regions, and the "10" and "20" refer to the distances between adjacent electrodes (10% or 20% of the total front–back or right–left distance of the skull). Although these electrode location labels provide location information pertaining to the scalp, this should not be confused with internal neural generator sources. CAEPs recorded from electrode F3, for example, reflect postsynaptic activity generated from sources outside of the left frontal lobe. "Z" (zero) refers to an electrode placed on the midline.

Audiologists generally use a small number of electrodes for ABR use, but many other clinical professions will use 20 or more for various types of neurologic evaluations. It is advantageous to use a smaller number of electrodes as it requires less preparation and cleaning time, and fewer channels can mean that a less expensive piece of EEG equipment is necessary. However, research should begin with high-density recording arrays (typically, via cap or net containing up to 256 electrodes), otherwise, relevant information will be missed. As an example, the N1 vertex potential has historically been described as a reliable and stable CAEP that does not change from test session to test session. This is true when recorded from electrode site Fz and other frontal-central midline locations; however, when viewed from temporal electrodes (e.g., T3 and T5), amplitude increases from session to session are evident (Tremblay et al., 2010). Changes in waveform morphology across different areas of the scalp are not necessarily viewed as a confound; with regard to the study of auditory exposure and auditory learning, the P1-N1-P2 is proving to be a relevant CAEP. So whereas it is important to design recording montages that are efficient and feasible for clinical application, the development of clinical CAEP protocols should start with high-density recordings (e.g., 64 channels). These protocols should then be further refined to a set of optimized recording parameters (e.g., fewer channels) that are more clinically feasible.

Electrical and myogenic noise can interfere with CAEP recordings. In addition to 60-Hz electrical line noise, eyeblink artifact is a major source of noise in CAEPs and should be monitored. Electrical activity related to eyeblinks may be recorded by using electro-oculograms (EOG) with vertical (VEOG) and/or horizontal (HEOG) electrodes placed in the vertical or horizontal planes of the eye, respectively. These electrodes may be placed close to the inner and outer canthi, as well as above the eyebrow and below the eye. EOG electrodes can help to adjust artifact rejection so that any sweeps with eyeblinks can be rejected. Eyeblink artifact is large; it is seen most clearly in frontal electrodes, although it can be picked up by electrodes across a wide area of the scalp.

Similar to ABR, each electrode is connected to one input of a differential amplifier (one channel per pair of electrodes); a common system reference electrode is connected to the other input of each differential amplifier. The voltage between the active, inverting electrode and the reference, noninverting electrode is amplified typically 1,000 to 100,000 times, or 60 to 100 dB of voltage gain. Analog-to-digital sampling typically occurs at 256 to 512 Hz in clinical scalp EEG for cortical AEPs; sampling rates of up to 20 kHz are used in some AEP research applications.

TYPES OF CAEPS

What follows is a brief overview of the auditory CAEPs including a description of each component and information that each AEP component provides to audiologists regarding speech-processing capabilities of their patients. Table 18.1 provides a cursory comparison to highlight how CAEP components can be distinguished from each other. Tables 18.2 and 18.3 provide the reader with recording protocols to guide data collection. The reader is also directed to publications that describe recommended methods for acquiring CAEPs as they pertain to central disorders, including psychiatric and neurologic disorders. Published guidelines are intended to assist investigators who use ERPs in clinical research, in an effort to standardize methodology and facilitate the comparability of data across laboratories. Duncan et al. (2009), for example, describe recording techniques to elicit, record, and quantify three major cognitive components MMN, P300, and N400. Picton et al. (2000) provide recording standards and publication criteria when using human ERPs to study human perception and cognition.

P1-N1-P2

Of all of the CAEPs, the P1-N1-P2 complex has received a great deal of attention by audiologists and clinician scientists because it is similar to the ABR in that it reflects exogenous properties of the incoming signal, making it relevant to the estimation of hearing threshold levels. It is an excellent tool to assess suspected functional hearing loss, as well as the neural detection of sound and its acoustic phonetic components at more central levels than the ABR (Hyde, 1997). As described by Picton (1994), much of today's AEP history began with the P1-N1-P2 complex. It was Hallowell and Pauline Davis who made the first recordings of the

TABLE 18.1

A Comparison of the P1-N1-P2 Onset Response and the ACC, MMN, and P300

Issue	P1-N1-P2 Onset	ACC	MMN	P3
What it indexes	Encoding (detection)	Encoding (detection) of acoustic change–discrimination capacity	Preattentive discrimination requires a sensory memory of the standard	Further processing of consciously discriminated sounds
Minimum number of electrodes	At least three: Cz, ground, reference (nose or one mastoid)	At least three: Cz, ground, reference (nose or one mastoid)	At least five: Fz, ground, reference, vertical eye channel. Mastoids (to examine response inversion) and additional electrodes (to examine scalp distribution) helpful for response identification	At least five: Pz, ground, reference, vertical eye channel. More channels permit examination of scalp distribution and are helpful for response identification
Elicited by	Sound onset	Acoustic change(s)	Stimulus or pattern deviance	Task-relevant deviance
Present when	Sound is detectable	Acoustic change is detectable	Stimulus deviance is detectable (some exceptions)	Stimulus deviance attended, detectable, and task relevant
Absent when	Sound not detectable or neurologic disorder	Acoustic change not detectable, presumably in some neurologic disorders	Stimulus deviance not detectable (some exceptions)	Stimulus deviance not attended and/or not task relevant in some psychiatric and neurologic disorders
Good at individual subject level?	Yes	Yes	No	Usually
Appropriate for young children?	Yes–may not obtain typical P1-N1-P2 pattern	Preliminary data indicate yes	Yes for groups, no at the individual subject level	No
Oddball paradigm required?	No	No	Yes	Yes
No. sweeps needed?	~100+ sweeps	~100+ sweeps	~200+ deviants	~100+ deviants
Test–retest reliability	Good	Good	Fair (better for adults than children)	Fair to good
Test time	Fast	Fast	Long	Medium
Ready to use in the clinic?	Yes to estimate threshold: yes to index pathway integrity; no for fine-grained diagnosis	No, but it is likely within 3–5 yrs	No for individuals; yes for groups	No for individuals; yes for groups
Biggest problem for clinical use	Lack of norms maturation effects	Lack of norms	Reliability at individual subject level particularly in children	Lack of norms

Reprinted with permission from Martin BA, Tremblay KL, Korczak P. (2008) Speech evoked potentials: from the laboratory to the clinic. *Ear Hear. 29* (3), 285–313.

TABLE 18.2

Recommended Recording Parameters for P1-N1-P2

Subjects	State	Awake and alert
	Eyes	Open during recording
	Attention	Ignore stimuli
Stimuli	Interonset interval	0.05–0.20 ms
	Stimulus duration	100–1,000 ms
	Intensity	50–300 ms 60–80 dB peSPL
Recordings	Channels	Minimum of four channels (Fz, Cz, mastoid, and vertical EOG) or electrode cap
	Reference electrode	Electrically neutral (tip of nose or average reference)
	Eye artifact	Record vertical EOG for eyeblink artifact, also horizontal EOG if possible
	Filters	0.1–100 Hz (online), 1–30 Hz (offline)
	Amplifier gain	10,000–30,000
	Time window	–50 to at least 400 ms, depending on stimuli
Response detection	Visual detection	Recordings replicate well Scalp topography should be appropriate Response should be two to three times larger than the prestimulus baseline
	Statistical detection	Statistical detection is preferred over visual detection
Measurements	Many options (see Duncan et al., 2009; Picton et al., 2000)	N1 latency and baseline-to-peak amplitude, integrated MMN, area under MMN, MMN duration, MMN peak latency, and amplitude For group data, use a grand mean MMN waveform to establish the latency of the MMN

human AEPs, beginning with the N1 CAEP (also called the vertex potential). In 1939, they published the founding papers for evoked potential audiometry but it was not until the 1960s when averaging computers made their recordings sufficiently objective for clinical use. In the 1960s and early 1970s, while studying the effects of attention on this response, Hallowell Davis also recorded some of the first recordings of the P300 wave, a wave that is related to human information processing. So why then, as pointed out by Hyde (1997), is not the P1-N1-P2 used more often in clinic? Probably because clinicians are usually not trained in the use of them in their audiology programs.

The P1 component of the P1-N1-P2 complex typically starts at about 50 ms following stimulus onset in adults with normal hearing. Ross and Tremblay (2009) showed different source locations for auditory-evoked N1 and P2 sources using MEG with the auditory P2 being generated at least in part in the auditory cortex, the temporal region, and the reticular activating system. P1-N1-P2 response analysis includes the measurement of latency and amplitude of each individual peak component; however, there are also objective detection algorithms that can be used. One example is a wavelet-based Rayleigh test on phase coefficients (Ross et al., 2007). When high-density EEG or MEG recordings are used, analyses such as source localization and dipole modeling may be carried out (e.g., BESA or LORETA).

When high-density recordings and modeling are used, multiple subcomponents contributing to the P1-N1-P2 can be defined (Woods, 1995). For example, N1 has multiple subcomponents with N1a, at approximately 75 ms poststimulus onset, being most prominent at temporal electrodes. N1b (at about 100 ms) is most prominent at vertex electrodes and is the peak most often described in the literature pertaining to audiology and hearing science. The N1c subcomponent (at approximately 130 ms) is most prominent at temporal electrodes but extends to fronto-polar and lateral central electrodes. Much of the research that appears in the audiology and hearing science literature pertains to the vertex recorded N1 (also known as N1b), even though it is not explicitly called N1b in many publications. This is also true when reviewing the P1-N1-P2 complex in this chapter. When using the term N1, we are mostly referring to the N1b vertex potential.

TABLE 18.3

Recommended Recording Parameters for MMN

Subjects	State	Awake and alert
	Eyes	Open during recording
	Attention	Ignore stimuli
Stimuli	Deviant stimuli	Use oddball paradigm with~200 sweeps minimum to deviant, and deviant probability of 0.05 to 0.20
	Interonset interval	100–1,000 ms
	Stimulus duration	50–300 ms
	Intensity	60–80 dB peSPL
Recordings	Channels	Minimum of four channels (Fz, Cz, mastoid, and vertical EOG) or electrode cap
	Reference electrode	Electrically neutral (tip of nose or average reference)
	Eye artifact	Record vertical EOG for eyeblink artifact, also horizontal EOG if possible
	Filters	0.1–100 Hz (online), 1–30 Hz (offline)
	Amplifier gain	10,000–30,000
	Time window	–50 to at least 400 ms, depending on stimuli
Response detection	Waveform subtraction	(Waveform for deviant stimuli)–(waveform for standard stimuli)
	Visual detection	Recordings replicate well Scalp topography should be appropriate for MMN Response should be two to three times larger than the prestimulus baseline
	Statistical detection	Statistical detection is preferred over visual detection
Measurements	Many options (Duncan et al., 2009; Picton et al., 2000)	N1 latency and baseline-to-peak amplitude, integrated MMN, area under MMN, MMN duration, MMN peak latency, and amplitude For group data, use a grand mean MMN waveform to establish the latency of the MMN

The P1-N1-P2 response is an onset response and can also be described as a change detector because it is elicited by the (1) change from silence to sound (onset), (2) transitions with an ongoing sound, and (3) sound to silence (offset). The acoustic contents of the stimulus (e.g., frequency, duration, rise/fall times) all affect response morphology of the P1-N1-P2 peaks. For example, rise/fall times less than 50 ms and stimulus durations of at least 30 ms elicit the most robust responses. Slower rates are associated with larger P1-N1-P2 amplitudes. Binaural stimulation elicits responses with larger amplitudes than monaural stimulation. Although the P1-N1-P2 is described as having exogenous aspects, there can also be endogenous contributions that involve attention. Active recordings, for example, where the individual is instructed to attend to a signal in one ear while ignoring a competing message in the other ear, will also enhance N1 amplitude (Hink and Hillyard, 1976). Figure 18.4 shows passively evoked P1-N1-P2 responses elicited by a 1-kHz tone 750 ms in duration. The tone, because of its duration, evokes onset and offset P1-N1-P2 responses that decrease in amplitude and increase in latency with decreasing intensity levels.

Table 18.2 provides a list of acquisition parameters used to record P1-N1-P2 responses. It is traditionally recorded using a block paradigm, where the same stimulus is repeated a number of times. CAEPs are usually larger in amplitude than the ABR; so fewer sweeps are necessary to obtain favorable signal-to-noise ratios (SNRs) (e.g., 50 to 300 sweeps). Response amplitude declines as a sound is repeated, thought to be related to neuronal habituation and adaptation processes with the amplitude decrement being most robust over the first five sweeps (Bourbon et al., 1987; Ritter et al., 1968). Unlike the ABR, the P1-N1-P2 complex is less robust when patients are asleep. Response amplitudes are most robust when patients are awake, even if they are not attending to the stimulus. For this reason, during passive recordings, individuals are asked to read a magazine or watch a closed-captioned video during the recording to reduce drowsiness, which can also affect response amplitude.

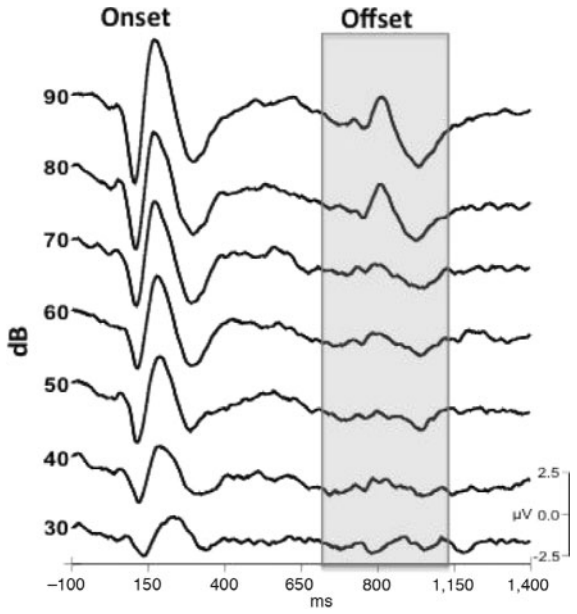


FIGURE 18.4 P1-N1-P2 responses elicited by a 1-kHz tone 750 ms in duration. The tone, because of its duration, evokes onset and offset P1-N1-P2 responses that decrease in amplitude and increase in latency with decreasing intensity levels. [Reprinted with permission from Billings CJ, Tremblay KL, Souza PE, Binns MA. (2007) Effects of hearing aid amplification and stimulus intensity on cortical auditory evoked potentials. *Audiol Neurootol.* 12 (4), 234–246].

P1-N1-P2 Change Complex

The P1-N1-P2 is also called an obligatory potential because its presence signals the first stages of neural detection. It does not provide information about stimulus discrimination, and therefore provides little information about speech discrimination. Instead its presence is followed by discriminatory potentials such as the MMN, P300, N400, and P600 that do reflect higher order auditory discrimination processing. With that said, it is possible to use the P1-N1-P2 to measure the neural discrimination of suprathreshold acoustic changes if long-time duration time-varying stimuli are used. Sometimes referred to as the acoustic change complex (ACC) (Ostroff et al., 1998), and originally introduced decades ago (e.g., Jerger and Jerger, 1970), the ACC signals the neural detection of stimulus change. In this respect it is a discriminatory potential because the eliciting stimulus contains acoustic contrasts, but does so using one stimulus rather than using two separate sounds like those used in the oddball paradigm for MMN and P300. Figure 18.5 shows how the acoustic change response can be used to quantify the neural detection of sound onset (silent to sound) as well as the detection of an acoustic change contained in an 800-ms duration vowel (seen here in a boxed area). At the 400-ms midpoint, the second formant frequency of the vowel changed creating a transition from /u/ to /i/.

Even though the P1-N1-P2 change response is not a direct measure of perception, many studies have begun to clarify the relationship between the presence of a change response and that same individual's behavioral threshold. For example, a number of studies have related an individual's P1-N1-P2 change response to that same individual's behavioral detection of the same stimulus contrast. Whereas the neural detection of sound onset evoked by short-duration stimuli has not proven to be a strong predictor of perception, good relationships between behavioral and physiological "change responses" of intensity, frequency, interaural phase, and spectral-ripple density have been documented (Harris et al., 2008; Ross et al., 2007; Won et al., 2011). As an example, Figure 18.6 shows the behavioral detection of interaural phase differences (IPDs) in relation

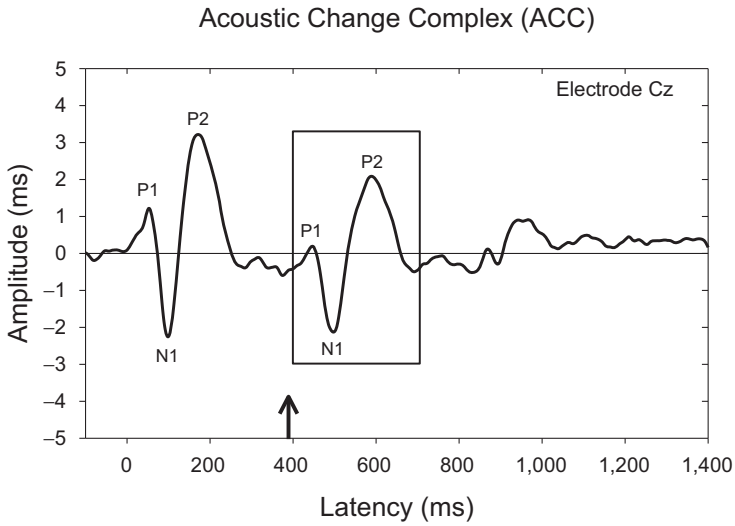


FIGURE 18.5 The P1-N1-P2 response acts as a change detector and here shows onset, change, and offset responses elicited by a 800-ms duration vowel stimulus. At the 400-ms midpoint, the second formant frequency of the vowel was altered, changing the vowel sound from /u/ to /i/. The onset of the sound elicits an onset response, detecting a change from silence to sound. The acoustic change at the midpoint of the vowel elicits a second P1-N1-P2 complex called the change response, indicating that the central auditory system detected the formant frequency change midway through the vowel. The offset of the sound elicits an offset response, smaller in amplitude around 900 ms, indicating the detection from sound to silence. [Modified from Martin BA, Tremblay KL, Korczak P. (2008) Speech evoked potentials: from the laboratory to the clinic. *Ear Hear.* 29 (3), 285–313.]

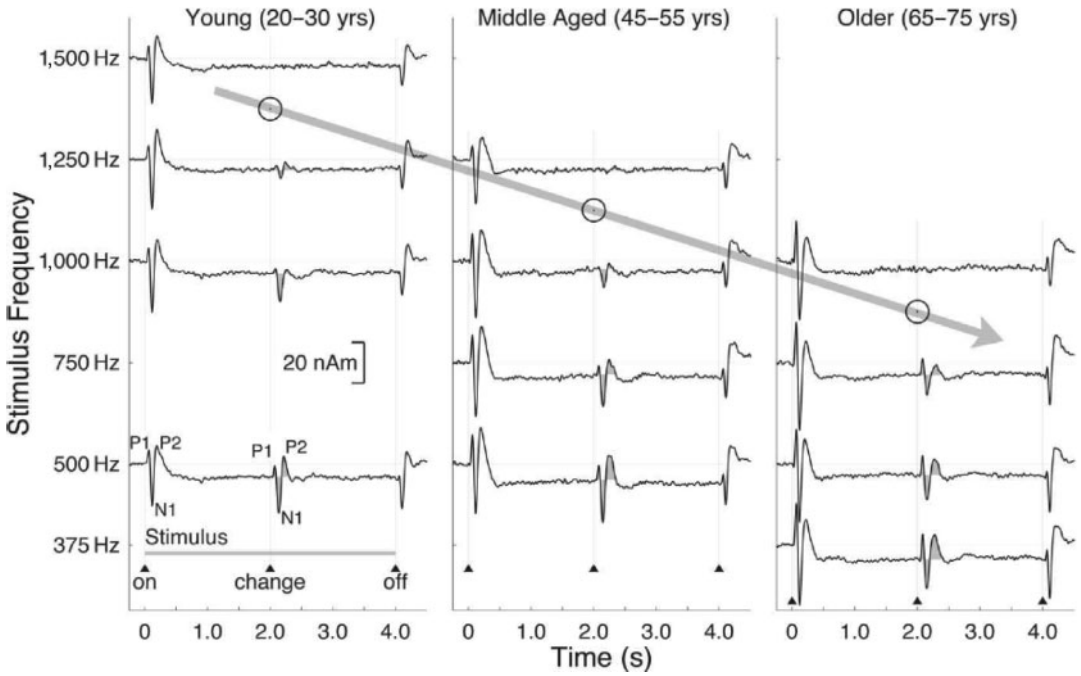


FIGURE 18.6 P1-N1-P2 responses for three age groups at different stimulus frequencies. Clear onset responses are observed for all groups at all test frequencies but the ability to detect an interaural phase difference (IPD) arriving at both ears diminishes as age increases. The stimuli are diodic at the beginning of the stimulus (shown in the *bottom left hand corner*), creating an onset response. The "change" response is evoked by an IPD into a dichotic signal halfway through the stimulus. (Reprinted with permission from Ross B, Fujioka T, Tremblay K, Picton TW. (2007) Aging in binaural hearing begins in mid-life: evidence from cortical auditory-evoked responses to changes in interaural phase. *J Neurosci.* 27 (42), 11172–11178.)

to the physiological detection of IPDs. For each carrier stimulus frequency, shown in the *y*-axis, there is a 180° IPD at the 2-second midpoint of the stimulus. P1-N1-P2 change responses are seen in response to stimulus onset, change, and offset. In this study, the change response is present if the central auditory system was able to detect the IPD that occurred midway within the stimulus. Open, black circles represent group averages for the upper frequency limit at which subjects made this detection behaviorally (recorded in a separate session). Note that no change responses were seen at the highest frequencies, where group members were not able to behaviorally detect the phase change. Based on these results, it can be said that the upper frequency limit of IPD detection deteriorates with advancing age. It is important to note that the P1-N1-P2 onset response (latency, amplitude) does not approximate a person's ability to perceive these sounds; but rather, it is the "change" response that corresponds well to a person's behavioral ability to discriminate the stimuli. In other words, the change from silence to sound is so robust that all age groups can detect it. It is the change in acoustic information, from one auditory signal to another, which shows age-related decreases in temporal processing related to the neural encoding of IPD. Similar approaches using gap detection, voice-onset time, and other time-varying stimuli have been used to study temporal processing in older adults, with and without hearing loss, and have yielded similar findings (for a review, see Billings et al., 2012a; Picton, 2013).

Advantages of using ACC over other discriminative responses such as the MMN and P300 include the following: (1) Reliably recorded in individual subjects (with fewer trials needed), (2) good intrasubject test–retest reliability (Tremblay et al., 2003b), (3) the use of a passive recording paradigm, and (4) evokes distinct waveform morphologies that reflect some of the acoustic content of speech sounds. It can therefore be used to examine the neural detection of important acoustic aspects of speech, like speech envelope, even when presented through a hearing aid (Tremblay et al., 2006a) or cochlear implant (CI) (Friesen and Tremblay, 2006; Friesen et al., 2009; Kim et al., 2009). Friesen et al. (2009), for example, used P1-N1-P2 change responses to show how the number of CI channels affected the neural detection and perception of vocoded CVC stimuli. The amplitude and latency of P1-N1-P2 responses were modulated by the amount of auditory information provided by increasing channel numbers. For example, neural conduction time (latency) decreased as the number of spectral channels increased. Perception of the CVC stimuli also improved with increasing the number of spectral channels; however, coinciding changes in P1-N1-P2 morphology did not fully predict changes in

perception. Even the ACC has its limits because it best reflects time-varying cues, like speech envelope, and less about the spectral content contained in a signal.

Mismatch Negativity

The MMN involves a recording paradigm that is much different than the P1-N1-P2 change complex. To elicit the MMN, stimuli must be presented via an oddball paradigm. The number of deviant stimuli, the percent ratios, and types of stimulus contrasts vary across studies. One example of a sound contrast used to elicit the MMN is tones of different frequencies, where tones of 1,000 and 990 Hz may serve as standard and deviant. Different speech sounds, sound durations, and sounds differing in spatial location can also be used to elicit an MMN. Whatever the stimulus might be, the order of stimulus presentation (standard versus deviant) is pseudo-randomized, and the amplitude of the MMN is greater when deviant stimuli are not presented consecutively. A reason for this has to do with insufficient buildup of auditory memory trace required by repeated presentations of the standard stimulus.

The MMN is seen as a negativity following N1 in the latency window of approximately 100 to 300 ms (Naatanen et al., 1978). It does not appear in Figure 18.1 because the MMN waveform is obtained by subtracting the waveform for the deviant stimulus from the waveform to the standard stimulus. This oddball paradigm involves presenting a standard sound the majority of the time (e.g., 80%) and presenting a deviant sound the rest of the time (e.g., 20%). In this example, the 1,000-Hz tone, the standard, would be presented 80% of the time, whereas the remaining 20% of presentations would consist of a 990-Hz tone, the deviant. If deviant stimuli are presented in close proximity (e.g., one to two intervening standard stimuli) to each other, then the response is less robust than when more intervening standard stimuli are presented between deviant sounds. Figure 18.7 illustrates the basic concept of MMN recording.

MMNs are best recorded from individuals who are awake and alert, although they do not have to actively pay attention to the stimulus. Other recording approaches that include multiple deviants as well as reversed order control conditions can be used as well. Although the response is optimally seen at electrode site Fz, the scalp topography and distribution of the MMN are also informative. For example, MMN and P1-N1-P2 responses observed over frontal electrode locations will reverse in polarity when viewed over temporal electrode sites, because electrodes are now on the opposite field of neural generators arising from superior temporal gyri. Seeing the polarity reversal assures the clinician that what was seen at frontal recording sites is in fact neural rather than eye blink or other possible confounding artifacts. For a detailed review of the MMN and the types of stimuli that are capable of eliciting the MMN, the reader is referred to Picton et al. (2000).

One of the initial reasons why scientists and clinicians first became interested in the MMN response is related to the potential application for assessing children with auditory-based learning problems and who are diagnosed as having SLI or central auditory processing disorders (CAPD) (Kraus et al., 1996). When people with communication disorders are assessed using a behavioral task, poor performance might relate to the demands related to test administration and not the content of the test (an inability to sit, attend, follow verbal or written instructions). No overt task is required to acquire the MMN, and the patient does not have to pay attention to the stimuli, so it was thought that the MMN would evolve into an assessment tool that

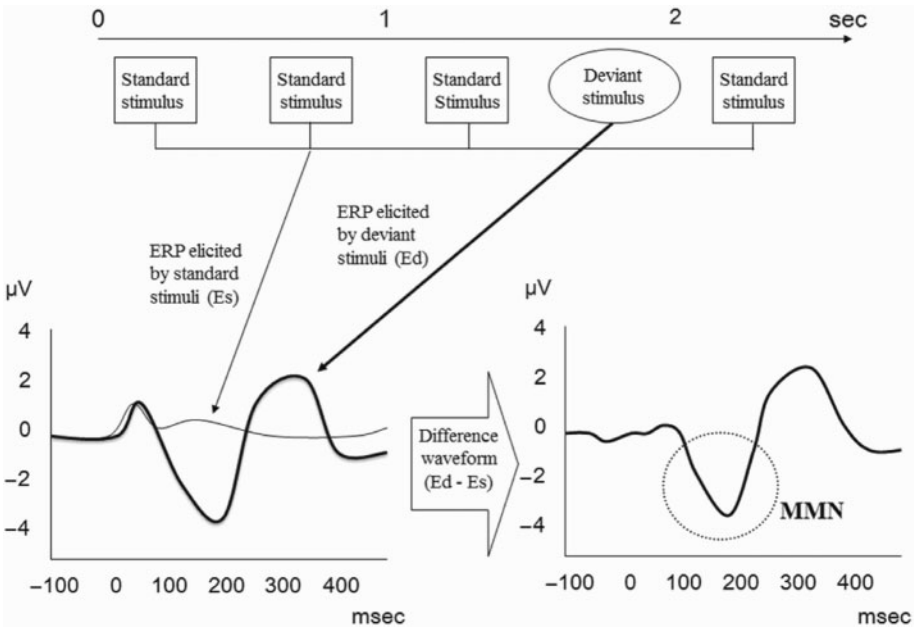


FIGURE 18.7 A visual illustration of the oddball stimulus condition used to evoke the MMN. [Reprinted with permission from Nagai T, Tada M, Kirihara K, Araki T, Jinde S, Kasai K. [2013] Mismatch negativity as a "translatable" brain marker toward early intervention for psychosis: a review. *Front Psychiatry.* 4, 115.]

could be used to objectively identify children with abnormal sound discrimination problems such as SLI and CAPD. The presence and magnitude of the MMN show reasonable agreement with behavioral discrimination performance, thereby making it a sensitive tool to assess the neurophysiological capacity of an individual's auditory discrimination abilities. However, a major obstacle in adopting the MMN as a clinical tool is the difficulty associated with quantifying it in individual people, thereby limiting its ability to diagnose individual patients. Although efforts to resolve this issue are underway, another important consideration is the sensitivity and specificity of the MMN. The MMN is sensitive to abnormal discriminative and memory processes related to audition; however, it does not appear to be specific to a particular disorder. Abnormal MMNs are being reported in a wide range of populations diagnosed with schizophrenia, personality disorders, and alcoholism (Marco-Pallares et al., 2007). Thus, it is difficult at this time to conclude how the clinical significance of an abnormal MMN would add to the audiologic assessment and rehabilitation of people with hearing loss.

Additional obstacles include the amount of time needed to collect the MMN response and the challenge of defining the MMN in individuals. A large number of trials are needed to achieve a sufficient SNR in which to view the MMN. This makes it more time consuming and less feasible for clinical application. The oddball paradigm involves a trade-off between how novel a sound is and how many sweeps are recorded to that deviant stimulus. A lower percentage of deviant stimuli are associated with a higher degree of novelty, but that lower percentage will result in a smaller number of sweeps recorded to the deviant, resulting in a less favorable SNR. Approximately 200 accepted sweeps to the deviant are necessary to obtain a favorable SNR, and even then it can be difficult to visualize the MMN in an individual subject. Subjective visual detection is often used to determine the presence or absence of an MMN; however, various statistical detection algorithms do exist (e.g., point-by-point *t*-tests and the bootstrapped, integrated MMN; Duncan et al., 2009; Picton et al., 2000). With that said, MMNs are not always detectable in recordings from a single individual, even when that individual can behaviorally discriminate the standard and deviant sounds. Collectively, because MMNs are not always present in individual data, and because of the SNR problems, MMN studies often need to use average waveforms from all individuals within a group, or group average waveforms, to optimize the detection of MMNs.

P300

The P300, or P3, is another CAEP that can reflect physiological processing of discrimination. It was first reported over 40 years ago (Sutton et al., 1965). The P3 is generated within auditory and nonauditory areas of the brain (e.g., frontal cortex) and is seen as a positive peak at approximately 200 to 300 ms in response to novel stimuli presented in an oddball paradigm (Figure 18.8). The reader is referred to Naatanen (1992) and Polich and Criado (2006) for a thorough review. Recording the P3 is similar to the approach used to record the MMN (see Table 18.3). However, the subject often attends to the deviant stimuli instead of ignoring them as in the "passive" MMN paradigm. Tasks for the subjects may include counting the number of deviant stimuli or pressing a button each time they perceive the deviant sound. Similar to the MMN, a wide variety of stimulus contrasts can be used to elicit the P3 such as differences in frequency, timing, or speech sounds.

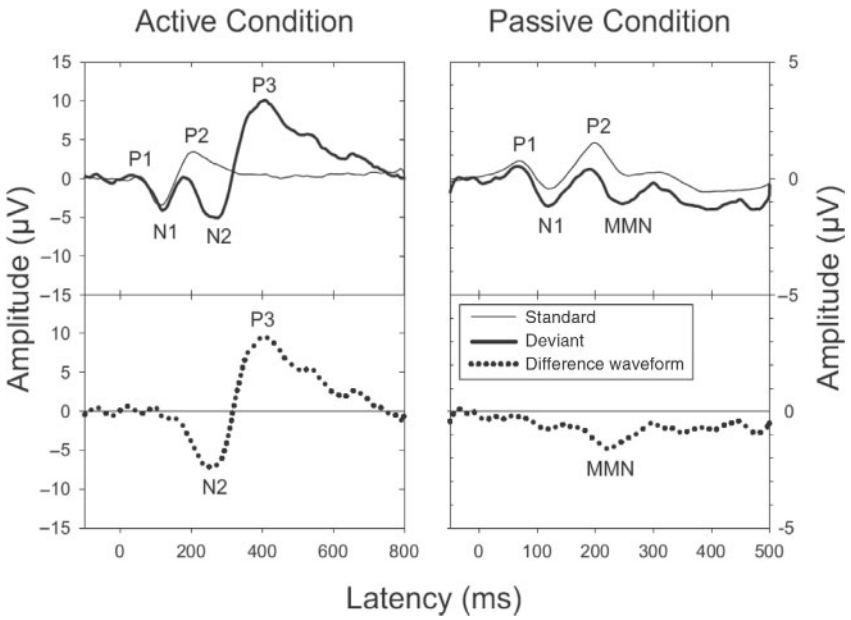


FIGURE 18.8 Illustration of how P300 and MMN are recorded in active and passive conditions, respectively. *Upper row of panels* shows how the P300 is present in the active condition [**left panel**] when an individual attends to the deviant stimulus; in the **right panel,** an enhanced negativity is seen when an individual ignores the stimuli. *Bottom row of panels* illustrates the difference waveforms [standard wave subtracted from deviant wave] where the enhanced positivity for the P300 [**left panel**] and enhanced negativity from the MMN [**right panel**] are seen. [Reprinted with permission from Martin BA, Tremblay KL, Korczak P. [2008] Speech evoked potentials: from the laboratory to the clinic. *Ear Hear.* 29 [3], 285–313.]

In general, it can be said that the morphology of the P3 changes as a function of stimulus discrimination ability. P3 amplitudes are larger for easy to discriminate stimuli and amplitudes decrease when the discrimination task becomes more difficult. The same can be said for latency. P3 latency decreases for easy discrimination tasks and increases for difficult tasks (Fitzgerald and Picton, 1983). P300 amplitude (μV) is defined as the difference between the mean prestimulus baseline voltage and the largest positive-going peak of the ERP waveform within a time window (e.g., 250 to 500 ms). Latency (ms) is defined as the time from stimulus onset to the point of maximum positive amplitude within a time window. P300 scalp distribution is defined as the amplitude change over the midline electrodes (Fz, Cz, Pz), which typically increases in magnitude from the frontal to parietal electrode sites and is sometimes referred to as a P300b when recorded over Pz in response to difficult stimulus contrasts. When the stimulus contrast is easy, a "P300a" can be seen even in the absence of a task (Squires et al., 1975).

A multitude of nonpathologic and pathologic subject factors influence the P300. For example, pathologic factors of dementia, depression, and dyslexia affect the P300, as do nonpathologic factors of fatigue, handedness, and personality (Polich, 2004). Considering the myriad of factors that can affect the P300, like the MMN, the clinical utility of this response, for audiologic purposes, is often debated. One of the arguments against using the P300 in the audiology clinic has to do with the active task used to evoke it. What additional information does that P3 offer to the clinician if a person is able to provide reliable behavioral responses, providing evidence of what that person can and cannot discriminate auditorily? For research purposes, however, it can be argued that it is important to have the brain active to define the neural mechanisms that are contributing to the perceptual event of interest. And in turn, the information that is obtained might translate into new technology or information that is relevant to the assessment and rehabilitation of people with communication disorders.

N400/P600

Other cortical CAEPs exist for the purpose of defining normal and disordered auditory processing, but they are not readily accessible to audiologists for many of the same reasons as the MMN. CAEP peaks appearing as late as 1,400 ms, for example, appear in the audiology literature as a means of assessing word comprehension (Mehta et al., 2009). Two additional examples are the N400 and P600 that are used in research laboratories and are related to linguistic processing. The N400 is a negative response occurring at approximately 400 ms and reflects language processing related to semantic incongruity (Kutas and Hillyard, 1980). Stimuli for the N400 are sentences with incongruous semantics, such as "the box is walking." The P600, on the other hand, is a positivity that occurs at approximately 600 ms and is elicited by violations of a language's rules, such as syntax. Figure 18.9 shows how

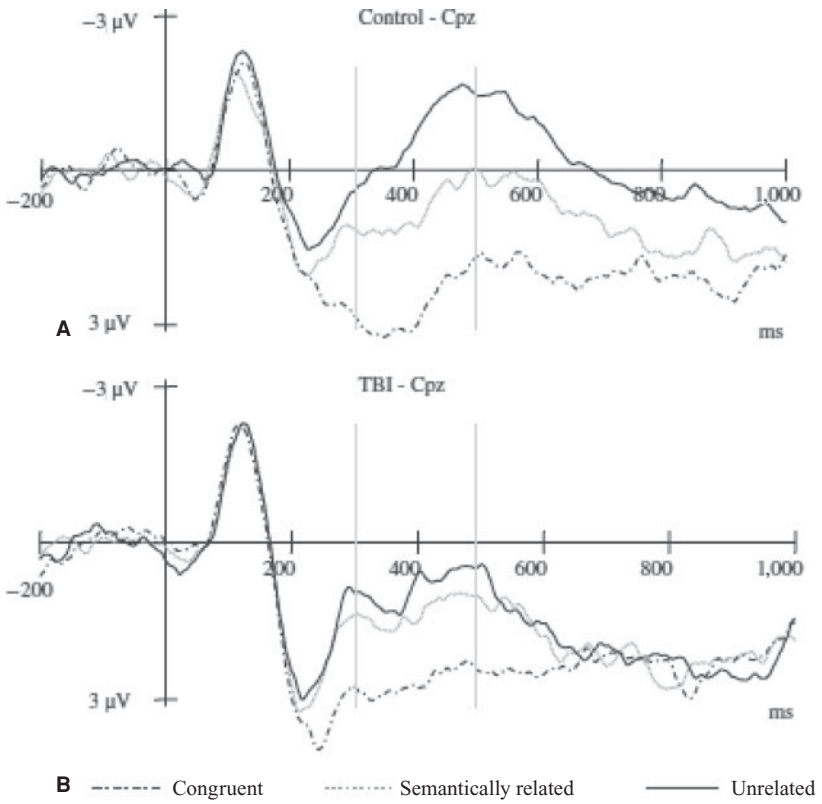


FIGURE 18.9 Event-related potentials elicited in response to an auditory target work at electrode sites Cpz within the –200 to 1,000 time window. **A:** The control group shows a clear distinction between all three conditions, commencing around 250-ms post-target stimulus. Negative polarity is plotted up. **B:** the TBI group shows a reduction in the N400 (shown between *two vertical lines* at 300 to 500 ms) compared to the control group between the three conditions. [Reprinted with permission from Knuepffer C, Murdoch BE, Lloyd D, Lewis FM, Hinchliffe FJ. (2012) Reduced N400 semantic priming effects in adult survivors of paediatric and adolescent traumatic brain injury. *Brain Lang.* 123 (1), 52–63.]

the N400 was used to examine the neural correlates of linguistic processing deficits reported following pediatric and adolescent traumatic brain injury (TBI). Participants in the TBI group had a history of pediatric or adolescent TBI, whereas participants in the control group never reported an insult to the brain. Three stimulus conditions were used to elicit the N400. The auditory target word was either preceded by a prime picture that was congruent, that is, the picture of an apple followed by a read-out of the word "apple," incongruent, but from the same semantic category, that is, the picture of an orange followed by the word "apple," or semantically and associatively unrelated, that is, the picture of a shoe followed by the word "apple." Results revealed a significantly smaller N400 response in the unrelated condition in the TBI group when compared to the control group. In other words, the N400 revealed physiological differences in the way TBI patients process linguistic information, with the TBI group being less able to detect linguistic violations involving unrelated words.

PATIENT SUBJECT FACTORS–MATURATION AND AGING

Whereas the brainstem is said to be neuroanatomically mature at birth, the maturation of commissural axons, and association fibers, that provide connections within and between hemispheres, are still maturing by the age of 12 years (Moore, 2002). From adulthood through the later stages in life, many physical, biologic, chemical, and psychologic changes take place. Together, they all impact the magnitude and distribution of synaptic activity, as well as the neural generators involved. Optimal CAEP recording paradigms therefore vary, depending on the age of the participant. CAEP latencies and amplitudes, and their distribution patterns across the scalp, are also influenced by the type of stimulus presentation paradigm (block or oddball), the degree of acoustic deviation when using an oddball paradigm, sleep–awake state, the length of the interstimulus interval (ISI) (e.g., stimulus presentation rate) (see Kushnerenko et al., 2013 for a review of infant and child recordings).

As an example, the morphology of the P1-N1-P2 responses changes across the lifespan. In adults, the amplitude of P1 is small and N1 and P2 often dominate the response. In contrast, for young children, P1 dominates the response and is followed by a slow negativity (N2) (Ponton et al., 1996). However, if the stimulus presentation rate is slowed down, P1-, N1-, and P2-like responses become visible in infants and children (Wunderlich and Cone-Wesson, 2006). Later in life, N1 and P2 components become delayed in middle and old age (Tremblay and Burkard, 2007) but can also appear youthlike when slower ISIs are used. The fact that CAEPs are affected by the amount of time that separates one stimulus from another hints at changes in temporal processing abilities that take place as a function of age (see Billings et al., 2012a). Moreover, the presence of age-related hearing loss compounds the effect by affecting both latency and amplitude of N1 and P2 peak waveforms (Martin et al., 2008; Tremblay et al., 2003a).

Maturational effects are seen for the MMN and N400 as well because neural mechanisms, and their spatial distribution, change with age (Kushnerenko et al., 2013; Martin et al., 2003). Like the P1-N1-P2, ISI greatly influences the morphology of the MMN. From birth through old age, the rate at which stimuli are presented appears to modulate the physiological index of auditory sensory memory and discrimination. It is therefore not surprising that some disorders, such as Alzheimer's (AD) and Parkinson's diseases (PD), have been linked to abnormal processing speed as reflected by the MMN.

CLINICAL APPLICATIONS: OPPORTUNITIES AND CHALLENGES

Hearing Loss and Hearing Aids

The ABR is typically the audiologist's tool of choice for estimating audiometric thresholds; however, the P1-N1-P2 is also a useful tool for this purpose. An advantage of using the P1-N1-P2 response is that the magnitude of the evoked response pattern is larger than the ABR (improved SNR); so fewer sweeps are necessary, resulting in a shorter test time than the ABR. An additional advantage of the P1-N1-P2 response for threshold estimations is that it indirectly provides an indication that sound reached the auditory cortex. The presence of any CAEP, however, does not imply that auditory pathways and cortical function are entirely normal because CAEPs only measure a small fraction of overall auditory function. Also, keep in mind that the P1-N1-P2 response is affected by maturation and sleep, and so it is best applied to adults who are awake. If it is used to estimate hearing thresholds in infants or young children, sleep state should be monitored and the child's age should be considered when interpreting waveform morphology (see Stapells, 2009). Unlike ABRs, there are no published norms for clinicians to use for P1-N1-P2 responses because the peak latencies and amplitude vary depending on the stimuli used to evoke it (e.g., rise time, duration, speech, tones).

P1-N1-P2 threshold estimates can be obtained in normal-hearing individuals as well as individuals with hearing loss, and CAEP thresholds typically fall within 10 dB of behavioral thresholds (Van Maanen and Stapells, 2005). Polen (1984), for example, used speech sounds to evoke CAEPs and reported a prolongation of N1, P2, N2, and P3 latencies, as well as a reduction in P2 and N2 amplitudes, in people with moderate to severe sensory/neural hearing loss. Readers are referred to Martin et al. (2008) for a complete review, but an overarching conclusion is that decreased

sound energy (attenuation because of middle ear or cochlear pathology), entering the CAS, results in increased CAEP peak latencies and decreased amplitudes. The effects of decreased audibility on the P1-N1-P2 complex can be seen in Figure 18.2. The MMN, P300, and N400 are not shown but they are similarly affected. For example, Kraus and colleagues used the MMN to test two listeners with sensory/neural hearing loss using a /da/-/ga/ oddball paradigm. Whereas the subject who could easily behaviorally discriminate the /da/-/ga/ contrast evoked a clear MMN, the subject who could not easily discriminate the two sounds had no MMN (Kraus et al., 1995). Once again, extending this direction of work into a clinic protocol has been problematic because of the difficulties recording MMNs in individual patients as well as insufficient data pertaining to sensitivity and specificity of the MMN.

There is also interest in using CAEPs (P1-N1-P2 and ACC) to quantify the effects of hearing aid amplification on the brain. As described by Billings (2013), there are different approaches to recording aided CAEPs; the most common is to have an individual wearing the hearing aid while stimuli are presented in sound field. Another approach is to record the hearing aid output, either in a coupler or a mannequin (e.g., Knowles Electronics Manikin for Acoustic Research (KEMAR)) or the Brüel and Kjær Head and Torso, and presenting the sounds through earphones to the participant. Stimuli can also be presented through a hearing aid, using direct audio input. Finally, hearing aid processing can be simulated using a master hearing aid approach, using signal-processing software, and then presenting the modified sound through insert earphones to the patient. Whereas the first approach is more familiar to audiologists, is more ecologically similar to everyday listening experiences, and best resembles current clinical practice, the last three methods have the advantage of minimizing extraneous variables related to sound field testing such as the effects of head movement and standing waves. With that said, there are a number of studies showing that CAEPs can be reliably recorded in individuals, with and without hearing loss, in sound field (Tremblay et al., 2006a, 2006b; see also Billings, 2013; Martin et al., 2008 for reviews).

One purpose of using aided CAEPs is to assist in the verification and validation of hearing aid fittings; another is to examine plasticity-related changes in the brain (for a review see Tremblay and Moore, 2012). From the ear to the brain, spectral and temporal acoustic information contained in the signal is represented using place and timing codes to reflect perceptually relevant acoustic speech cues and these codes are impacted by hearing loss, sometimes referred to as "deprivation- or injury-related plasticity." When sound is reintroduced to the auditory system, there is "use-related plasticity"; modifications to sensory maps, synaptic alterations, and neurochemical changes (Irvine et al., 2001). When fit with a hearing aid, additional forms of plasticity are presumed to take place (Willott, 1996). For example, when a hearing aid increases the intensity of a signal, areas of the auditory system that were once deprived of sounds are now being stimulated. Then, the amplified sound that comes out of a hearing aid stimulates new neural networks and people need to learn how to make use of these new modified sounds. The duration of hearing loss, the age of the patient, and the length of amplification are all believed to contribute to the neuroplastic effects.

The idea that brain measures might provide valuable audiologic information related to hearing aid use and plasticity is not new. ABR recordings have been recorded in response to auditory stimuli presented in sound field and amplified by hearing aids. They proved to be unsuccessful because the short-duration signal (click, tone-pip) interacted with the hearing aid circuitry in a way that introduced ringing and other artifacts (Gorga et al., 1987). Anderson and Kraus (2013) revisited this concept by using speech-evoked ABRs (also called the FFR). They showed that it is possible to record FFRs while a person is wearing a hearing aid; however, this direction of research is still new and it will be necessary to explore some of the opportunities and obstacles recently uncovered when using CAEPs.

Rapin and Graziani (1967) were among the first to use CAEPs such as the P1-N1-P2 response to assess the effect of increased audibility related to hearing aid use. They found that the majority (5/8) of 5- to 24-month-old subjects with severe-to-profound sensory/neural hearing loss showed an improved P1 morphology in the aided testing condition, presumably reflecting improved audibility provided by the hearing aid. However, two of the infants showed no change in their cortical CAEP thresholds across the aided and unaided conditions and these types of inconsistencies are still present in the literature almost 50 years later. Glista et al. (2012), for example, reported instances when cortical P1-N1-P2 responses were absent in normal-hearing children.

Although there is converging evidence in the literature to show that CAEPs (P1-N1-P2 responses as well as the ACC) to tones and speech sounds can be reliably recorded in individuals while wearing a hearing aid, and that different speech sounds evoke different aided CAEP patterns (Billings et al., 2007; Tremblay et al., 2006a, 2006b), there is also evidence to show that CAEPs do not reliably reflect hearing aid gain and may be affected by many different signal-processing functions (Billings, 2013; Billings et al., 2012b; Marynewich et al., 2012). To better understand amplification and the brain, and to move the field forward, Tremblay et al. (in press) and Tremblay (2013) edited special issues to explore the relationship between "hearing aids and the brain." These publications summarize the current status of hearing aid verification and validation using evoked potentials and indicate the many opportunities and challenges in doing so. One challenge that is discussed in this special issue is that CAEPs do not reliably reflect hearing aid gain even when different types of hearing aids (analog and digital) and their parameters (e.g., gain, frequency response)

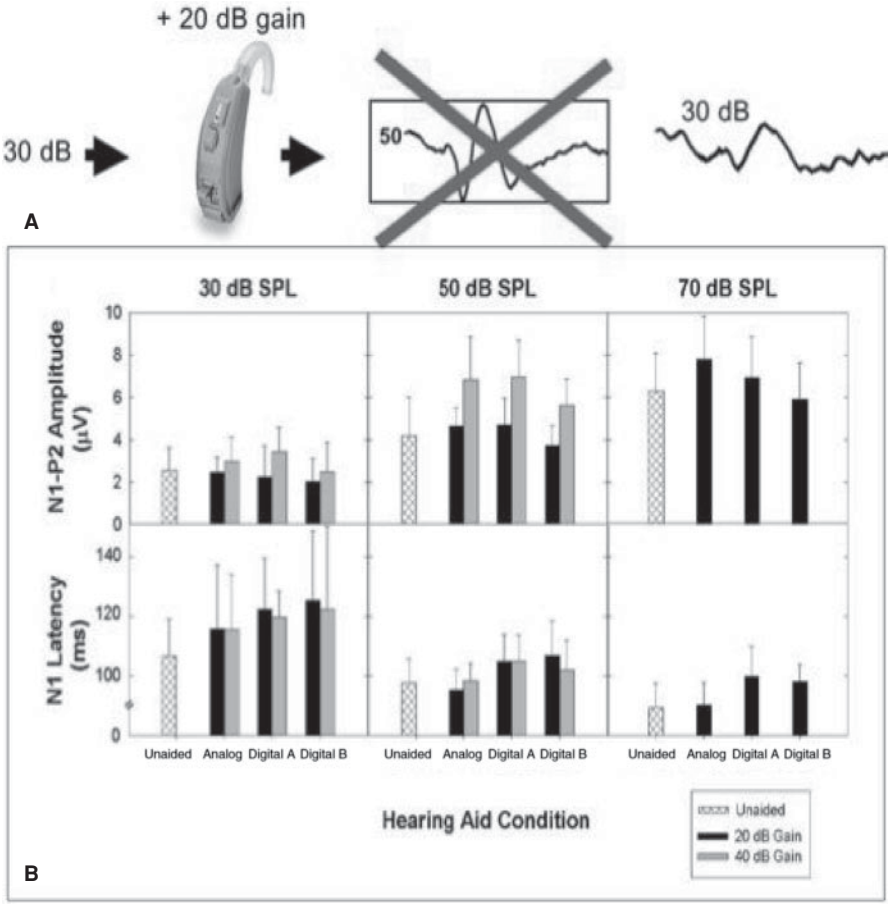


FIGURE 18.10 P1-N1-P2 peaks are earlier in latency and larger in amplitude when evoked by a 50-dB SPL signal compared to a 30-dB SPL signal (**A**). But when a 30-dB signal is processed through a hearing aid, and 20 dB of gain is provided, the resultant waveform does not reflect the increase in signal level (**B**). Despite the fact that a 50-dB signal is being transmitted to the cochlea, the aided waveform still appears similar in morphology to the response elicited by the 30-dB signal. [Reprinted with permission from Marynewich S, Jenstad LM, Stapells DR. [2012]. Slow cortical potentials and amplification-part I: n1-p2 measures. *International journal of otolaryngology*. 2012, 921513. doi:10.1155/2012/921513.]

are manipulated (Billings et al., 2007, 2011, 2012b; Jenstad et al., 2012; Marynewich et al., 2012). Figure 18.10A shows how 30 dB of sound input, amplified by 20 dB of gain, does not result in a CAEP generated by a 50-dB signal. Instead, the resultant waveform is similar to that generated by a 30-dB input signal. Marynewich et al. (2012) reported similar findings that are summarized in Figure 18.10B. N1 latency and N1-P2 amplitudes were not significantly affected when a hearing aid provided 20 or 40 dB of gain. One of the reasons for not seeing these gain effects has been attributed to the SNR of the signal coming out of the hearing aid. Billings et al. (2012b) demonstrated that the signal that comes out of the hearing aid is in fact amplified, but so is the noise level. Therefore, the SNR entering the hearing aid is not any different than the SNR being delivered to the brain. CAEPs, such as the P1-N1-P2, are sensitive to SNR, in a way that obscures the effectiveness of hearing aid gain.

The example of SNR is discussed here but there are many other signal-processing issues (channel-specific compression time constants, noise reduction algorithms, and adaptive directionality) that can impact P1-N1-P2 (and ACC) and subcortical responses because they are driven by the acoustic content of the signal. So, whereas the absence of change (in peak latencies or amplitudes) across time could be interpreted to mean a lack of neuroplasticity related to sound transmission from the ear to the brain, an alternative explanation could be that the pattern of evoked neural activity reflects the individual signal-processing strategies of the device (e.g., changes in SNR) rather than brain plasticity. It would also be possible to appropriately fit a hearing aid (for degree and configuration of the hearing loss) in a client who showed no evoked brain response or no improvement from the unaided cortical response. Therefore, earlier studies championing the use of CAEPs as a clinical tool for assessing hearing aid functioning (Martin et al., 2008) have been tempered somewhat by more recent studies showing some of the challenges and limitations still needing to be addressed. Despite these concerns, commercial equipment is being marketed to clinicians for the purpose of measuring amplified CAEPs (Munro et al., 2012). If it becomes possible to resolve some of these issues, and take advantage of the opportunities amplified brain measures could provide, it will then be up to the clinicians to determine whether or not aided CAEPs are helpful enough to justify the additional testing time and expense of including it as an additional clinical procedure.

CAEPs and Cochlear Implants

CAEPs can also be recorded from CI users. CAEPs have been used to track developmental changes (maturation) following

cochlear implantation as well as to assist in the programming of CIs (Brown et al., 2008). Ponton et al. (1996), for example, used the P1-N1-P2 complex to define the normal maturation of auditory pathways in children with and without hearing loss. More specifically, he tracked maturational changes associated with the P1 component (because of the age group) and showed neuroplastic changes (inferred from decreased neural conduction times (latency) and increased amplitudes) following the reintroduction of sound using a CI. These findings are important because the central auditory system was thought to be hard-wired at one time, resistant to change following periods of deprivation. Ponton et al.'s results also provide further physiological evidence to reinforce the importance of early identification of hearing loss so that intervention can be achieved as early as possible, so as to capitalize on the plasticity of a developing central auditory system.

The issues described here pertaining to hearing aids also apply when sound is delivered in sound field and processed by a CI. Like hearing aids, CIs have a microphone and automatic gain controls. The signal goes through a speech processor and is then directed to select locations (channels) along an electrode array that affect place-pitch mapping as well as current intensity. SNR is not as problematic in CIs compared to hearing aids, because the level of noise is not audible to the listener. However, CAEP peak latency and amplitude values are affected by the number of channels that are active (Friesen et al., 2009), the electrode location being stimulated (Brown et al., 2008), as well as the stimulus type, level, and rate (Davids et al., 2008; Firszt et al., 2002). CAEP morphology can therefore vary widely from one CI user to the next, solely based on signal processor settings. Moreover, CI speech processors are inherently nonlinear; increases in the level of an acoustic signal presented in the sound field may or may not translate into a change in the amount of current provided by the CI. These issues pose a problem when cross-sectional designs are used to track changes over time because CAEPs are averaged across individuals who are wearing different devices. Because each individual is wearing an implant that is programmed differently, these implant settings are what will drive CAEP latencies and amplitudes. Therefore, when auditory stimuli are presented in sound field, and CI controls affecting stimulus level and location are not held constant, it is possible that CAEP neural response patterns reflect device rather than experience-related changes in brain activity (Sharma et al., 2002).

The Ponton et al.'s (1996) stimuli were delivered directly to the electrode array, bypassing the implant processor. Thus, implant settings likely did not confound the latency and amplitude of the P1 changes observed following cochlear implantation. This approach also helps to minimize the amount of implant artifact that can contaminate EEG recordings. However, understanding the functional significance of P1 changes in relation to speech discrimination and language development is an area that is still being explored. Although Sharma and Dorman (2006) report a correlation between CAEP latency and speech and language skills, there is also evidence that P1 onset response, evoked by a brief synthetic stimulus, is not specifically related to sound discrimination. For example, P1 latencies are abnormal in alcoholics who do not have speech and language problems (Campanella et al., 2009). P1 latencies are normal in older adults who do have speech perception difficulties (Tremblay, 2005; Tremblay et al., 2003a). When people improve their perceptual skills through auditory training, there is no change in P1 latency with improved speech perception. Also, when compared to the ACC or P1-N1-P2 change response, the P1 component (signaling the onset of sound) does not correspond to behavioral discrimination thresholds; it is the change response (see Figure 18.8 as well as Won et al., 2011). As shown in Figure 18.9, P1 latencies to sound onset do not appear to be different across groups or conditions even though the TBI group showed difficulty with linguistic processing. Thus, it appears that the change responses and later discriminative CAEP responses could provide much information to the clinician if they proved to be sensitive to communication problems, specific to known disorders, and feasible to execute in the clinic.

Because CAEPs are modulated by HA and CI settings, this means CAEPs can also be used to examine the effects of different signal processing on evoked neural activity. And of all the discriminative CAEPs, the ACC might be the one that proves to be most helpful to clinicians when programming CIs. Brown et al. (2008) demonstrated that it is possible to record P1-N1-P2 change responses in CI patients by bypassing the speech processor and directly stimulating different electrode positions. In doing so, the authors demonstrated the feasibility of using the ACC in response to changes in stimulating electrode position so that the effect of different stimulation patterns on perception can be studied. He et al. (2013) showed that the ACC can be used to evaluate electrode-discrimination capacities in children who wear CIs and can serve as an objective tool for evaluating spectral-pattern detection in such subjects as well as their potential speech perception performance.

To summarize, the basic principles of EEG/MEG do not apply when stimuli are delivered through a hearing prosthesis (hearing aids and CIs). Hearing aids and CIs introduce artifact and alter the acoustic/electric properties of the incoming stimulus into the ongoing EEG/MEG, making it difficult (but not impossible) to separate biology from technology. It is therefore necessary to understand the interaction between the signal processing introduced by the hearing aid or CI and the CAEPs before clinical protocols can be considered.

CAEPs and Auditory Training

Thus far CAEPs have been described as having been used to assess the neural detection and discrimination of sound.

However, CAEPs have also been used to characterize how a person makes use of sound through various types of interactions, including auditory training exercises, music training, and other forms of auditory learning. For example, the P1-N1-P2 response has been used in human speech sound training experiments and has shown that neural response patterns change quite rapidly (Ben-David et al., 2011; Tremblay et al., 1998), precede changes in perception, and are retained for months (Tremblay et al., 2010). These points are important to the rehabilitation of people with hearing loss because they provide information about brain–behavior relationships, as well as their time course. One of the motivations underlying this area of research is the hope that EEG recordings might someday assist clinicians with aural rehabilitation. If changes in neurophysiology of a hearing/language impaired person occur following a series of auditory training sessions, this would imply that the brain's representation of sound is changing neurally and would reinforce the use of the current (re)habilitation strategy. However, a change in neurophysiology without a change in behavior may suggest that the intervention method being used is successfully altering the brain's ability to code the sounds, but that behavioral changes may be lagging in time or impaired because of other intervening issues such as cognition and motivation. In contrast, if there is no evidence of physiological discriminability, the audiologist could readjust device settings, or training approach, so as to improve the neural detection, discrimination, and use of the incoming signal.

ABRs (see Chapter 13) have also been used to examine the effects of different forms of training (music, commercially available clinical training programs, etc.) on brain–behavior relationships. The connection between brainstem and cortical processing, and how they work together, is nicely summarized by Shahin (2011), who also goes on to explain how learning to play a musical instrument can modify neural circuitry in a way that improves one's ability to perceive speech more clearly in noisy environments. Importantly, auditory training exercises have been shown to partially reverse age-related declines in neural temporal precision that have been described in this chapter, as well as other publications (Anderson et al., 2013).

SUMMARY

A future goal for clinician scientists is to find ways of quantifying brain activity in a way that can better quantify the real-world processing events that are involved in perception and communication. An additional challenge is to define brain–behavior relationships in a way that is sensitive and specific to communication disorders, using methodologic approaches that can be integrated into everyday practice. Here we reviewed how CAEPs are being used to achieve this goal. Although there are many types of neuroimaging tools (MEG, fMRI, PET), the feasibility and affordability of most preclude their use in routine clinical situations. EEG, however, is already in place in most audiology clinics and is within the audiologists' scope of practice.

Of all the CAEPs described here, perhaps the most clinic-ready CAEP is the P1-N1-P2 complex and its use as a change response. The presence of the P1-N1-P2 complex indicates the detection of speech at the level of auditory cortex. The presence of ACC indicates that the auditory cortex can discriminate the acoustic change contained in a speech signal, and therefore provides information about speech discrimination capacity. Together, these two measurements provide tremendous insight into the capacity of the patient to access and make use of sound. If that sound happens to be amplified, or delivered by a CI, then it will be important to understand how the prosthesis is contributing to the observed CAEPs.

FOOD FOR THOUGHT

1. Which of the discriminatory cortical AEPs has the most clinical utility, being robust when recorded from individuals?
2. What is meant by the following sentence "the basic principles of CAEPs do not apply when stimuli are delivered through a hearing prosthesis or cochlear implant?"
3. Do you see the need for CAEPs being used in the clinic to assist with hearing aid and CI fittings?

KEY REFERENCES

thePoint A full list of references for this chapter can be found at http://thePoint.lww.com. Below are the key references for this chapter.

Bear M, Connors B, Pardiso M. (2007) *Neuroscience: Exploring the Brain.* 3rd ed. Philadelphia, PA: Lippincott Williams and Wilkins.

Billings C. (2013) Uses and limitations of electrophysiology with hearing aids. *Semin Hear.* 34 (4), 257–269.

Billings C, Tremblay K, Willott J. (2012a) The aging auditory system. In: Tremblay K, Burkard RF, eds. *Translational Perspectives in Auditory Neuroscience: Hearing Across the Lifespan – Assessment and Disorders.* San Diego, CA: Plural Publishing; pp 4357–4362.

Billings CJ, Papesh MA, Penman TM, Baltzell LS, Gallun FJ. (2012b) Clinical use of aided cortical auditory evoked potentials as a measure of physiological detection or physiological discrimination. *Int J Otolaryngol.* 2012, 365752.

Billings CJ, Tremblay KL, Miller CW. (2011) Aided cortical auditory evoked potentials in response to changes in hearing aid gain. *Int J Audiol.* 50 (7), 459–467.

Billings CJ, Tremblay KL, Souza PE, Binns MA. (2007) Effects of hearing aid amplification and stimulus intensity on cortical auditory evoked potentials. *Audiol Neurootol.* 12 (4), 234–246.

Brown CJ, Etler C, He S, O'Brien S, Erenberg S, Kim JR, et al. (2008) The electrically evoked auditory change complex: preliminary results from nucleus cochlear implant users. *Ear Hear.* 29 (5), 704–717.

Eggermont JJ. (2001) Between sound and perception: reviewing the search for a neural code. *Hear Res.* 157, 1–42.

Firszt JB, Chambers RD, Kraus N Reeder RM. (2002) Neurophysiology of cochlear implant users I: effects of stimulus current level and electrode site on the electrical ABR, MLR, and N1-P2 response. *Ear Hear.* 23 (6), 502–515.

Friesen L, Tremblay K. (2003) Electrophysiologic measures of speech, language and hearing. *Perspect Neurophysiol Neurogenic Speech Lang Disord.* 13 (1), 3–10.

Friesen LM, Tremblay KL. (2006) Acoustic change complexes recorded in adult cochlear implant listeners. *Ear Hear.* 27 (6), 678–685.

Friesen LM, Tremblay KL, Rohila N, Wright RA, Shannon RV, Baskent D, et al. (2009) Evoked cortical activity and speech recognition as a function of the number of simulated cochlear implant channels. *Clin Neurophysiol.* 120 (4), 776–782.

Glista D, Easwar V, Purcell DW, Scollie S. (2012) A pilot study on cortical auditory evoked potentials in children: aided CAEPs reflect improved high-frequency audibility with frequency compression hearing aid technology. *Int J Otolaryngol.* 2012, 982894.

Harris KC, Mills JH, He N, Dubno JR. (2008) Age-related differences in sensitivity to small changes in frequency assessed with cortical evoked potentials. *Hear Res.* 243, 47–56.

He S, Grose JH, Teagle HF, Buchman CA. (2013) Objective measures of electrode discrimination with electrically evoked auditory change complex and speech-perception abilities in children with auditory neuropathy spectrum disorder. *Ear Hear.* [Epub ahead of print].

Hyde M. (1997) The N1 response and its applications. *Audiol Neurootol.* 2 (5), 281–307.

Knuepffer C, Murdoch BE, Lloyd D, Lewis FM, Hinchliffe FJ. (2012) Reduced N400 semantic priming effects in adult survivors of paediatric and adolescent traumatic brain injury. *Brain Lang.* 123 (1), 52–63.

Kraus N, McGee TJ, Carrell TD, Zecker SG, Nicol TG, Koch DB. (1996) Auditory neurophysiologic responses and discrimination deficits in children with learning problems. *Science.* 273 (5277), 971–973.

Malmivuo J, Suihko V, Eskola H. (1997) Sensitivity distributions of EEG and MEG measurements. *IEEE Trans Biomed Eng.* 44 (3), 196–208.

Martin BA, Shafer VL, Morr ML, Kreuzer JA, Kurtzberg D. (2003) Maturation of mismatch negativity: a scalp current density analysis. *Ear Hear.* 24 (6), 463–471.

Martin BA, Tremblay KL, Korczak P. (2008) Speech evoked potentials: from the laboratory to the clinic. *Ear Hear.* 29 (3), 285–313.

Naatanen R. (1992) *Attention and Brain Function.* Hillsdale, NJ: Lawrence Erlbaum Associates.

Naatanen R, Gaillard AW, Mantysalo S. (1978) Early selective-attention effect on evoked potential reinterpreted. *Acta Psychol (Amst).* 42 (4), 313–329.

Naatanen R, Paavilainen P, Rinne T, Alho K. (2007) The mismatch negativity (MMN) in basic research of central auditory processing: a review. *Clin Neurophysiol.* 118 (12), 2544–2590.

Nagai T, Tada M, Kirihara K, Araki T, Jinde S, Kasai K. (2013) Mismatch negativity as a "translatable" brain marker toward early intervention for psychosis: a review. *Front Psychiatry.* 4, 115.

Ostroff JM, Martin BA, Boothroyd A. (1998) Cortical evoked response to acoustic change within a syllable. *Ear Hear.* 19 (4), 290–297.

Picton TW, Alain C, Otten L, Ritter W, Achim A. (2000) Mismatch negativity: different water in the same river. *Audiol Neurootol.* 5 (3–4), 111–139.

Polich J, Criado JR. (2006) Neuropsychology and neuropharmacology of P3 a and P3b. *Int J Psychophysiol.* 60 (2), 172–185.

Ponton CW, Don M, Eggermont JJ, Waring MD, Masuda A. (1996) Maturation of human cortical auditory function: differences between normal-hearing children and children with cochlear implants. *Ear Hear.* 17 (5), 430–437.

Ross B, Fujioka T, Tremblay K, Picton TW. (2007) Aging in binaural hearing begins in mid-life: evidence from cortical auditory-evoked responses to changes in interaural phase. *J Neurosci.* 27 (42), 11172–11178.

Sharma A, Dorman MF, Spahr AJ. (2002) A sensitive period for the development of the central auditory system in children with cochlear implants: implications for age of implantation. *Ear Hear.* 23 (6), 532–539.

Tremblay K. (2005) Beyond the ear: physiological perspectives on auditory rehabilitation. *Semin Hear.* 26 (3), 127–136.

Tremblay K. (2013) Hearing aids: the brain connection. *Semin Hear.* 34 (4), 255–256.

Tremblay K, Billings CJ, Friesen LM, Souza PE. (2006) Neural representation of amplified speech sounds. *Ear Hear.* 27 (2), 93–103.

Tremblay K, Burkard RF. (2007) The aging auditory system: confounding effects of hearing loss on AEPs. In: Burkard RF, Don M, Eggermont JJ, eds. *Auditory Evoked Potentials: Basic Principles and Clinical Application.* Baltimore, MD: Lippincott Williams & Wilkins; pp 403–425.

Tremblay K, Kraus N, McGee T. (1998) The time course of auditory perceptual learning: neurophysical changes during speech-sound training. *Neuroreport.* 9, 3557–3560.

Tremblay K, Piskosz M, Souza PE. (2003a) Effects of age and age-related hearing loss on the neural representation of speech cues. *Clin Neurophysiol.* 114, 1332–1343.

Tremblay K, Scollie S, McMahon CM, Sullivan JR, Abrams H. (in press) Hearing aids and the brain. *Int J Otolaryngol.* Available online at: http://www.hindawi.com/journals/ijoto/si/257084/.

Tremblay KL, Friesen L, Martin BA, Wright R. (2003b) Test-retest reliability of cortical evoked potentials using naturally produced speech sounds. *Ear Hear.* 24 (3), 225–232.

Tremblay KL, Inoue K, McClannahan K, Ross B. (2010) Repeated stimulus exposure alters the way sound is encoded in the human brain. *PLoS One.* 5 (4), e10283.

Tremblay KL, Kalstein L, Billings CJ, Souza PE. (2006b) The neural representation of consonant-vowel transitions in adults who wear hearing AIDS. *Trends Amplif.* 10 (3), 155–162.

Won JH, Clinard CG, Kwon S, Dasika VK, Nie K, Drennan WR, et al. (2011) Relationship between behavioral and physiological spectral-ripple discrimination. *J Assoc Res Otolaryngol.* 12 (3), 375–393.

CHAPTER 19

Otoacoustic Emissions

Beth Prieve and Tracy Fitzgerald

INTRODUCTION

Otoacoustic emissions (OAEs) are an auditory phenomenon of interest to both auditory scientists and clinicians. OAEs are sounds that result from energy generated in the cochlea that are propagated through the middle ear and into the ear canal where they can be measured using a sensitive microphone. OAEs were first described by David Kemp in 1978, and since that time, OAEs have become a standard part of the diagnostic test battery and a screening for hearing loss. The goal of this chapter is to introduce students and clinicians to the theories underlying OAE generation, the types of OAEs, and their measurement and clinical use.

HYPOTHESES OF OAE GENERATION AND THEIR RELATIONSHIP TO AUDITORY FUNCTION

The Traveling Wave and the Cochlear Amplifier

A healthy, living cochlea demonstrates nonlinear behavior and refined frequency specificity at low stimulus levels, similar to the characteristics demonstrated by individual hair cells and auditory nerve fibers (Rhode, 1971). Active biologic mechanisms, often referred to as the "cochlear amplifier," are believed to be responsible for the nonlinear characteristics of cochlear responses, as well as the exceptional sensitivity and frequency selectivity seen in a healthy cochlea as compared to a damaged or dead cochlea. The cochlear amplifier is hypothesized to contribute additional energy that enhances the vibration of the basilar membrane at the peak of the traveling wave, particularly at low stimulus levels (Davis, 1983). Evidence indicates that outer hair cells (OHCs) contribute to this process. Investigators have reported reduced auditory sensitivity, broader tuning, and abnormal response growth when OHCs are damaged or missing (e.g., Dallos and Harris, 1978; Liberman and Dodds, 1984). OAEs measured in the ear canal are thought to be a byproduct of the cochlear amplifier and normal OHC function.

OAEs and Outer Hair Cells

OAEs are a pre-neural phenomenon and can be measured even when the eighth nerve has been severed (Siegel and Kim, 1982). Unlike neural responses, OAEs are unaffected by stimulus rate (Kemp, 1982) and reverse polarity along with the stimulus (Schmiedt and Adams, 1981). In addition, OAEs, particularly those evoked using low stimulus levels, are vulnerable to such agents as acoustic trauma (Schmiedt, 1986), hypoxia (Rebillard and Lavigne-Rebillard, 1992), and ototoxic medications (Brown et al., 1989), which cause hearing loss and damage to OHCs (Dallos and Harris, 1978). OAEs do not appear to be vulnerable to selective loss of inner hair cells (IHCs) (Liberman et al., 1997).

Two hypotheses regarding the OHCs' role in the cochlear amplifier have been explored: Somatic motility of OHCs and nonlinear mechanics of the OHC stereocilia bundle. OHCs demonstrate rapid changes in length in response to electrical stimulation (Ashmore, 1987; Brownell et al., 1985). "Prestin" is the molecular motor responsible for somatic OHC motility (Zheng et al., 2000). Reduced OHC length, absence of OHC motility, and IHC and OHC loss in the basal portion of the cochlea were observed in mice when the prestin gene was deleted. The mutant mice had thresholds elevated by 35 and 60 dB when measured by auditory brainstem responses (ABRs) and OAE thresholds (Liberman et al., 2002).

It seems unlikely that somatic motility of OHCs is the sole source of cochlear amplifier energy and OAEs. OAEs have been measured from species that do not have OHCs (e.g., Manley et al., 1996). OAEs measured in nonmammalian species, whose hair cells are not capable of somatic motility, have been attributed to active hair bundle movements of the hair cell stereocilia (Ricci et al., 2000). Hair cell stereocilia bundles demonstrate frequency selectivity, can provide amplification, and have a force-generating component, all properties needed for the cochlear amplifier. Both OHC somatic motility and stereocilia may contribute to the production of OAEs in mammals, and their contributions may be stimulus-level dependent (Liberman et al., 2004).

OHCs receive the majority of the ear's efferent innervation (Warr et al., 1986), which may act as a regulatory system that allows higher neurologic centers to exert control over cochlear processes such as OHC motility. Direct electrical

stimulation of efferent fibers has been shown to reduce or enhance OAE responses (Siegel and Kim, 1982). Indirect stimulation of the efferent system by means of ipsilateral, contralateral, or binaural sound stimulation has also been shown to alter OAE levels (Berlin et al., 1995; Collet et al., 1990).

Two OAE Generation Mechanisms

Initially, all OAEs were thought to arise from the same mechanism, that is, nonlinear electromechanical distortion within the cochlea resulting, at least in part, from OHC somatic motility (e.g., Kemp and Brown, 1983). The theory that OAEs arise from at least two different mechanisms has changed the way researchers talk about the sources of OAEs and OAE classification (Shera and Guinan, 1999; Talmadge et al., 1999). In early work, Kemp and colleagues studied phase changes as a function of stimulus frequency for different types of OAEs and found two distinct patterns (Kemp, 1986; Kemp and Brown, 1983). Shera and Guinan later attributed the two patterns to different mechanisms: Nonlinear distortion and linear coherent reflection. Nonlinear distortion emissions are attributed directly to the action of OHCs. The source of nonlinear distortion, or "wave-fixed," emissions is believed to follow the traveling wave envelope of the stimulus (e.g., Shera and Guinan, 1999). Therefore, because the shape of the traveling wave does not change significantly as the stimulus is swept in frequency, the phase at any point moving with the traveling wave envelope will not change significantly, as schematized in the left panel of Figure 19.1. Thus, nonlinear distortion emissions are characterized by gradual phase changes as the stimulus frequencies are increased.

Reflection, or "place-fixed," emissions are characterized by phase that rotates rapidly with changes in stimulus frequency, shown by a schematic diagram in the right panel of Figure 19.1. These emissions are proposed to be the result of the incoming traveling waves scattering off of

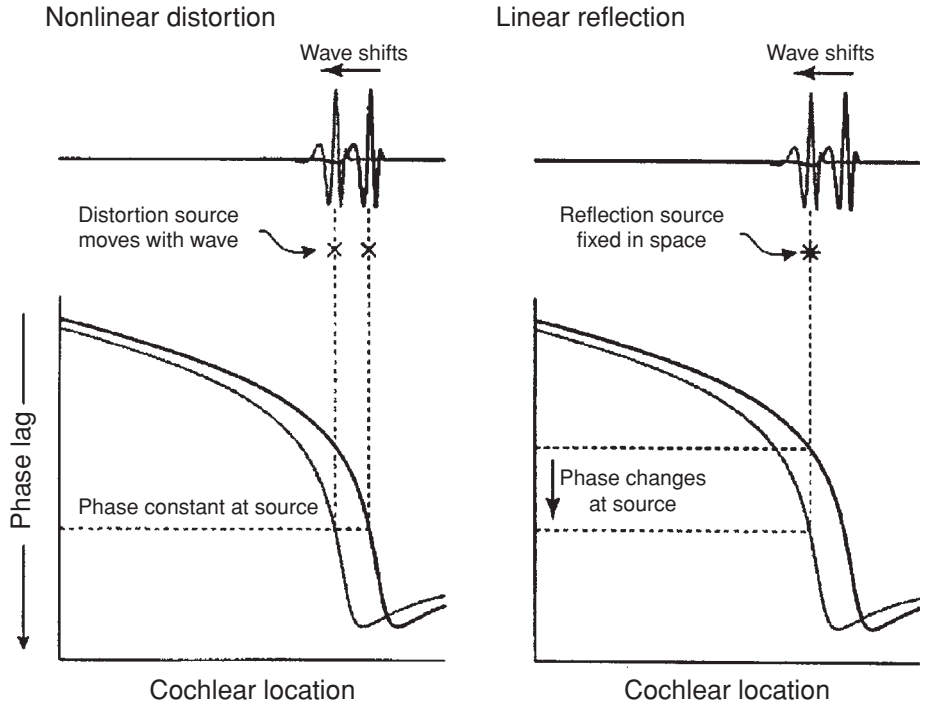


FIGURE 19.1 Schematic illustrations of the phase behavior for emissions arising from either nonlinear distortion (**left panel**) or coherent linear reflection (**right panel**) mechanisms. In either panel, the f_2 traveling wave at two frequencies is shown, one peaking at a more apical location than the other (*top*) along with the corresponding phase lag versus the distance along the basilar membrane (*bottom*). The phase lag function for the more apical wave lies above that for the more basilar wave. For ease of viewing, the f_2 traveling waves have been exaggerated relative to the size of the stapes, the f_1 traveling waves are not shown, and the distortion and reflection sources are idealized as single points (*asterisks*). As seen in the panel on the left, as f_2 is changed to a higher frequency (more basal), the distortion source moves with the wave; therefore, the phase of the wave at the source remains constant as frequency is increased. In the right panel, as f_2 is changed to a higher frequency, the reflection source remains fixed; therefore, the phase at the source changes rapidly as frequency is increased. (Reprinted with permission from Kalluri R, Shera C. (2001) Distortion-product source unmixing: a test of the two-mechanism model for DPOAE generation. *J Acoust Soc Am.* 25 (2), 86–97; ©2001, American Institute of Physics.)

random impedance perturbations in the mechanics of the cochlea or impedance mismatches present at or near the largest displacement of the traveling wave (e.g., Shera and Guinan, 1999). The source of the impedance perturbations is not known, but hypotheses include variations in OHC arrangement or variations in OHCs at the cellular level (Shera, 2004). Shera and Guinan (1999) explain the nonlinear behavior of reflection emissions, such as compressive growth functions, as the result of level-dependent amplification of the forward and reverse traveling waves because of the action of the cochlear amplifier. In this way, reflection emissions, although not generated by the action of OHCs, would be acted on by these forces and would, therefore, still be vulnerable to changes in OHC function.

OAEs measured in the ear canal are thought to be a combination of energy from both mechanisms (Knight and Kemp, 2000; Shera and Guinan, 1999). At this time it is not known whether emissions arising from the two mechanisms might be used differently to provide information about cochlear function. Shera (2004) and others have suggested that by "un-mixing" the energy from the two mechanisms and examining each separately, diagnostic and screening tests with OAEs might be improved. This issue is addressed later in this chapter.

MEASUREMENT OF OAEs

A general recording setup for measuring OAEs includes a sensitive, miniature microphone that fits in the ear canal. Typically, the microphone is housed in a small probe that is coupled to the ear with a foam or rubber tip. The probe contains one or two speakers that allow for presentation of sound stimuli. The microphone measures the OAE coming from the ear and, in the case of some OAEs, also measures the stimuli presented to the ear. The output of the microphone is then amplified. Typically, the amplified output is sampled via an analog-to-digital converter, housed either in a computer or in a stand-alone piece of equipment. The output is then appropriately analyzed for the type of OAE.

The noise level arising from a combination of environmental and internal sources has a significant effect on OAE recordings. High noise levels can obscure low-level OAEs. When high noise levels are present, the number of averages collected must be increased, thereby increasing the test time necessary to obtain a clear OAE recording. Several factors can aid in reducing the effects of noise. First, it is essential to minimize the amount of environmental noise by choosing a quiet or, if possible, sound-treated room as the location for testing. Nonessential equipment, such as fans, should be turned off. Second, the state of the patient being tested must be assessed and addressed. The high noise levels produced by an infant or child that is crying, talking, or generally restless will make testing impossible or extremely difficult. Instructing parents ahead of time as to the nature of the test and that an infant should be asleep during testing can be extremely helpful. Adult patients and older children should be given clear instructions to remain still and quiet during testing. Finally, the importance of an adequate probe fit cannot be overemphasized. A snug and secure probe fit will generally reduce the effects of environmental noise, as well as prevent the loss of low-frequency stimulus energy.

TYPES OF OAEs

Before the recent classification of OAEs based on generation mechanism (e.g., Shera and Guinan, 1999), OAEs were classified into two types based on the recording paradigm, spontaneous and evoked. As the name suggests, spontaneous OAEs (SOAEs) are recorded in the absence of any external stimulation. Evoked OAEs (EOAEs) are measured during or following presentation of an acoustic stimulus to the ear. EOAEs are further subcategorized by the type of stimulus used and related measurement procedure. The clinical literature continues to use this traditional taxonomy; therefore, we will use it here for description of OAEs and their clinical applications.

Spontaneous Otoacoustic Emissions

SOAEs are measured in the absence of external stimulation. They can be measured by viewing what is recorded by the microphone in the frequency domain. As Figure 19.2 illustrates, SOAEs appear as puretone-like signals coming from the ear. This ear has multiple SOAEs, that is, SOAEs at more than one frequency.

SOAEs are measurable in approximately 50% of normal-hearing children and adults. The actual estimates range from 40% (Strickland et al., 1985) to as high as 72% (Talmadge et al., 1993). The wide range of estimates could be because of

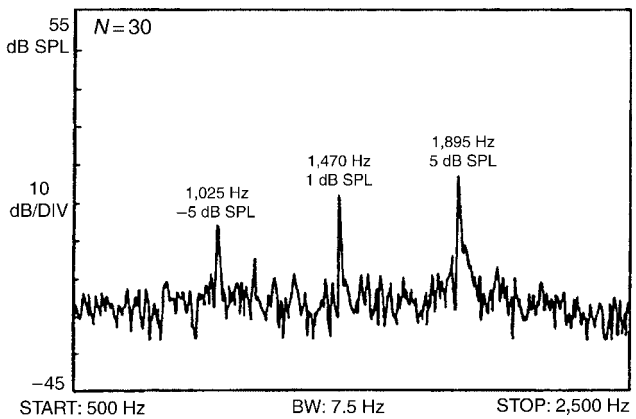


FIGURE 19.2 An example of SOAEs measured from a normal-hearing human. Three SOAEs are measurable. [Modified and used with permission from Lonsbury-Martin BL, Whitehead ML, Martin GK. [1991] Clinical applications of otoacoustic emissions. *J Speech Hear Res.* 34 [5], 964–981; ©1991, American Speech-Language-Hearing Association.]

small sample sizes and recording systems with different noise floors. SOAEs can be measured in ears having hearing loss no greater than 25 to 30 dB HL (Bright and Glattke, 1986). Because they can be measured in only 50% of normal-hearing ears, they are not a useful clinical test. For example, if an individual does not have SOAEs, the clinician cannot distinguish whether that ear has normal hearing or hearing loss. Statistical analyses based on grouped data indicate that the prevalence of SOAEs is higher in females than in males and in right ears than in left ears. Having an SOAE in one ear increases the likelihood that an SOAE will be present in the other ear (Bilger et al., 1990).

Following Kemp's (1979) first report of SOAEs, speculation arose that they might be an objective correlate of at least some forms of tinnitus (Moller, 1989). Studies examining the connection between SOAEs and tinnitus have generally reported that the two phenomena appear to be independent events (Penner and Burns, 1987). Reports of a possible causative relationship between SOAEs and tinnitus have been limited to a few case studies of patients who had normal-hearing sensitivity in at least some frequency regions (Penner, 1988).

Stimulus-Frequency Otoacoustic Emissions

Stimulus-frequency OAEs (SFOAEs) occur at the same frequency and at the same time as a continuous puretone applied to the ear. The microphone in the ear canal records the combination of the puretone being presented to the ear and the SFOAE evoked by the puretone; therefore, specialized measurement techniques must be used to extract the SFOAE from the total signal measured in the ear canal. Common techniques involve introducing a second stimulus differing in intensity or frequency that takes advantage of the nonlinear properties of the SFOAE (e.g., Brass and Kemp, 1991; Schairer and Keefe, 2005). For example, using a tone that is slightly higher or lower in frequency than the evoking stimulus suppresses the SFOAE. A vector subtraction can be made between the sound pressure level in the ear canal when the tone is presented alone (tone plus SFOAE) and the SPL when the tone is presented in the presence of the suppressor (tone alone). The difference between the two conditions is attributed to the SFOAE, or depending on the specifics of the second tone, the portion of the SFOAE that remains unsuppressed by the second tone (e.g., Shera and Guinan, 1999). Other methods, employing three or four condition intervals, have also been used and produce similar results (Brass and Kemp, 1991; Schairer and Keefe, 2005).

Auditory researchers have used SFOAEs to examine various aspects of cochlear function, including cochlear tuning (Shera and Guinan, 2003), and function of the efferent auditory system (Guinan et al., 2003). SFOAEs have not been used as a routine clinical measure, and there are no commercial devices designed to record SFOAEs. Ellison and Keefe (2005) demonstrated that SFOAEs identified hearing loss as well as other EOAEs at 1,000 and 2,000 Hz and were superior to other EOAEs in identifying hearing loss at 500 Hz in their sample of 85 ears. Improvements in measurement techniques and further research on SFOAEs measured under clinical conditions may open the door to future use in general clinical settings.

Transient-Evoked Otoacoustic Emissions

Transient-evoked OAEs (TEOAEs) were the first type of OAE reported in the literature (Kemp, 1978). As their name suggests, TEOAEs are measured following the presentation of a transient or brief stimulus. A click or toneburst is presented to the ear, and the response occurs following a brief time delay. Measurement of TEOAEs is accomplished using time-synchronous averaging. Although the averaging reduces the amount of noise in the trace, it does not remove the stimulus artifact at the start of the recording. The stimulus artifact under typical recording conditions is much larger than the recorded TEOAE. The energy from the stimulus may also persist in the ear canal long enough to obscure the onset of the TEOAE response. Therefore, the first few milliseconds of the trace are usually eliminated from the final averaged waveform to remove energy because of the stimulus.

Figure 19.3 is an example of TEOAE waveform evoked by a click recorded from an adult ear. This example was obtained using Otodynamics ILO88 software and accompanying hardware, the first commercially available program for recording TEOAEs. When a click is used as the stimulus, the resulting TEOAE is often referred to as a click-evoked OAE (COAE or CEOAE). The waveform of the click stimulus is located in the upper left-hand corner of the display. The amplitude spectrum of the click is displayed in the box under the heading "Stim = 80.0 dB." The click used to

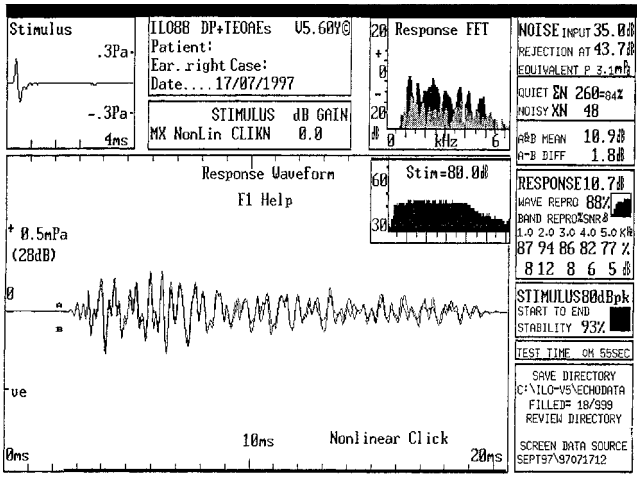


FIGURE 19.3 An example of a COAE recorded from the ear canal of an adult using the Otodynamics ILO88 system.

evoke the COAE was 80 dB pSPL. The largest portion of the display in the lower right contains the waveform of the COAE in the time domain. Notice that the first 2.5 ms have been eliminated to remove the stimulus artifact. If you look closely, you can see that the response is actually composed of two waveforms that have been superimposed. During testing, TEOAE measurement software alternately stores an averaged response in each of two separate buffers, resulting in two averaged traces (labeled "A" and "B" in Figure 19.3).

Comparison of these two waveforms allows the software to determine several TEOAE parameters. For instance, the software calculates the difference between the two waveforms and reports this as the noise level in dB SPL, displayed next to "A–B" in the right-hand column (1.8 dB SPL). The cross-power spectrum of the two waveforms is also calculated and displayed. The cross-power spectrum can be seen in the upper right portion of the display under the heading "Response FFT" (response fast Fourier transform). TEOAE levels are indicated by the dark-shaded regions, whereas the noise levels are indicated by the superimposed lighter-shaded regions.

TEOAEs are often evaluated in terms of level, percent reproducibility, and TEOAE/noise (sometimes called signal-to-noise ratio or SNR). The level of the TEOAE is usually expressed in dB SPL. The level in the example is 10.7 dB SPL and can be found next to the term "Response" in the right-hand column. Below the heading "Band Repro% SNR dB," the percentage reproducibility of the TEOAE in each of the linear frequency bands beginning at 1,000, 2,000, 3,000, 4,000, and 5,000 Hz is listed. Percent reproducibility in this case refers to how well the two TEOAE traces correlate with one another. The software computes inter-waveform correlations in each frequency band, as well as for the broadband waveform, and displays them as percentages. TEOAE/noise or SNR is a ratio of the level of the TEOAE (the "signal") to the level of the noise expressed in dB. TEOAE/noise may be given for the overall TEOAE response or in separate frequency bands. TEOAE/noise levels in Figure 19.3 are listed for the same linear frequency bands under percent reproducibility.

Basic Characteristics of TEOAEs and the Effects of Stimulus and Recording Parameters

Kemp noted in his initial report on TEOAEs that the different frequency components of the evoked responses emerge at different times. Kemp called this phenomenon "frequency dispersion" (Kemp, 1978, p 1387). The dominant period of oscillation changes over time such that higher frequency components of the response appear first, followed by lower frequency components. Typically this phenomenon is described in terms of "latency": higher frequency components have shorter latencies, whereas lower frequency components have longer latencies. Although the measured latencies vary across individuals, the pattern of increasing latency with decreasing frequency remains constant across individuals (Kemp, 1978).

The spectrum of a TEOAE is dependent upon several factors related to the stimulus and recording parameters. One such factor is the spectrum of the evoking stimulus. Broadband click stimuli generally evoke broadband responses. Toneburst-evoked OAEs (TBOAEs) are more frequency specific and typically limited to the frequency range of the narrowband stimuli used to evoke them (Probst et al., 1986). Figure 19.4 displays TBOAEs evoked by tonebursts with carrier frequencies at 1,000 (panel A) and 4,000 Hz (panel B) that were measured with the Otodynamics ILO88 system. The features of the display are identical to those in Figure 19.3. The spectrum of TBOAE energy is narrower than that of the broadband COAE response shown in Figure 19.3.

TEOAE spectra are also influenced by the filter setting and recording time window. As mentioned previously, the stimulus artifact must be removed from the start of the

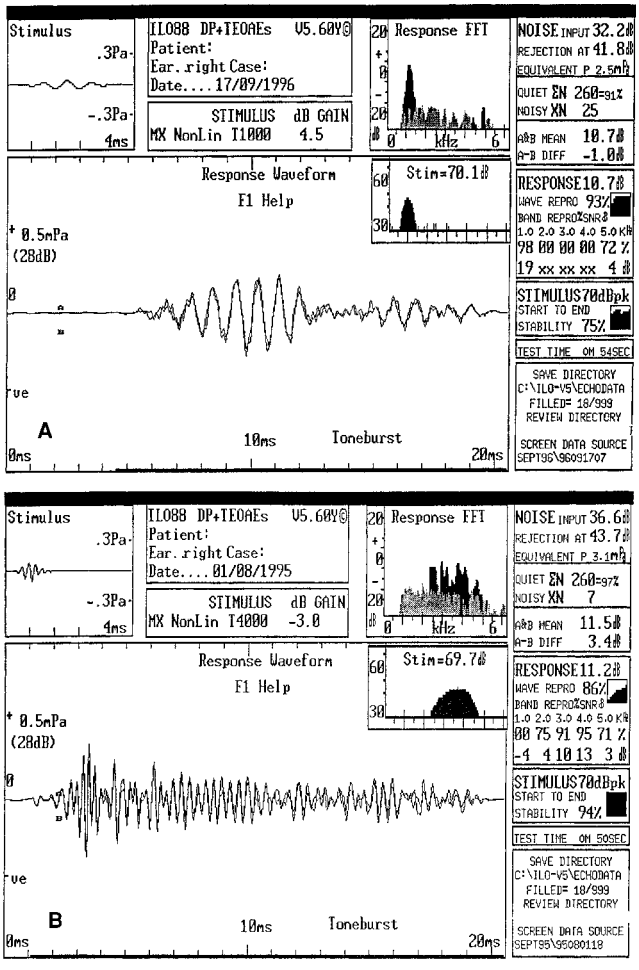


FIGURE 19.4 Examples of TBOAEs recorded from adults using tonebursts centered at [**A**] 1,000 Hz and [**B**] 4,000 Hz. The recordings were obtained using the Otodynamics ILO88 system.

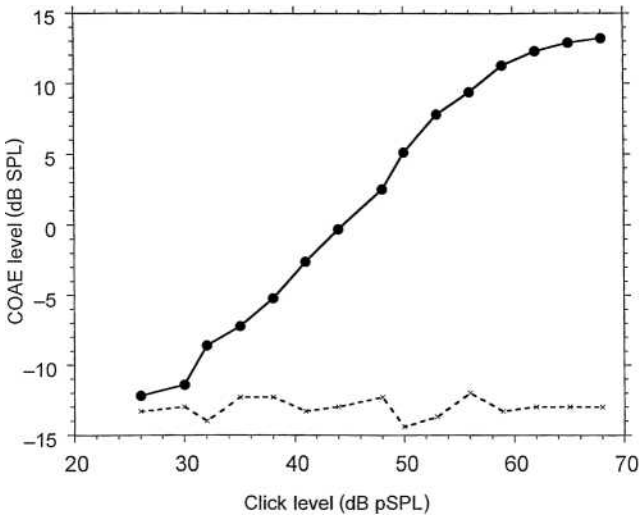


FIGURE 19.5 A COAE growth function recorded from an adult. The *filled circles* indicate COAE levels in dB SPL. The *small Xs connected by dotted lines* indicate the corresponding noise floor levels in dB SPL. The COAE level grows linearly with increases in stimulus levels at low–moderate levels of stimulation; however, the COAE level begins to saturate at higher levels of stimulation (60 to 70 dB pSPL).

averaged trace. Unfortunately, the removal of the first few milliseconds from the trace may also result in the loss of some high-frequency components of the response. The greater the number of milliseconds eliminated from the start of the trace, the greater the amount of high-frequency energy that is potentially eliminated. Conversely, reducing the length of the response time window can result in the loss of some low-frequency energy. The exact TEOAE spectrum and the latency of the different frequency components measured are unique to each individual (Kemp, 1978). These individual TEOAE features are stable within a given ear over time, barring changes in hearing sensitivity (Antonelli and Grandori, 1986).

TEOAE level varies with stimulus level. TEOAE level displays nonlinear growth characteristics in the majority of ears. An example of a TEOAE growth or input/output (I/O) function can be seen in Figure 19.5. At low-to-moderate stimulus levels, growth is fairly linear. However, at higher levels of stimulation, typically between 50 and 80 dB pSPL depending on the type of stimulus used, growth functions show saturation (Zwicker, 1983). As the stimulus level is increased at these high levels, little or no growth in the TEOAE level is noted. The growth function in Figure 19.5 shows saturation between click levels of 60 and 70 dB pSPL.

TEOAE level also varies widely across individuals. For example, Robinette (1992) reported COAE levels for a group of 265 normal-hearing adults that ranged from 0.1 to 22.3 dB SPL in response to clicks presented at a mean level of 81 dB pSPL (SD = 2.7 dB). Results from studies in our laboratory have indicated that the standard deviation for COAE levels recorded at click levels from 40 to 80 dB pSPL is approximately 5 dB (Prieve et al., 1997a).

It is important to keep in mind the spectrum of the stimulus when examining TEOAE growth functions and, in particular, when making comparisons between COAEs and TBOAEs. Although a toneburst and click may have the same overall peak level, the energy from the click is distributed across a much broader range of frequencies. This will result in a lower spectrum level at any given frequency for the click. When click and toneburst stimuli are equated for spectrum level, levels and growth behavior of TEOAEs evoked by the two stimuli are similar (Prieve et al., 1996).

Distortion-Product Otoacoustic Emissions

Distortion-product OAEs (DPOAEs) are measured simultaneously with the presentation of two puretone stimuli, called "primaries", to the ear. The frequencies of the primaries are conventionally designated as "f_1" and "f_2" ($f_1 < f_2$) and the corresponding levels of the primaries as "L_1" and "L_2." When f_1 and f_2 are reasonably close in frequency, interaction of the two primaries on the basilar membrane results in the output of energy by the cochlea at other discrete frequencies that are arithmetically related to the frequencies of the primaries (e.g., $f_2–f_1$, $2f_1–f_2$, $3f_1–2f_2$, $2f_2–f_1$). DPOAEs can therefore be measured using narrowband filtering centered at the frequency of interest. The spectrum in Figure 19.6 displays

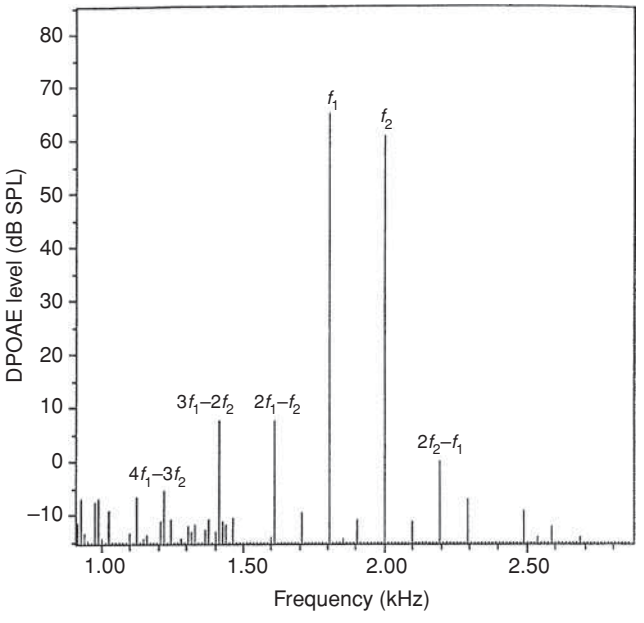


FIGURE 19.6 A spectrum showing two input primaries ($f_1 = 1.807$ kHz, $f_2 = 2.002$ kHz, $L_1 = 65$ dB SPL, and $L_2 = 60$ dB SPL) and the resulting DPOAEs from the ear of a 16-month-old child. Several DPOAEs occurring at frequencies below the primaries ($4f_1–3f_2 = 1.222$ kHz, $3f_1–2f_2 = 1.417$ kHz, $2f_1–f_2 = 1.612$ kHz) and above the primaries ($2f_2–f_1 = 2.197$ kHz) are labeled.

the primaries and DPOAEs measured in the ear canal of a 16-month-old child (using the Otodynamics ILO92 software). Notice that DPOAEs are present at frequencies both lower (e.g., $2f_1–f_2$, $3f_1–f_2$) and higher (e.g., $2f_2–f_1$) than the primaries.

DPOAEs measured in the ear canal are a combination of energy from a nonlinear distortion component originating at the region of overlap between the primaries and a reflection component originating from the region of the DPOAE frequency (e.g., Shera and Guinan, 1999). The mechanism that dominates the response seems to depend on which DPOAE is being measured, as well as the primary levels and the frequency relationships between the primaries (Knight and Kemp, 2000). Additional sources located basal to the f_2 primary tone may also contribute to the $2f_1–f_2$ DPOAE level under some measurement conditions (Martin et al., 2011).

DPOAE measurement systems provide a measure of both the DPOAE and surrounding noise level. The noise level is most often determined by averaging the levels in several frequency bins on either side of the DPOAE of interest. The presence of a particular DPOAE is determined by comparing the level measured within its frequency bin with the noise levels in the surrounding frequency bins and employing some difference criterion. For instance, the DPOAE might be considered present if its level is 3 dB or more above the level of the surrounding noise floor, or if its level exceeds 2 standard deviations above the mean noise level.

On average, the $2f_1–f_2$ DPOAE has the largest level in human and other mammalian ears compared to other DPOAEs (Gaskill and Brown, 1990). As a result, $2f_1–f_2$ is the DPOAE that has been the most extensively investigated, particularly for clinical purposes. The $2f_1–f_2$ DPOAE has sometimes been referred to as the cubic difference tone (CDT). Unless otherwise specified, the use of the term DPOAE will henceforth refer to $2f_1–f_2$.

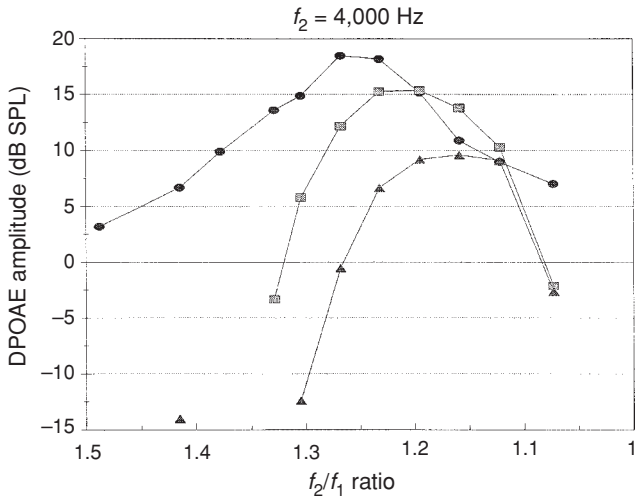


FIGURE 19.7 A graph of DPOAE level as a function of f_2/f_1 ratio from an adult ear at three different stimulus levels (f_2 = 4,000 Hz). The primary levels used to evoke the DPOAEs were L_1 = 75 and L_2 = 60 dB SPL (*circles*), L_1 = 65 and L_2 = 50 dB SPL (*squares*), and L_1 = 55 and L_2 = 40 dB SPL (*triangles*). As the ratio f_2/f_1 is decreased from a value of approximately 1.5, the DPOAE level increases to a broad maximum at ratios of 1.2 to 1.3 and then declines as the ratio is further decreased. The bandpass-shaped function is typical in both infants and adults.

Basic Characteristics of the $2F_1–F_2$ DPOAE and the Effects of Stimulus and Recording Parameters

DPOAE levels vary widely across individual ears. Typical DPOAE levels reported for adults range from 45 to 75 dB below the level of equal-level primaries (Lonsbury-Martin et al., 1990). Early studies revealed that $2f_1–f_2$ level is highly dependent upon various parameters of the primary tones, including their frequency, frequency separation, level separation, and overall level (Gaskill and Brown, 1990; Harris et al., 1989). The frequency separation of the two primaries, generally described as the f_2/f_1 ratio, influences the DPOAE level that will be measured. Figure 19.7 displays DPOAE level as a function of f_2/f_1 as measured from an individual adult ear for f_2 = 4,000 Hz and L_2 levels of 40, 50, and 60 dB SPL when $L_1–L_2$ = 15 dB. As f_2/f_1 is decreased from larger values (~1.5) to progressively smaller values, DPOAE level increases to a broad maximum and then progressively decreases. The exact shape of the function and the f_2/f_1 that produces the maximum DPOAE level vary across individuals, stimulus levels, and frequencies (Neely et al., 2005). However, when data are averaged across many persons, the largest DPOAE levels have repeatedly been obtained with an f_2/f_1 of approximately 1.2 (Harris et al., 1989). As a result, an f_2/f_1 ratio of approximately 1.2 is a common default setting for DPOAE measurement in the clinic. The general shape and features of f_2/f_1 functions appear to be the same for newborns and adults (Abdala, 1996).

The decrease in level at large ratios, that is, when the two primary frequencies are widely separated, is not surprising. The further apart the primaries are from one another, the less the interaction of their respective traveling waves on the basilar membrane. At small ratios, the most likely scenario is that interaction of waves from two or more sources within the cochlea results in cancellation of some energy (Stover et al., 1999). Clinical studies have fixed f_2/f_1 to values approximating 1.2 to evoke maximal DPOAE levels in the greatest number of persons.

The level difference between the two primaries (L_1 vs. L_2) also affects the measured DPOAE level. The level difference that produces the largest DPOAE levels depends on overall primary levels. Equal-level primaries produce the largest DPOAEs at high levels of stimulus presentation; however, the level difference between the two primaries that produces the largest DPOAEs increases ($L_1 > L_2$) as the overall level of the primaries decreases (Gaskill and Brown,

1990; Harris et al., 1989). Kummer et al. (1998) suggested using the equation $L_1 = 0.4 \times L_2 + 39$ dB (for $L_2 < 65$ dB SPL) to set the primary levels to generate maximal DPOAE levels. The equation was based on earlier data (Gaskill and Brown, 1990) and designed to allow for level-dependent changes in the overlap between the two primaries at the f_2 place. Later work by Neely and colleagues indicated that larger DPOAE levels could be measured if the L_1 and L_2 relationship varied with f_2 frequency as well as with L_2 level (Johnson et al., 2006; Neely et al., 2005); however, customizing level and frequency ratios has not improved identification of hearing loss (Johnson et al., 2010).

As would be expected, changes in the overall levels of the primaries also affect DPOAE level. As with TEOAEs, the effects of overall primary levels on DPOAE level are usually graphed as a growth function. With the primaries fixed at a specific pair of frequencies, the DPOAE level is recorded as the levels of the primaries are increased. In general, DPOAE level increases as the level of the primaries increase. Growth functions that include data averaged across a group of persons usually display growth similar to that seen for TEOAEs (e.g., Lonsbury-Martin et al., 1990). However, individual growth functions have a variety of shapes.

TEOAEs and DPOAEs and Patient Characteristics

Whenever a measurement tool is proposed for clinical use, an important step is to determine normative values. One part of this process is to determine whether measured differences can be explained by patient characteristics such as age or gender. TEOAE and DPOAE levels change with development from infancy to adulthood; however, the exact time course remains uncertain. Early group data established that COAE and DPOAE levels in neonates (infants less than 1 month of age) are larger than in adults (Kok et al., 1993). TEOAEs and DPOAEs are also significantly larger in infants than in toddlers, older children, and adults (Prieve et al., 1997a, 1997b). Differences in level with development are frequency dependent, with infants and children having higher EOAE levels than older children and adults for the higher frequencies but not lower frequencies (Prieve et al., 1997a, 1997b). The differences in EOAE levels across age groups have most often been attributed to anatomical changes in the outer or middle-ear systems that occur with development (e.g., Keefe and Abdala, 2007).

The effect of increased age on COAEs and DPOAEs has been more difficult to study, because behavioral thresholds tend to worsen with increasing age, creating a confounding factor. Several early studies reported decreasing COAE and DPOAE levels with increasing age; however, they did not control for auditory threshold (Bonfils et al., 1988). The results of most studies that attempted to control for behavioral thresholds indicated no aging effects on TEOAEs (Stover and Norton, 1993). Older ears have lower DPOAE levels at 8,000 Hz, but the difference is small and has little clinical significance (Dorn et al., 1998).

There are small but significant differences in TEOAE levels depending on ear and gender. Females, on average, have larger TEOAEs than males and right ears, on average, have larger TEOAEs than left ears. The exact reasons for these differences are unknown, although investigators hypothesize that the smaller size of the female ear canal on average compared to the male ear canal may result in the higher TEOAE levels measured in female ears (Robinette, 1992). The greater number of SOAEs measured in right ears as compared to left ears and in females as compared to males may also contribute to the noted TEOAE level differences (Bright and Glattke, 1986). For DPOAEs, some researchers have found that DPOAE levels are larger in females than in males (Gaskill and Brown, 1990). Others have reported significant differences between the sexes at only select frequencies (Lonsbury-Martin et al., 1997). Lonsbury-Martin et al. (1997) reported no significant differences in DPOAE levels between the right and left ears. Both TEOAE and DPOAE levels are generally larger in ears with measurable SOAEs compared to ears without any measurable SOAEs (Prieve et al., 1997a, 1997b). The presence of SOAEs also affects the spectrum of TEOAEs. SOAEs can synchronize to the evoking stimulus, resulting in peaks at those frequencies in the TEOAE spectrum (Probst et al., 1986).

OAEs ARE EXCELLENT CLINICAL TESTS

Before beginning a discussion of clinical applications of EOAEs, it should be noted that middle-ear status has an effect on EOAE measurements. Stimuli used to evoke the EOAE must pass through the middle ear to stimulate the cochlea, and the EOAE energy must pass through the middle ear for it to be detected in the ear canal. Middle-ear pathology may reduce OAE amplitude or eliminate the ability to measure OAEs entirely, depending on the type and severity of the pathology (Owens et al., 1992; Prieve et al., 2008). The majority of the clinical research studies of OAEs have attempted to include only ears free of middle-ear pathology, usually confirmed by routine immittance testing and/or the absence of any air-bone gaps on the audiogram. Obtaining middle-ear measures to elucidate middle-ear function is essential for interpretation of OAE results, particularly when using OAEs for differential diagnosis.

Any clinical test must be robust and accurately identify diseased and nondiseased systems. In his original paper, Kemp (1978) demonstrated that individuals with at least moderate hearing loss had no TEOAEs for low-level stimuli. As audiologists, the critical questions are, given an EOAE test result, what is the expected hearing loss, and what is the range of error? The first step in addressing these questions is to view distributions of EOAEs in normal-hearing and hearing-impaired populations. The goal is to select appropriate

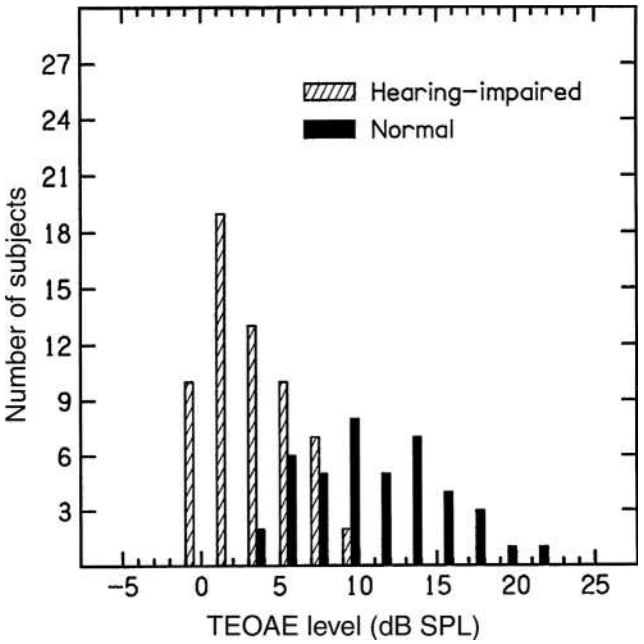


FIGURE 19.8 Frequency distributions of TEOAE level [dB SPL] evoked by 80-dB peSPL clicks [nonlinear recording mode] for normal-hearing [*solid bars*] and hearing-impaired ears [*striped bars*]. [Modified and reprinted with permission from Prieve BA, Gorga MP, Schmidt A, Neely S, Peters J, Schultes L, et al. [1993] Analysis of transient-evoked otoacoustic emissions in normal-hearing and hearing impaired ears. *J Acoustic Soc Am.* 93, 3308–3319; ©1993, Acoustical Society of America.]

criteria that will classify a person as either normal or hearing-impaired. Figure 19.8 illustrates histograms for TEOAE level evoked by 80-dB pSPL clicks from normal-hearing participants (solid bars) and hearing-impaired participants (striped bars). Normal-hearing participants tend to have TEOAEs with higher levels (bars further to the right on the *x*-axis) and hearing-impaired participants tend to have TEOAEs with lower levels (further to the left on the *x*-axis). However, some overlap exists between the two groups of participants, that is, there are some normal-hearing and hearing-impaired participants who have similar TEOAE levels. Unfortunately, this overlap means that it will not be possible to separate the two groups with complete accuracy using EOAEs as they are currently measured, although test performance, as we will see, is still fairly good.

One way to evaluate how well EOAE levels identify hearing status as normal or impaired is to use statistical decision theory. This theory underlies signal detection theory (Green and Swets, 1966, 1974) and clinical decision analysis (Weinstein and Fineberg, 1980). To complete these analyses, data must be collected on a large group of participants for whom hearing status is known. Audiometric threshold is used as the "gold standard" against which the EOAE results are compared. An audiometric criterion is chosen to classify each person as normal hearing or hearing impaired. A typical criterion is 20 dB HL; those with audiometric thresholds of 20 dB HL or better would be classified as having normal hearing whereas those with thresholds higher than 20 dB HL would be classified as having hearing impairment. Second, various EOAE parameters and criteria (e.g., range of EOAE levels in dB SPL, range of EOAE/noise, range of percent reproducibility) are varied to determine the percentage of "hits" and "false alarms" for each EOAE criterion. The percentage of hits ("hit rate") can be plotted as a function of the percentage of false alarms from all the two-by-two tables, resulting in a relative operating characteristic (ROC) curve. Example ROC curves from Prieve et al. (1993) for overall TEOAE level (squares), TEOAE/noise (circles), and percent reproducibility (triangles) are shown in Figure 19.9. The dotted lines illustrate theoretical ROC curves and their corresponding *d'*, which is a measure of the distance between two means of populations having equal-variance, Gaussian distributions.

The less overlap there is between the two populations, the more accurately the test will identify members of the two groups and the more the data in the ROC curve will be located in the upper left-hand corner of the plot. One way to measure the accuracy of the test is to measure the area under the ROC curve, referred to as $P(A)$. The area under the curve

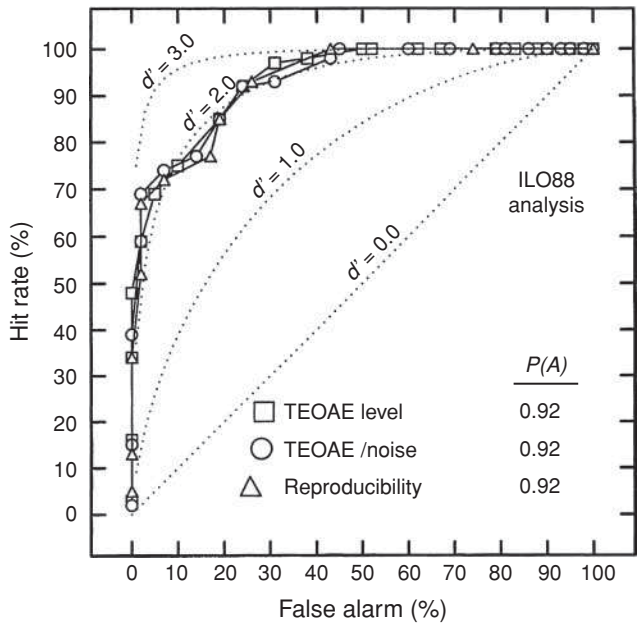


FIGURE 19.9 ROC curves for broadband responses from the ILO88. TEOAE level, TEOAE/noise, and percent reproducibility are represented by different symbols. [Modified and reprinted with permission from Prieve BA, Gorga MP, Schmidt A, Neely S, Peters J, Schultes L, et al. [1993] Analysis of transient-evoked otoacoustic emissions in normal-hearing and hearing impaired ears. *J Acoust Soc Am.* 93, 3308–3319; ©1993, Acoustical Society of America.]

provides an approximation of the hit rate averaged across all false alarm rates. A perfect test that completely separates the two groups would yield a $P(A)$ of 1.0 (hit rate rises to 1.0 whereas false alarm rate remains at 0) whereas a test that separates the two groups no better than chance would yield a $P(A)$ of 0.5 (equal hit and false alarm rates for the various criteria).

TEOAEs

For broadband TEOAEs, the ability to separate normal and hearing-impaired ears is good, and equally so for the three different TEOAE parameters analyzed. The areas under the ROC curves shown in Figure 19.9 are all 0.92. For this analysis, the broadband TEOAE data were compared to the worst threshold at either 500, 1,000, 2,000, or 4,000 Hz. To determine if reduction or absence of TEOAEs in a particular frequency band could predict hearing loss at the corresponding audiometric frequency, the broadband TEOAE was parsed into one-octave bands. The ROC curves for each of the bands are shown in Figure 19.10. Again, all three TEOAE parameters perform almost equally well for separating normal-hearing from hearing-impaired ears. Identification of hearing loss is excellent at 2,000 and 4,000 Hz, indicated by the fact that the ROC curves fall into the upper left-hand corner of the plots and the $P(A)$ values are high. Identification of hearing loss is fair at 1,000 Hz and there is virtually no separation in the responses from normal-hearing and hearing-impaired ears at 500 Hz. These results suggest that TEOAEs evoked by 80-dB pSPL clicks parsed into frequency bands identify hearing loss well at 2,000 and 4,000 Hz, but not at lower frequencies. Prieve et al. (1993) also found that ROC curves were best when using audiometric criterion of 20 or 25 dB HL to separate normal-hearing and hearing-impaired ears.

Subsequent studies have expanded on this early research to determine if improvement in identification of hearing loss can be obtained when using tonebursts, different stimulus levels, and combinations of toneburst and click stimuli presented at different levels (Vinck et al., 1998). Tonebursts centered at 500 Hz provide better separation of normal and hearing-impaired ears than using a 500-Hz band analyzed from a wide-band, click-evoked TEOAEs, but TEOAEs evoked by 2,000-Hz tonebursts were not better than the 2,000-Hz band of a click-evoked response (Lichtenstein and Stapells, 1996).Various stimulus levels have also been studied. Harrison and Norton (1999) reported that the hit rate for identification of hearing loss given a 5% false-positive rate is higher for 80-dB peSPL clicks than for 86-dB peSPL clicks. Vinck et al. (1998) reported that TEOAEs evoked by 86-dB peSPL clicks have a lower hit rate, but higher correct rejection rate, than those evoked by 65-dB peSPL clicks. Based on these results, it appears that mid-level clicks (65 to 80 dB pSPL) may be best for identification of hearing loss.

Investigators have performed multivariate analysis to determine whether combining information from several different TEOAE parameters (such as TEOAE level and the surrounding noise) for various frequencies can identify hearing loss better than using a single parameter to identify hearing loss at the corresponding frequency. The assumption is that a multivariate approach may take advantage of any interactions across frequency and result in an improvement of identification of hearing loss. Hussain et al. (1998) measured TEOAEs over a 12.5-ms window evoked by 80-dB pSPL clicks in participants having normal-hearing and hearing loss. They used two types of multivariate analysis, discriminant analysis and logistic regression, to analyze TEOAE and noise levels in octave bands centered at 1,000, 2,000, and 4,000 Hz. The areas under the ROC curves were calculated and univariate results (TEOAE level,

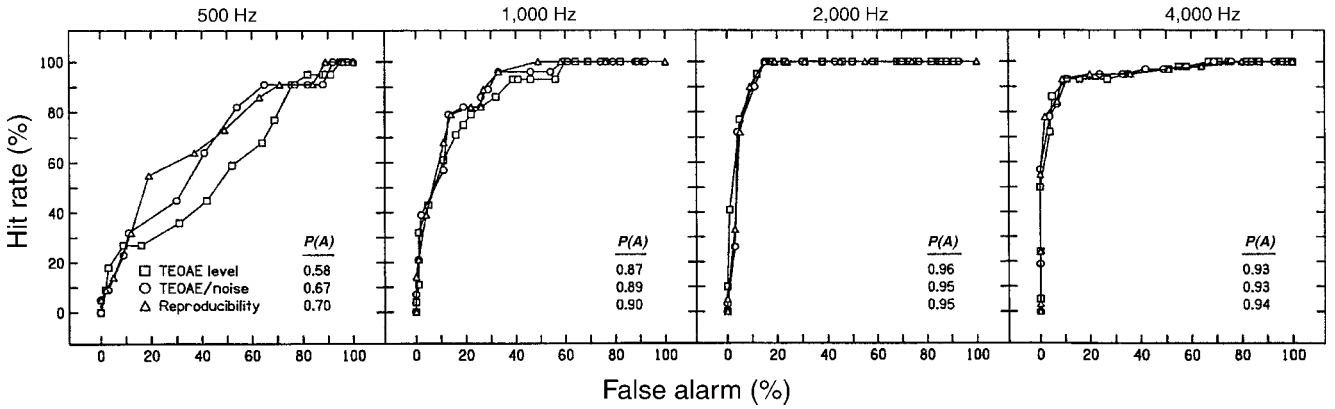


FIGURE 19.10 ROC curves for octave bands centered at 500, 1,000, 2,000, and 4,000 Hz. Bands were analyzed from the broadband TEOAE response. [Modified and reprinted with permission from Prieve BA, Gorga MP, Schmidt A, Neely S, Peters J, Schultes L, et al. [1993] Analysis of transient-evoked otoacoustic emissions in normal-hearing and hearing impaired ears. *J Acoust Soc Am.* 93, 3308–3319; ©1993, Acoustical Society of America.]

TEOAE/noise) were compared to multivariate results (those obtained using discriminant analysis and logistic regression). Hussain et al. (1998) found significant improvement in identification of hearing loss at 1,000 Hz using multivariate analysis.

Vinck et al. (1998) used two stimulus levels (86 and 65 dB pSPL) and two stimulus types (tonebursts and clicks) to determine if these combinations would result in better identification of hearing loss. Using discriminant functions, they found that hearing status at 4,000 and 8,000 Hz could be classified into three groups (<20, 20 to 35, >35 dB HL) with 100% accuracy by using two click levels and four tonebursts presented at two levels. The studies using multivariate analyses suggest that combinations of OAEs at more than one frequency will predict auditory status with the greatest accuracy.

DPOAEs

For clinical purposes, DPOAE levels are examined as a function of primary tone frequency. The primary tone levels, level difference, and frequency ratio are held constant whereas the primaries are changed in frequency. The resulting graph of DPOAE level as a function of frequency has been called a "DP-gram." Figure 19.11 is an example of a DP-gram from an adult ear. Most commonly, DPOAE level is graphed as a function of f_2 frequency. This convention is a result of evidence suggesting the principal source of DPOAEs was the nonlinear distortion component originating in the region of overlap between the primaries (Kummer et al., 1995; Martin et al., 1987).

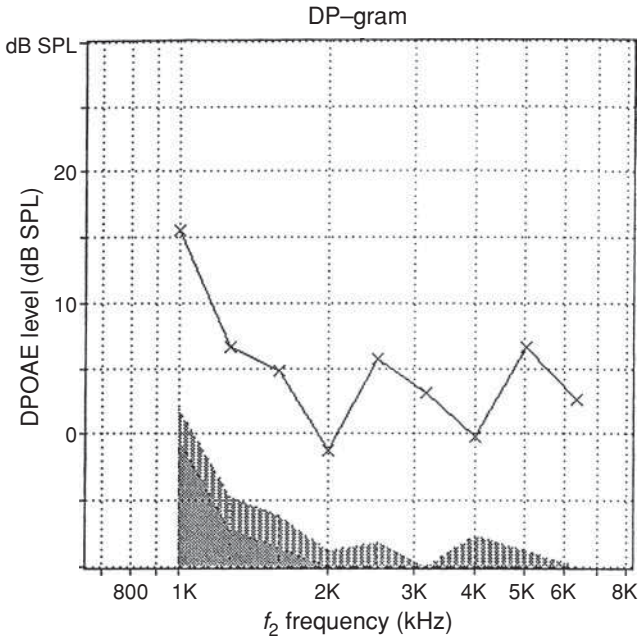


FIGURE 19.11 A DP-gram measured from an adult. The DPOAE level in dB SPL, indicated by the *X*s, is graphed as a function of f_2 frequency. The *shaded areas* indicate noise levels in dB SPL. [Adapted from the output of the Otodynamics ILO92 system.]

Typical stimulus parameters for measuring a DP-gram are levels of $L_1 = 65$ dB SPL and $L_2 = 55$ dB SPL with the f_2/f_1 ratio set to approximately 1.2. The use of these parameters resulted primarily from the laboratory research investigating the stimulus parameters that produced, on average, the largest $2f_1–f_2$ in ears with normal hearing (e.g., Harris et al., 1989) discussed previously. Early animal research indicated that DPOAEs evoked by low-level stimuli more accurately reflected changes in the "active" mechanism of the cochlea (e.g., Schmiedt and Adams, 1981) and that $2f_1–f_2$ DPOAE levels evoked by low- and mid-level stimuli were greatest when unequal-level primaries were presented (Gaskill and Brown, 1990). Later work by Stover et al. (1996) used unequal-level stimuli ($L_1 = L_2 + 10$ dB) at nine different frequencies and 12 different L_2 levels to evoke DPOAEs in a group of normal-hearing and hearing-impaired participants. They found that areas under the ROC curves were greatest for L_2 levels of 55 dB SPL, regardless of the frequency. In addition, using several different L_2 levels did not improve the accuracy of identification of hearing loss. DPOAEs identified hearing loss best at frequencies above 500 Hz.

Based on this preliminary work by Stover et al. (1996), Gorga and his colleagues tested 1,267 ears using primary levels of $L_1 = 65$ dB SPL and $L_2 = 55$ dB SPL in a clinical setting. The data were analyzed in many ways to determine the best way to use DPOAEs to diagnose hearing loss. Gorga et al. (1997) reported how well hearing loss could be identified at a particular audiometric frequency by using the DPOAE evoked by an f_2 of the same frequency. ROC curves indicated that identification of hearing loss was best at 4,000 and 6,000 Hz, with good identification at 1,500, 2,000, 3,000, and 8,000 Hz. They found that DPOAE/noise was marginally superior at identifying hearing loss for most frequencies.

To investigate whether identification of hearing loss could be improved, multivariate analyses were performed. Discriminant function analysis and logistic regression were used to probe whether DPOAE level and noise at other frequencies could aid in the prediction of hearing loss at a chosen frequency. The logistic regression analyses provided the best identification of hearing loss and provided the highest $P(A)$ values. The results of the multivariate analysis suggest that taking into account DPOAE level and noise at various frequencies can improve DPOAE test performance at a given audiometric frequency (Dorn et al., 1999).

In 2005, Gorga and colleagues published a follow-up study that confirmed and expanded on the earlier data set published between 1997 and 1999. DPOAEs were collected from 345 ears of 187 participants. Their ages ranged from 2 to 86 years, with a median of 29.7 years. Data were collected using the same paradigm as the previous studies; however, a commercially available system was used (Bio-logic Scout 3.45) instead of the custom system used in the previous

studies. DPOAE test performance, measured by $P(A)$ areas under ROC curves, that matched an f_2 frequency with an audiometric frequency was similar to their previous report (Gorga et al., 1997). They used the coefficient constants generated by the logit functions (LFs) from the logistic regression analyses performed on data from their previous work (Dorn et al., 1999) to ensure they could be generalized to routine clinic use. The $P(A)$s of ROC curves were similar to their previous study, confirming that the equations could be generalized to new data sets. They found that calculating the LF scores resulted in improved identification of hearing loss, especially for lower frequency primaries, and that many participants with mild hearing loss met passing criteria, similar to their previous work. In summary, the results from the studies on the $2f_1–f_2$ DPOAE indicate that it is an excellent tool for identifying hearing loss. Identification of hearing loss is better at mid-to-high frequencies than at lower (≤1,000) and higher (8,000 Hz) frequencies. Furthermore, taking into account DPOAE parameters at frequencies other than the frequency of interest improves identification of hearing loss.

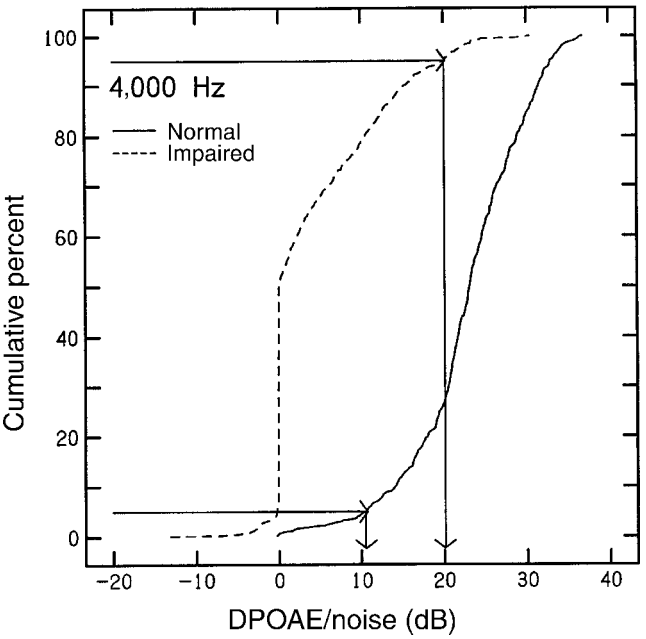


FIGURE 19.12 Cumulative distributions for DPOAE/noise from normal-hearing (*solid line*) and hearing-impaired ears (*dotted line*) for f_2 = 4,000 Hz. (Modified and used with permission from Gorga MP, Neely ST, Ohlrich B, Hoover B, Redner J, Peters J. (1997) From laboratory to clinic: a large scale study of distortion product otoacoustic emissions in ears with normal hearing and ears with hearing loss. *Ear Hear.* 18, 440–455; ©1997, Lippincott Williams and Wilkins.)

CLINICAL USE AND INTERPRETATION OF EOAEs

Identification of Hearing Loss as Part of a Diagnostic Test Battery

Accurate interpretation of EOAE results is essential for providing the best care for patients. Clinicians have interpreted OAEs using different techniques, including templates, ranges from normally hearing ears, and a priori–selected criteria. Each of these techniques is discussed below, along with the strengths and limitations of each technique.

Templates

Gorga et al. (1997) were the first to construct an OAE template for clinical evaluation of hearing loss. To determine criteria that should be used clinically to identify hearing loss, they plotted cumulative distributions of DPOAE level and DPOAE/noise for groups of normally hearing and hearing-impaired ears at each f_2 frequency. Figure 19.12 illustrates the cumulative distributions for an f_2 frequency of 4,000 Hz. In this graph, the percent of persons having a DPOAE/noise value equal to or less than that shown on the *x*-axis is plotted. Data from normal-hearing participants are represented by solid lines and data from hearing-impaired participants are represented by dotted lines. By plotting data in this manner, the value that represents the lowest 5% of DPOAE/noise from normal-hearing ears and the DPOAE/noise that represents the highest 5% from hearing-impaired ears can be determined. The panel for 4,000 Hz illustrates how these cumulative distributions can be used. The 95th percentile of DPOAE/noise for hearing-impaired ears (read from the *y*-axis) is at 20.0 dB (read from the *x*-axis). The 5th percentile for the normal-hearing persons is at 10.5 dB.

Gorga and colleagues incorporated the DPOAE levels corresponding to the 5th and 10th percentiles from normal-hearing ears and the 90th and 95th percentiles from hearing-impaired ears into a plot that can be used clinically, shown in Figure 19.13. The top panel is a template for DPOAE level and the bottom panel is one for DPOAE/noise. In the top panel, the top solid line represents the 95th percentile of DPOAE level for the hearing-impaired ears. The top of the darkened area is the 90th percentile of DPOAE level from the hearing-impaired ears. The lowest solid line represents the 5th percentile of DPOAE level from the normal-hearing ears, and the bottom of the solid portion represents the 10th percentile of DPOAE level from the normal-hearing ears. The solid area represents DPOAE level from the 10th percentile from the normal-hearing ears to the 90th percentile for the hearing-impaired ears. This template allows the clinician to classify the DPOAE level as "normal," "abnormal but present," or "absent." First, the clinician determines whether the DPOAE is higher than the noise floor for each frequency. If the DPOAE is ≥6 dB and an individual's DPOAE level falls above the top line, the audiologist can be confident that hearing at the f_2 frequency is within normal limits. If the patient's

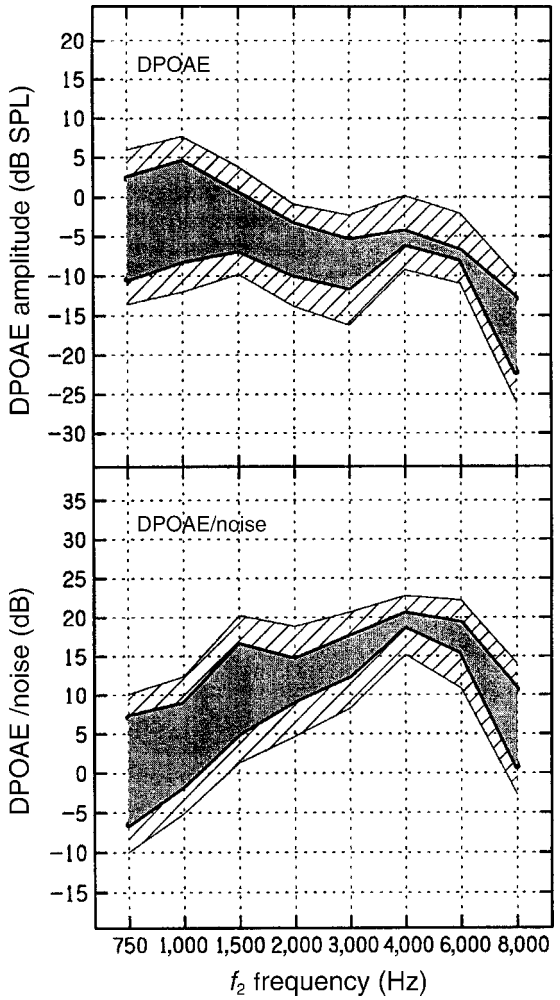


FIGURE 19.13 DPOAE level [**top panel**] and DPOAE/noise [**bottom panel**] plotted as a function of f_2 frequency. The *top-most solid line* represents the 95th percentile of responses from hearing-impaired ears. The *bottom-most solid line* represents the 5th percentile of responses from normal-hearing ears. The *area with diagonal lines on the bottom of the darkened area* represents the 5th to 10th percentile of responses from normal-hearing ears. The *area with diagonal lines at the top of the darkened area* represents the 90th to 95th percentile of responses from hearing-impaired ears. [Modified and used with permission from Gorga MP, Neely ST, Ohlrich B, Hoover B, Redner J, Peters J. From laboratory to clinic: a large scale study of distortion product otoacoustic emissions in ears with normal hearing and ears with hearing loss. *Ear Hear,* 18, 440–455; ©1997, Lippincott Williams and Wilkins.]

DPOAE level falls below the lowest solid line, the audiologist determines that hearing at that f_2 frequency is impaired. The solid area is the "area of uncertainty." If an individual's DPOAE level falls in that range, the clinician cannot be sure whether hearing at that f_2 frequency is impaired or normal, although often, a mild hearing loss is suspected. The classification would be "abnormal level, but present." Templates are also available for TEOAEs for ages 1 to 27 years collected using the ILO88 equipment (Nicholson and Widen, 2007) and for DPOAEs using discriminant function and logistic regression analyses (Dorn et al., 1998).

Using templates increases confidence in clinical interpretation because EOAEs from individual ears can be evaluated relative to a large group of normal-hearing *and* hearing-impaired ears. The limitation of using a template is that the patient's age should be within the range of participant ages for which the template was constructed. EOAEs should be measured using similar stimulus and recording parameters to those used to construct the templates. Care must be taken while using DPOAE/noise comparisons if the number of averages differs from those used to construct the template.

A Priori Criteria

Interpretation using *a priori criteria* means that some EOAE criterion is used to determine whether an EOAE is present or absent at each frequency tested. The presence of EOAEs at a given frequency is assumed to indicate normal OHC function and likely normal hearing at that frequency. Over the years, clinicians have typically adopted SNR criteria for TEOAEs or DPOAEs or percent reproducibility for TEOAEs. Using a priori criteria has been common for TEOAE interpretation. Specifically, a TEOAE SNR of ≥6 dB is often used as the criterion for the presence of TEOAEs in a given frequency band.

The question arises, how does a clinician choose a certain criterion, and how well does it identify hearing loss? Gorga et al. (1999) investigated identification of hearing loss using common a priori DPOAE criteria of 3-, 6-, and 9-dB DPOAE/noise ratios for various combinations of audiometric data. They found that if only one f_2 frequency was evaluated, sensitivity never reached 100%. Sensitivity improved if three to five frequencies were required to meet the criteria, which would result in an interpretation for the ear as a whole, not interpretation of frequency by frequency within an ear. They concluded that clinicians should not assume that a priori criteria identify all ears with hearing loss, even using a stringent criterion of 9 dB.

The strength of using SNR is that it is age independent, but there are severe limitations. First, EOAEs can only be classified as present or absent using this method; there is not an alternative interpretation of present but abnormal. Second, on a particular test, there may be spurious patient or room noise that can affect an SNR, often for only some frequencies, making frequency-by-frequency interpretation difficult. Finally, continued averaging reduces the noise and even small-level EOAEs may meet a criterion SNR. If clinicians feel they must use a priori criteria for interpretation, we recommend that the test be run twice to ensure repeatability.

Comparison to Range for Normally Hearing Ears

Another approach to interpret OAEs is to have a range of OAE levels from normally hearing ears and compare OAE levels from individual ears to it. The range of values is either the mean ±1 or 2 standard deviations or the range of OAE levels between the 10th and 90th percentiles from a group of normally hearing ears. In this paradigm, if the responses from the tested ear are within the range from normally hearing ears, the OAE is considered "normal." If it is lower than the normal range, but higher than the noise, it is considered "abnormal." Finally, if the OAE level is the same or lower than the noise level, the OAE is considered "absent." The determination of normal, present but abnormal, or absent is made on a frequency-by-frequency basis for both ears and included in the patient's report relative to the audiogram. The strength of this method is that hearing loss can be interpreted into three categories. The limitations of this method are that the patient's age must be in the age range as the participants used to construct the normal range. In addition, there is no indication of what DPOAE levels might be like in ears with hearing loss and how those levels overlap with those from normal-hearing ears. This approach is often used for DPOAEs, and many manufacturers provide their own normative range on their DPOAE equipment. This method has not been routinely used for TEOAEs; however, such data could be easily collected.

Differential Diagnosis of Hearing Loss

In addition to identifying hearing loss, EOAEs enhance the audiometric test battery for cases of differential diagnosis. Because EOAEs are generated in the cochlea independent of afferent fiber activation, they allow us to test for a "mechanical" versus an IHC or neural hearing loss. One type of auditory disorder that can now be identified is auditory neuropathy. In these cases, the EOAEs are normal, ABRs and acoustic reflexes are abnormal or absent, and hearing thresholds range from mild to profound in severity.

The addition of EOAEs to the test battery for patients with vestibular schwannoma is also an important consideration. Although most patients having tumors also do not have EOAEs, which is thought to be because of interrupted blood supply to the cochlea, there are a small number of patients that have EOAEs in spite of poor behavioral thresholds. In these cases, the audiologist could recommend that a surgical procedure that preserves the cochlea be taken, so that any residual hearing may be saved (Robinette et al., 2002).

OAE measures identify damage because of drug ototoxicity or noise exposure prior to increases in thresholds (e.g., Stavroulaki et al., 2002). OAEs may be useful for determining susceptibility to noise (Lapsley Miller and Marshall, 2007). Some researchers are investigating the possibility that EOAEs are altered in unique ways in individuals with Meniere's disease (Kusuki et al., 1998; Sakashita et al., 1998).

Newborn Hearing Screening

The Joint Committee on Infant Hearing (JCIH, 2007) recommends universal newborn hearing screening, meaning that all newborn infants are screened for hearing loss. The JCIH has recommended that newborns cared for in the well-baby nursery (WBN) be screened with either EOAEs or ABRs (JCIH, 2007). Because EOAEs do not detect auditory neuropathy or dys-synchrony, EOAEs are not recommended for use as the primary screening tool in the neonatal intensive care nursery.

When used as a screening tool, EOAE results from each ear are evaluated using criteria chosen by the screening program directors. If the criteria are met in both ears, then a newborn is said to "pass" the newborn hearing screening. If the criteria are not met in both ears, the newborn is considered to have "failed" the in-hospital screening, and either returns at a later time for a rescreening or referred for diagnostic testing. EOAEs have been widely used in large-scale screening programs that included both high-risk and large-scale universal newborn hearing screening programs (e.g., Spivak et al., 2000; Vohr et al., 1998). The referral rates for outpatient retesting reported by these programs range between 3% and 10%. After a second, outpatient screening before 1 month of age, referral for diagnostic testing is less than 2% (e.g., Vohr et al., 1998).

A large-scale study, funded by the National Institutes of Health (NIH) directly compared the screening tools of DPOAEs, TEOAEs, and screening ABR (SABR). The NIH study was conducted on 4,478 infants cared for in the NICU, 353 well-babies with risk indicators for hearing loss, and 2,348 infants cared for in the WBN with no risk indicators for hearing loss, with data pooled from seven different institutions across the United States. TEOAEs, DPOAEs evoked by two different primary level pairs, and ABRs evoked by 30-dB nHL clicks were measured in random order from both ears of each infant using a computerized test program that included passing criteria, response filtering, noise/artifact rejection, and minimum, maximum, and low-noise stopping rules specifically chosen for each measure (Gorga et al., 2000b; Norton et al., 2000a; Sininger et al., 2000). One of the important findings from the NIH study is that the percentage of infant passes was similar for SABRs, TEOAEs, and DPOAEs ($L_1 = 65$, and $L_2 = 50$ dB SPL).

To determine the sensitivity and specificity of each screening tool, 4,911 infants were tracked for follow-up behavioral hearing testing at 8 to 12 months corrected age. Sixty-four percent of infants targeted for follow-up returned for audiometric testing, and of those, 95.6% could be reliably tested using visual reinforcement audiometry with

insert earphones. Minimal response levels (MRLs) were determined using frequency-modulated tones at frequencies of 1,000, 2,000, and 4,000 Hz and speech awareness threshold (SAT) was determined using speech presented by live voice through a microphone. MRLs higher than 20 dB HL indicated hearing loss, and infant responses at 20 dB HL were considered normal (Widen et al., 2000). ROC curves were constructed for SABR, TEOAEs, and DPOAEs using several gold standards. The gold standards were MRL at 1, 2, or 4 kHz; the SAT or puretone average (PTA) of 2 and 4 kHz; or a PTA including 1, 2, and 4 kHz. The criterion for normal was ≤20 dB HL (Norton et al., 2000b). The areas under the ROC curves for all four tests ranged from 0.70 to 0.94 and were similar to each other for most measures, indicating that each test identified hearing loss well. None of the areas were 1.0, indicating that no test identified hearing loss with 100% accuracy. There were slight differences among the measures. ROC curves for DPOAEs evoked by equal primary levels of 75 dB SPL had lower areas than those for DPOAEs evoked by primary levels of $L_1 = 65$ and $L_2 = 50$ dB SPL, indicating that DPOAEs evoked by higher level primaries were not as good at identifying hearing loss as those evoked by mid-level primaries. TEOAEs, DPOAEs at 65/50 dB SPL, and SABR performed similarly for MRLs of 2, 4 kHz, PTA including 2 and 4 kHz, and SAT. TEOAEs and DPOAEs at 65/50 dB SPL outperformed SABR slightly for most of these measures. Finally, SABR outperformed TEOAEs and DPOAEs for an MRL at 1,000 Hz and was slightly better for a PTA that included 1,000 Hz. An analysis of failure rate based on severity of hearing loss was performed for a hit rate of 80%. The majority of moderate, severe, and profound hearing losses were identified at a rate at or close to 100% using any of the measures. Mild hearing loss was the severity most incorrectly identified, with 40% to 50% of ears with mild hearing loss meeting the screening criteria (Norton et al., 2000b). An extensive set of data is provided by this study and can help guide clinicians in understanding EOAE and ABR tools as newborn hearing screening tools. Additionally, clinicians should be aware that ABR and EOAE screening are equally effective at identifying moderate, severe, and profound hearing loss.

CLINICAL EOAEs IN THE FUTURE

In the past, the majority of research on the clinical use of EOAEs has focused on the identification of hearing loss or screening for hearing loss. Based on early literature, both TEOAEs and DPOAEs identify hearing loss well (e.g., Gorga et al., 1993; Norton et al., 2000b; Prieve et al., 1993) and are "frequency specific," meaning that either can provide information about hearing loss in a specific frequency region. Although EOAEs have turned out to be excellent at identifying hearing loss, the future use of EOAEs in probing the auditory system for hearing loss could see many changes.

Using DPOAE Components to Diagnose Hearing Loss

Current clinical interpretation of DPOAEs ignores the two-source taxonomy of DPOAE generation. DPOAEs measured in the ear canal are a composite of OAEs from two different generation mechanisms from at least two different places in the cochlea. When DPOAEs are measured in small frequency steps (<50 Hz) or with a sweeping tone (Long et al., 2008), large variations in the DPOAE level, termed "fine structure," are evident (He and Schmiedt, 1993). An example of DPOAE fine structure recorded from an adult is shown in Figure 19.14. Fine structure is the result of the interaction of energy from the nonlinear distortion and reflection components. Depending on the phases of these two sources, they may combine constructively, such that a higher DPOAE level is measured in the ear canal (peaks), or destructively, such that a lower level is measured (dips) (Talmadge et al., 1999).

One approach to improve identification of hearing loss is to suppress the EOAE due to the reflection component, leaving the EOAE due to the generator component (Heitmann et al., 1998). The rationale behind the procedure of this work is that reducing the reflection component reduces the fine structure and thereby reduces the variability of the DPOAE level measured in the ear canal. Johnson and colleagues (2007) used a suppressor to reduce the reflection component on a large number of ears but did not note any improvements in the diagnosis of hearing loss. However, others have found minor improvements in identification when the reflection component is suppressed and optimal calibration and stimulus parameters are taken into account (Kirby et al., 2011).

A second approach is to improve identification, etiology, and/or severity of hearing loss by separating the compound DPOAE into the two underlying components and interpreting them both. Support for looking at both components comes from research indicating that the two components are differentially affected by hearing loss

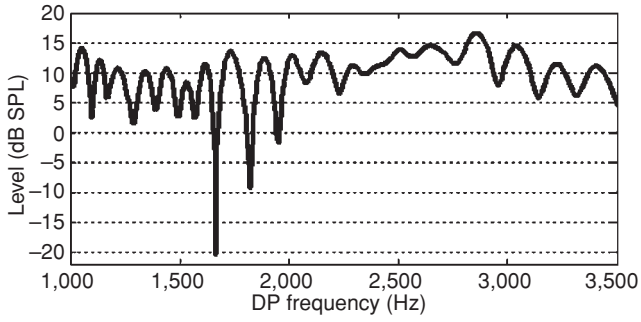


FIGURE 19.14 DPOAE level plotted as a function of $2f_1$–f_2 frequency in an adult. DPOAEs were collected using sweeping-tone primaries which allow observation of fine structure.

(Mauermann et al., 1999), development (Abdala and Dhar, 2012) and aspirin (Rao and Long, 2011). Although separation of DPOAEs into the two subcomponents has been done for years in research settings (Shera, 2004), only recently has it been attempted in large-scale studies with clinical intent (Poling et al., 2014).

As an alternative to measure both DPOAE components, clinical testing could use TEOAEs, which are basically a reflection-type emission (Kalluri and Shera, 2007), and DPOAEs evoked by mid- to high-level primaries to enhance clinical diagnosis. It has been known for many years that DPOAEs can be measured in ears with greater hearing loss than TEOAEs (Harris and Probst, 2002); however, systematic studies of using the two types together are lacking. There are case study examples that indicate absent or abnormal TEOAEs in the presence of DPOAEs (see case study no. 3 in this chapter).

Extended High Frequencies

Extending EOAE measurement to high frequencies (10 to 16 kHz) has been studied in research settings for both DPOAEs (Dreisbach and Siegel, 2001) and TEOAEs (Goodman et al., 2009) and more recently in a large sample of ears (Poling et al., 2014). High-frequency click-evoked TEOAEs identify sensory/neural hearing loss up to 12.7 kHz with excellent accuracy (Keefe et al., 2011). Measuring high-frequency EOAEs is important for understanding age-related changes early in life and for monitoring effects of ototoxic drugs. Presentation of high-frequency stimuli and EOAE recording requires specialized speakers and calibration. Calibration using forward pressure level and techniques to detect standing waves in the ear canal has improved identification of hearing loss using DPOAEs even up to 8,000 Hz (Kirby et al., 2011; Reuven et al., 2013).

Measurement of Input/Output Functions

Another characteristic of EOAEs that can be measured is the input/output (I/O) function. To measure a DPOAE I/O function, the stimulus frequencies (f_1 and f_2) and ratio (f_2/f_1) are fixed and both stimulus levels (L_1 and L_2) are increased from low to high levels. From the resultant DPOAE I/O function, the slope of the function can be evaluated and a threshold can be determined. Typically, threshold is defined as the lowest stimulus level producing a DPOAE that is 3 dB above the noise floor with subsequent growth in level for successively higher stimulus levels. DPOAE I/O functions in human ears with cochlear impairment reveal elevated thresholds, reduced compression, and a steeper slope of the I/O function in comparison to normal ears (Kummer et al., 1998). EOAE thresholds can be estimated by I/O functions; however, they cannot accurately predict behavioral thresholds or provide accurate identification of hearing loss (Gorga et al., 2003; Stover et al., 1999). Input/output functions differ between neonatal ears with middle-ear and cochlear hearing loss (Janssen et al., 2005).

Different DPOAEs

Although almost all research on DPOAEs has focused on $2f_1–f_2$, the $2f_2–f_1$ DPOAE has been evaluated for clinical testing in a few studies. Results indicated that $2f_1–f_2$ predicted hearing status (normal vs. impaired) better than $2f_2–f_1$ at all test frequencies (Fitzgerald and Prieve, 2005; Gorga et al., 2000a), even when stimulus parameters were set to obtain the most robust $2f_2–f_1$ levels (Fitzgerald and Prieve, 2005). Combining information from both $2f_1–f_2$ and $2f_2–f_1$ using multivariate analyses improved prediction of hearing status, but the improvements were very slight compared to the performance of $2f_1–f_2$ alone (Fitzgerald and Prieve, 2005; Gorga et al., 2000b).

CASE STUDIES

Case Study No. 1: Adult COAEs

Patient no. 1 is an 18-year-old adult male with bilateral high-frequency sensory/neural hearing loss. The patient reported that he had been hearing impaired since birth but that the etiology of the hearing loss was unknown. A hearing evaluation revealed a unique pattern of thresholds with two frequency regions of extremely low thresholds in the right ear. The top half of Figure 19.15 is the patient's audiogram for the right ear. Hearing levels improved from borderline-normal at 250 to 500 Hz to extremely low values (−10 dB HL) at 1,000 and 1,500 Hz and back to 15 dB HL at 2,000 Hz. His hearing levels then sloped to a mild-to-moderate hearing loss from 3,000 to 4,000 Hz, followed by another rise to −10 dB HL at 6,000 Hz and a borderline-normal value of 25 dB HL at 8,000 Hz.

A TEOAE was measured from the right ear using clicks presented at approximately 82 dB pSPL. The display from the ILO88 system is shown in the bottom half of Figure 19.15 with the COAE analyzed into half-octave bands. COAEs were present in the half-octave bands from 1,000 to 2,000 Hz, where hearing levels were within normal limits. As expected, no COAE energy was present in the 3,000- to 4,000-Hz bands where hearing loss is indicated on the audiogram. The COAE screening does not yield information above 4,000 Hz for comparison with the normal and borderline-normal thresholds obtained in this patient at higher frequencies. However, this case does illustrate good correspondence between the presence/absence of COAEs and the measured hearing levels from 1,000 to 4,000 Hz. Also of note are the excellent probe fit and low noise level that are typically easy to obtain in a cooperative adult.

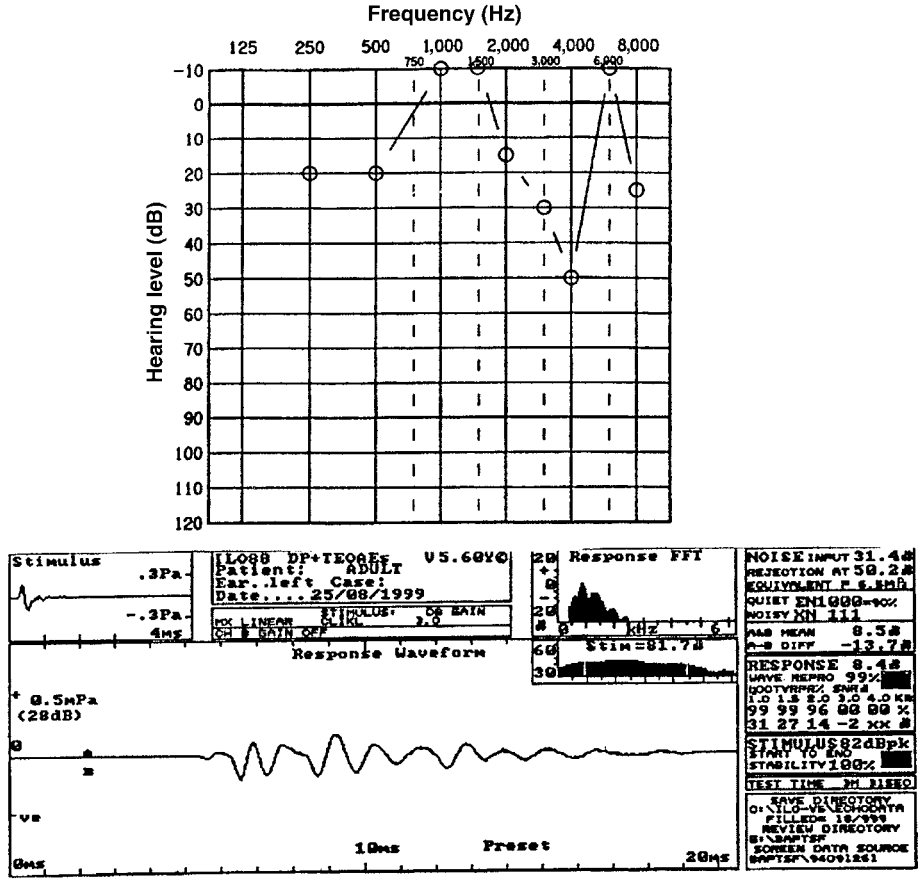


FIGURE 19.15 Audiogram and TEOAEs for case study no. 1: TEOAEs in an adult.

Case Study No. 2: TEOAEs from a Child

Patient no. 2 was referred to us when he was 3:3 (years: months). His parents had first suspected he had a hearing loss by the time he was 1:0. He was taken to an ENT, who inserted ventilating tubes into his eardrums when the child was 1:6. At 2:1 this patient had a hearing evaluation, which revealed a moderate-to-severe mixed hearing loss bilaterally. Expressive and receptive language and phonologic skills were significantly reduced. At that time, the patient underwent a second surgery for PE tubes and afterward was fit with binaural hearing aids. Because the patient did not seem to be responding well to hearing aids, and was difficult to test behaviorally, he was seen for hearing and OAE testing.

The top panel in Figure 19.16 illustrates the patient's audiogram, which was obtained after several visits. Patient no. 2 has a mild, sloping-to-profound sensory/neural hearing loss bilaterally. Because the patient was not responding well to hearing aids, it was important to rule out auditory neuropathy. The middle panel in Figure 19.16 illustrates the TEOAEs measured from the left ear and the bottom panel illustrates the TEOAE from the right ear. Click stimuli presented at approximately 80 dB pSPL were used. No TEOAEs are present, consistent with OHC pathology. Notice the unusual click stimulus waveform, a typical finding when open ventilating tubes are present. Because of the absence of TEOAEs in the presence of a mild-to-profound sensory/neural hearing loss, it was felt that this child's hearing loss contained a cochlear component and that amplification was an appropriate method of rehabilitation. As the child matured, became adjusted to his hearing aids, and received special services, the child became more verbal and compliant in behavior.

Case Study No. 3: Sensory/Neural Hearing Loss in an Infant

Patient no. 3 was referred because she failed a newborn hearing screening before hospital discharge. She was seen at 8 months of age, after having audiologic evaluations at other facilities on three different occasions. Figure 19.17 shows estimates of behavioral thresholds, depicted by triangles, based on ABRs evoked by tonebursts collected during natural sleep. Figure 19.17A shows data for the right ear and Figure 19.17B shows data for the left ear. ABR thresholds estimate a mild-to-severe sensory/neural hearing loss

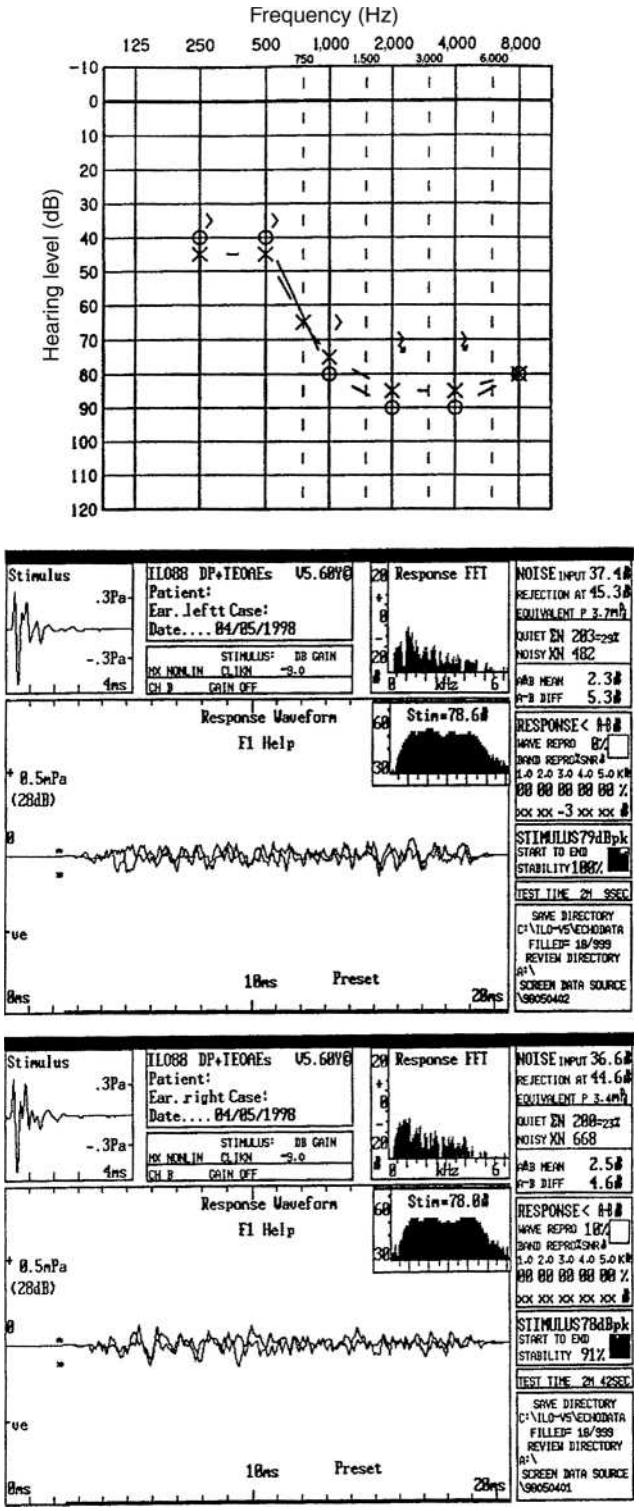


FIGURE 19.16 Audiogram and TEOAEs for case study no. 2: TEOAEs in a child.

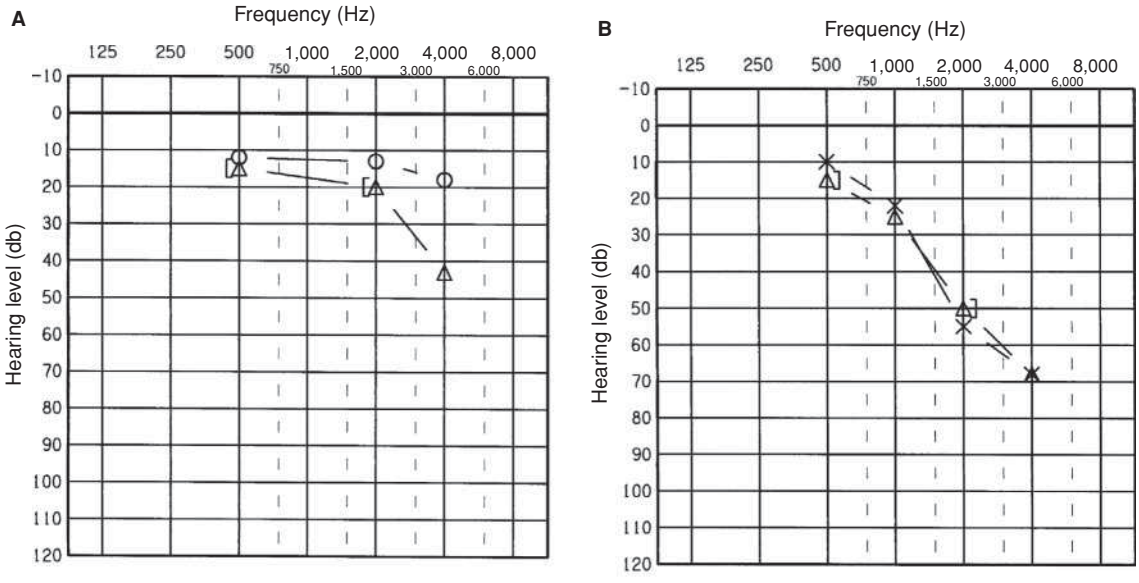
in the left ear and a mild hearing loss in the right ear at 4,000 Hz. Tympanometry was within normal limits for both ears. TEOAEs and DPOAEs were absent in the left ear. In the right ear, TEOAEs were present in 2,000- and 3,000-Hz half-octave bands, but were absent in the 4,000-Hz band. DPOAEs were higher than 2 standard deviations above the noise floor between 1,500 and 4,000 Hz. Behavioral thresholds obtained using visual reinforcement audiometry are shown on the audiograms using typical conventions (x for left ear, circle for right ear). ABR estimates of behavioral thresholds were in good agreement with actual behavioral thresholds except for 4,000 Hz in the right ear. The behavioral threshold was 20 dB HL, which is a slight, rather than mild hearing loss, as suggested by ABR threshold estimates. Lack of TEOAEs in the 4,000-Hz band was consistent with the slightly higher thresholds.

Case Study No. 4: Conductive Hearing Loss in an Infant

Patient no. 4 is a baby aged 4 months who passed his newborn hearing screening at birth, but had developed middle-ear effusion because of a congenital disorder. Based on ABR thresholds, it appeared that patient no. 4 had a conductive hearing loss in the right ear at 500 Hz. A tympanogram using a 1,000-Hz probe tone was flat. Figure 19.18 shows that TEOAEs in the right ear (lower panel of the figure) were of low level, but had >4-dB OAE-to-noise ratio from 2,000 to 4,000 Hz. Patient no. 4 underwent a myringotomy 1 week after testing. The pediatric otolaryngologist noted effusion in the right ear. Although one might expect that middle-ear pathology would eliminate EOAEs, TEOAEs were present in this infant despite an air–bone gap based on toneburst ABR, a flat tympanogram, and confirmed effusion.

FOOD FOR THOUGHT

1. To what extent do otoacoustic emissions change our ability to identify "sensory" and "neural" hearing loss separately, rather than the combined category of "sensorineural" hearing loss?
2. What are the possible errors if you are assessing hearing loss in a 4-month old infant and you use a DPOAE template constructed from data that included patients aged 1–96 years?
3. What are the sources of noise in an OAE recording? Can you reduce the noise from these sources, and if so, how can it best be done?



Right ear

TEOAE

	2 kHz	3 kHz	4 kHz
Level (dB SPL)	0.4	−0.8	−13.8
SNR (dB)	9	6	xx

DPOAE

	2 kHz	3 kHz	4 kHz
Level (dB SPL)	12	5.7	−3.1
SNR (dB)	12.4	20.4	12.5

Left ear

TEOAE

	2 kHz	3 kHz	4 kHz
Level (dB SPL)	xx	xx	xx
SNR (dB)	xx	xx	xx

DPOAE

	2 kHz	3 kHz	4 kHz
Level (dB SPL)	−7.4	xx	xx
SNR (dB)	5.9	xx	xx

FIGURE 19.17 Case study no. 3. Behavioral thresholds based on toneburst auditory brainstem response, behavioral thresholds measured with visual reinforcement audiometry at 8 months of age, and TEOAEs and DPOAEs measured at 3 months of age.

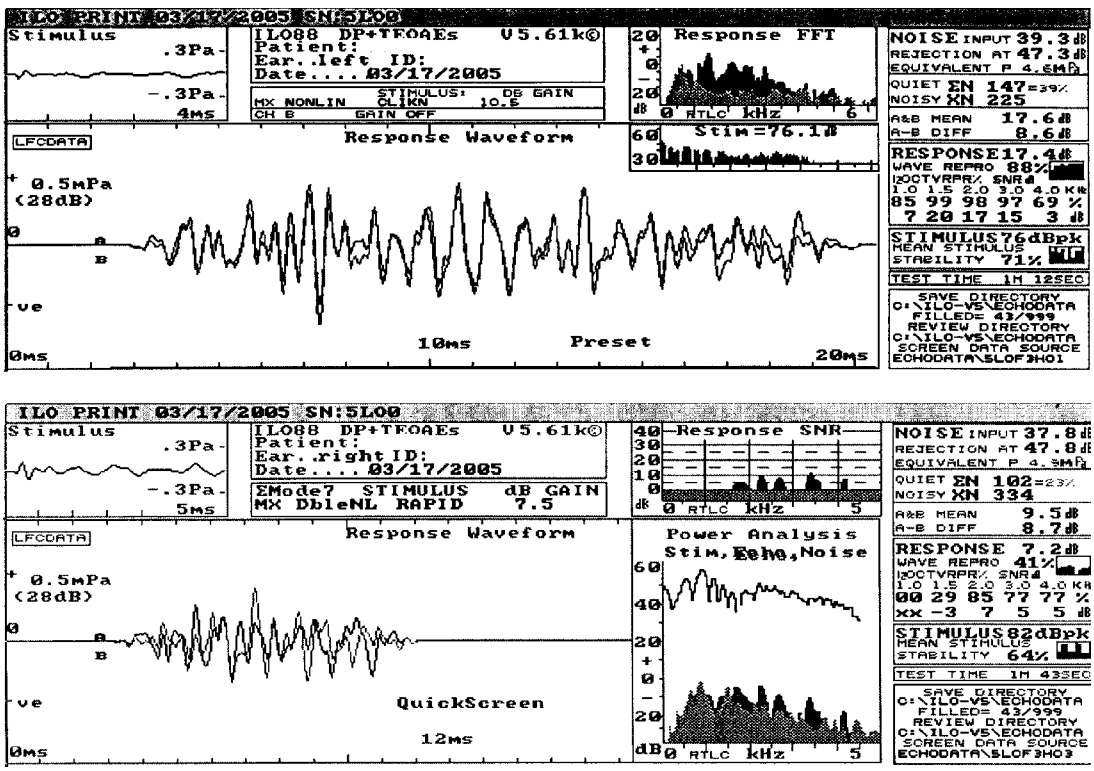


FIGURE 19.18 Case study no. 4. TEOAEs for an infant aged 3 months with middle-ear dysfunction in both ears. Middle-ear effusion was confirmed surgically 1 week after audiologic measures were obtained.

REFERENCES

Abdala C. (1996) Distortion product otoacoustic emission (2f1-f2) amplitude as a function of f2/f1 frequency ratio and primary tone level separation in human adults and neonates. *J Acoust Soc Am.* 100, 3726–3740.

Abdala C, Dhar S. (2012) Maturation and aging of the human cochlea: a view through the DPOAE looking glass. *J Assoc Res Otolaryngol.* 13 (3), 403–421.

Antonelli A, Grandori F. (1986) Long term stability, influence of the head position and modelling considerations for evoked otoacoustic emissions. *Scand Audiol Suppl.* 25, 97–108.

Ashmore JF. (1987) A fast motile response in guinea-pig outer hair cells: the cellular basis of the cochlear amplifier. *J Physiol.* 388, 323–347.

Berlin CI, Hood LJ, Hurley AE, Wen H, Kemp DT. (1995) Binaural noise suppresses linear click-evoked otoacoustic emissions more than ipsilateral or contralateral noise. *Hear Res.* 87, 96–103.

Bilger RC, Matthies ML, Hammel DR, Demorest ME. (1990) Genetic implications of gender differences in the prevalence of spontaneous otoacoustic emissions. *J Speech Hear Res.* 33, 418–432.

Bonfils P, Bertrand Y, Uziel A. (1988) Evoked otoacoustic emissions: normative data and presbycusis. *Audiology.* 27, 27–35.

Brass D, Kemp DT. (1991) Time-domain observation of otoacoustic emissions during constant tone stimulation. *J Acoust Soc Am.* 90, 2415–2427.

Bright KE, Glattke TJ. (1986) Spontaneous otoacoustic emissions in normal ears. In: Collins MJ, Glattke TJ, Harker LA, eds. *Sensorineural Hearing Loss.* Iowa City, IA: University of Iowa Press; pp 201–208.

Brown AM, McDowell B, Forge A. (1989) Acoustic distortion products can be used to monitor the effects of chronic gentamicin treatment. *Hear Res.* 42, 143–156.

Brownell WE, Bader CR, Betrand D, de Ribaupierre Y. (1985) Evoked mechanical responses of isolated cochlear outer hair cells. *Science.* 227, 194–196.

Collet L, Kemp DT, Veuillet E, Duclaux R, Moulin A, Morgon A. (1990) Effect of contralateral auditory stimuli on active cochlear micromechanical properties in human subjects. *Hear Res.* 43, 251–262.

Dallos P, Harris D. (1978) Properties of auditory nerve responses in absence of outer hair cells. *J Neurophysiol.* 41, 365–383.

Davis H. (1983) An active process in cochlear mechanics. *Hear Res.* 9, 79–90.

Dorn PA, Piskorski P, Gorga MP, Neely ST, Keffe DH. (1999) Predicting audiometric status from distortion product otoacoustic emissions using multivariate analyses. *Ear Hear.* 20, 149–163.

Dorn PA, Piskorski P, Keefe DH, Neely ST, Gorga MP. (1998) On the existence of an age/threshold/frequency interaction in distortion product otoacoustic emissions. *J Acoust Soc Am.* 104, 964–971.

Dreisbach LE, Siegel JH. (2001) Distortion-product otoacoustic emissions measured at high frequencies in humans. *J Acoust Soc Am.* 110 (5), 2456–2469.

Ellison JC, Keefe DH. (2005) Audiometric predictions using SFOAE and middle-ear measurements. *Ear Hear.* 26 (5), 487–503.

Fitzgerald TS, Prieve BA. (2005) Detection of hearing loss using 2 f2-f1 and 2f1-f2 distortion product otoacoustic emissions. *J Speech Hear Res.* 48, 1165–1186.

Gaskill SA, Brown AM. (1990) The behavior of the acoustic distortion product, 2f1-f2, from the human ear and its relation to auditory sensitivity. *J Acoust Soc Am.* 88, 821–839.

Goodman SS, Fitzpatrick DF, Ellison JC, Jesteadt W, Keefe DH. (2009) High-frequency click-evoked otoacoustic emissions and behavioral thresholds in humans. *J Acoust Soc Am.* 125 (2), 1014–1032.

Gorga MP, Dierking DM, Johnson TA, Beauchaine KL, Garner CA, Neely ST. (2005) A validation and potential clinical application of multivariate analyses of distortion-product otoacoustic emission data. *Ear Hear.* 26, 593–607.

Gorga MP, Neely ST, Dorn PA. (1999) DPOAE test performance for a priori criteria and for multifrequency audiometric standards. *Ear Hear.* 20, 345–362.

Gorga MP, Neely ST, Dorn PA, Hoover B. (2003) Further efforts to predict pure tone thresholds from distortion product emissions input/output functions. *J Acoust Soc Am.* 113, 3275–3284.

Gorga MP, Neely ST, Ohlrich B, Hoover B, Redner J, Peters J. (1997) From laboratory to clinic: a large scale study of distortion product otoacoustic emissions in ears with normal hearing and ears with hearing loss. *Ear Hear.* 18, 440–455.

Gorga MP, Nelson K, Davis T, Dorn PA, Neely ST. (2000a) Distortion product otoacoustic emission test performance when 2f1-f2 and 2 f2-f1 are used to predict auditory status. *J Acoust Soc Am.* 107, 2128–2135.

Gorga MP, Norton SJ, Sininger YS, Cone-Wesson B, Folsom RC, Vohr BR, et al. (2000b) Identification of neonatal hearing impairment: distortion product otoacoustic emissions during the perinatal period. *Ear Hear.* 21, 400–424.

Green DM, Swets JA. (1966, 1974) *Signal Detection Theory and Psychophysics.* New York: Wiley.

Guinan JJ, Backus BC, Lilaonitkul W, Aharonson V. (2003) Medial olivocochlear efferent reflex in humans: otoacoustic emission (OAE) measurement issues and the advantages of stimulus frequency OAEs. *J Assoc Res Otolaryngol.* 4, 521–540.

Harris FP, Lonsbury-Martin BL, Stagner BB, Coats AC, Martin GK. (1989) Acoustic distortion products in humans: systematic changes in amplitude as a function of f2/f1 ratio. *J Acoust Soc Am.* 85, 220–229.

Harris FP, Probst R. (2002) Otoacoustic emissions and audiometric outcomes. In: Robinette MS, Glattke TJ, eds. *Otoacoustic Emissions.* 2nd ed. New York: Thieme; pp 213–242.

Harrison WA, Norton SJ. (1999) Characteristics of transient evoked otoacoustic emissions in normal-hearing and hearing impaired children. *Ear Hear.* 20, 75–86.

He N-J, Schmiedt RA. (1993) Fine structure of the 2f1-f2 acoustic distortion product: changes with primary level. *J Acoust Soc Am.* 94, 2659–2669.

Heitmann J, Waldmann B, Schnitzler HU, Plinkert PK, Zenner HP. (1998) Suppression of distortion product otoacoustic emissions (DPOAE) near 2f1-f2 removes DP-gram fine structure – evidence for a secondary generator. *J Acoust Soc Am.* 103, 1527–1531.

Hussain DM, Gorga MP, Neely ST, Keefe DH, Peters J. (1998) Transient evoked otoacoustic emissions in patients with normal hearing and in patients with hearing loss. *Ear Hear.* 19, 434–449.

Janssen T, Gehr DD, Klein A, Müller J. (2005) Distortion product otoacoustic emissions for hearing threshold estimation and differentiation between middle-ear and cochlear disorders in neonates. *J Acoust Soc Am.* 117 (5), 2969–2979.

Johnson TA, Neely ST, Garner C, Gorga MP. (2006) Influence of primary-level and primary frequency ratios on human distortion product otoacoustic emissions. *J Acoust Soc Am.* 119, 418–428.

Johnson TA, Neely ST, Kopun JG, Dierking DM, Tan H, Gorga MP. (2010) Clinical test performance of distortion product otoacoustic emissions using new stimulus conditions. *Ear Hear.* 31 (1), 74–83.

Johnson TA, Neely ST, Kopun JG, Dierking DM, Tan H, Converse C., . . . Gorga MP. (2007) Distortion product otoacoustic emissions: cochlear-source contributions and clinical test performance. *J Acoust Soc Am*. 122, 3539–3553.

Joint Committee on Infant Hearing (JCIH). (2007) Year 2007 position statement: principles and guidelines for early hearing detection. *Pediatrics.* 120 (4), 898–921.

Kalluri R, Shera CA. (2007) Near equivalence of human click-evoked and stimulus-frequency otoacoustic emissions. *J Acoust Soc Am.* 121 (4), 2097–2110.

Keefe DH, Abdala C. (2007) Theory of forward and reverse middle-ear transmission applied to otoacoustic emissions in infant and adult ears. *J Acoust Soc Am.* 121 (2), 978–993.

Keefe DH, Goodman SS, Ellison JC, Fitzpatrick DF, Gorga MP. (2011) Detecting high-frequency hearing loss with click-evoked otoacoustic emissions. *J Acoust Soc Am.* 129, 245–261.

Kemp DT. (1978) Stimulated acoustic emissions from within the human auditory system. *J Acoust Soc Am.* 64, 1386–1391.

Kemp DT. (1979) Evidence of mechanical nonlinearity and frequency selective wave amplification in the cochlea. *Arch Otorhinolaryngol.* 224, 37–45.

Kemp DT. (1982) Cochlear echoes: implications for noise-induced hearing loss. In: Hamernik RP, Henderson D, Salvi R, eds. *New Perspectives on Noise-Induced Hearing Loss.* New York: Raven Press; pp 189–207.

Kemp DT. (1986) Otoacoustic emissions, traveling waves and cochlear mechanisms. *Hear Res.* 22, 95–104.

Kemp DT, Brown AM. (1983) A comparison of mechanical nonlinearities in the cochleae of man and gerbil from ear canal measurements. In: Klinke R, Hartmann R, eds. *Hearing – Physiological Bases and Psychophysics.* Berlin: Springer-Verlag; pp 82–88.

Kirby BJ, Kopun JG, Tan H, Neely ST, Gorga MP. (2011) Do "optimal" conditions improve distortion product otoacoustic emission test performance? *Ear Hear.* 32 (2), 230–237.

Knight RD, Kemp DT. (2000) Indications of different distortion product otoacoustic emission mechanisms from a detailed f1, f2 area study. *J Acoust Soc Am.* 107 (1), 457–473.

Kok MR, van Zanten GA, Brocaar MP, Wallenburg HCS. (1993) Click-evoked otoacoustic emissions in 1036 ears of healthy newborns. *Audiology.* 21, 213–224.

Kummer P, Janssen T, Arnold W. (1995) Suppression tuning characteristics of the 2f1-f2 distortion product otoacoustic emission in humans. *J Acoust Soc Am.* 98, 197–210.

Kummer P, Janssen T, Arnold W. (1998) The level and growth behavior of the 2f1-f2 distortion product otoacoustic emission and its relationship to auditory sensitivity in normal hearing and cochlear hearing loss. *J Acoust Soc Am.* 103, 3431–3444.

Kusuki M, Sakashita T, Kubo T, Kyunai K, Ueno K, Hikawa H, et al. (1998) Changes in distortion product emissions from ears with Meniere's disease. *Acta Otolaryngol (Stockh).* 538 (suppl), 78–89.

Lapsley Miller JA, Marshall L. (2007) Otoacoustic emissions as a preclinical measure of noise-induced hearing loss and susceptibility to noise-induced hearing loss. In: Robinette MS, Glattke TJ, eds. *Otoacoustic Emissions: Clinical Applications.* 3rd ed. New York: Thieme; pp 321–341.

Liberman MC, Chesney CP, Kujawa SG. (1997) Effects of selective inner hair cell loss on DPOAE and CAP in carboplatin-treated chinchillas. *Aud Neurosci.* 3 (3), 255–268.

Liberman MC, Dodds LW. (1984) Single-neuron labeling and chronic cochlear pathology. III. Stereocilia damage and alterations of threshold tuning curves. *Hear Res.* 16, 55–74.

Liberman MC, Gao J, He DZ, Wu X, Jia S, Zuo J. (2002) Prestin is required for electromotility of the outer hair cell and for the cochlear amplifier. *Nature.* 419, 300–304.

Liberman MC, Zuo J, Guinan JJ. (2004) Otoacoustic emissions without somatic motility: can sterocilia mechanics drive the mammalian cochlea? *J Acoust Soc Am.* 116 (3), 1649–1655.

Lichtenstien V, Stapells DR. (1996) Frequency-specific identification of hearing loss using transient-evoked otoacoustic emissions to clicks and tones. *Hear Res.* 98, 125–136.

Long GR, Talmadge CL, Lee J. (2008) Measuring distortion product otoacoustic emissions using continuously sweeping primaries. *J Acoustic Soc Am*. 124, 1613–1626.

Lonsbury-Martin BL, Harris FP, Stagner BB, Hawkins MD, Martin GK. (1990) Distortion product emissions in humans. I. Basic properties in normally hearing subjects. *Ann Otol Rhinol Laryngol.* 99, 3–14.

Lonsbury-Martin BL, Martin GK, Whitehead ML. (1997) Distortion product otoacoustic emissions. In: Robinette MS, Glattke TJ, eds. *Otoacoustic Emissions: Clinical Applications.* New York: Thieme; pp 83–109.

Manley GA, Gallo L, Koppl C. (1996) Spontaneous otoacoustic emissions in two *Gecko* species, *Gekko gecko* and *Eublepharis macularius*. *J Acoust Soc Am.* 99, 1588–1603.

Martin GK, Lonsbury-Martin BL, Probst R, Scheinin SA, Coats AC. (1987) Acoustic distortion products in rabbit ear canal. II. Sites of origin revealed by suppression contours and pure-tone exposures. *Hear Res.* 28, 191–208.

Martin GK, Stagner BB, Chung YS, Lonsbury-Martin BL. (2011) Characterizing distortion-product otoacoustic emission components across four species. *J Acoust Soc Am.* 129 (5), 3090–3103.

Mauermann M, Uppenkamp S, van Hengel PWJ, Kollmeier B. (1999) Evidence for the distortion product frequency place as a source of distortion product otoacoustic emission (DPOAE) fine structure in humans. II. Fine structure for different shapes of cochlear hearing loss. *J Acoust Soc Am.* 106 (6), 3484–3491.

Moller AR. (1989) Possible mechanisms for tinnitus. *Hear J.* 42, 68–76.

Neely ST, Johnson TA, Gorga MP. (2005) Distortion-product otoacoustic emission measured with continuously varying stimuli. *J Acoust Soc Am.* 117 (3), 1248–1259.

Nicholson N, Widen JE. (2007) Evoked otoacoustic emissions in the evaluation of children. In: Robinette M, Glattke T, eds. *Otoacoustic Emissions: Clinical Application.* 3rd ed. New York: Thieme; pp 365–399.

Norton SJ, Gorga MP, Widen JE, Folsom RC, Sininger Y, Cone-Wesson B, et al. (2000a) Identification of neonatal hearing impairment: evaluation of transient evoked otoacoustic emission, distortion product otoacoustic emission, and auditory brain stem response test performance. *Ear Hear.* 21, 508–528.

Norton SJ, Gorga MP, Widen JE, Vohr BR, Folsom RC, Sininger YS, et al. (2000b) Identification of neonatal hearing impairment: transient evoked otoacoustic emissions during the perinatal period. *Ear Hear.* 21, 425–442.

Owens JJ, McCoy MJ, Lonesbury-Martin BL, Martin GK. (1992) Influence of otitis media on evoked otoacoustic emission in children. *Semin Hear.* 13, 53–65.

Penner MJ. (1988) Audible and annoying spontaneous otoacoustic emissions. *Arch Otolaryngol Head Neck Surg.* 114, 150–153.

Penner MJ, Burns EM. (1987) The dissociation of SOAE's and tinnitus. *J Speech Hear Res.* 30, 396–403.

Poling GL, Siegel JH, Lee J, Lee J, Dhar S. (2014) Characteristics of the 2f1-f2 distortion product otoacoustic emission in a normal hearing population. *J Acoust Soc Am.* 135 (1), 287–299.

Prieve BA, Calandruccio L, Fitzgerald T, Mazevski A, Georgantas LM. (2008) Changes in transient-evoked otoacoustic emission levels with negative tympanometric peak pressure in infants and toddlers. *Ear Hear.* 29 (4), 533–542.

Prieve BA, Fitzgerald TS, Schulte LE. (1997a) Basic characteristics of click-evoked otoacoustic emissions in infants and children. *J Acoust Soc Am.* 102, 2860–2280.

Prieve BA, Fitzgerald TS, Schulte LE, Kemp DT. (1997b) Basic characteristics of distortion product otoacoustic emissions in infants and children. *J Acoust Soc Am.* 102, 2871–2879.

Prieve BA, Gorga MP, Neely ST. (1996) Click- and tone-burst-evoked otoacoustic emissions in normal-hearing and hearing-impaired ears. *J Acoust Soc Am.* 99, 3077–3086.

Prieve BA, Gorga MP, Schmidt A, Neely ST, Peters J, Schultes L, et al. (1993) Analysis of transient-evoked otoacoustic emissions in normal hearing and hearing impaired ears. *J Acoust Soc Am.* 93, 3308–3319.

Probst R, Coats AC, Martin GK, Lonsbury-Martin BL. (1986) Spontaneous, click-, and toneburst-evoked otoacoustic emissions from normal ears. *Hear Res.* 21, 261–275.

Rao A, Long GR. (2011) Effects of aspirin on distortion product fine structure: interpreted by the two-source model for distortion product otoacoustic emissions generation. *J Acoust Soc Am.* 129 (2), 792–800.

Rebillard G, Lavigne-Rebillard M. (1992) Effect of reversible hypoxia on the compared time courses of endocochlear potential and 2f1-f2 distortion products. *Hear Res.* 62, 142–148.

Reuven M, Neely ST, Kopun JG, Rasetshwane DM, Allen JB, Tan H, et al. (2013) Effect of calibration method on distortion-product otoacoustic emission measurements at and around 4 kHz. *Ear Hear.* 34 (6), 779–788.

Rhode WW. (1971) Observations of the vibrations of the basilar membrane in squirrel monkeys using the Mossbauer technique. *J Acoust Soc Am.* 49, 1218–1231.

Ricci AJ, Crawford AC, Fettiplace R. (2000) Active hair bundle motion linked to fast transducer adaptation in auditory hair cells. *J Neurosci.* 20 (19), 7131–7142.

Robinette MS. (1992) Clinical observations with transient evoked otoacoustic emissions with adults. *Semin Hear.* 13, 23–36.

Robinette MS, Cevette MJ, Webb TM. (2002) Otoacoustic emissions in differential diagnosis. In: Robinette MS, Glattke TJ, eds. *Otoacoustic Emissions.* 2nd ed. New York: Thieme; pp 297–324.

Sakashita T, Takeshi K, Kusuki M, Kynai K, Ueno K, Hikawa C, et al. (1998) Patterns of change in growth function of distortion product otoacoustic emissions in Meniere's disease. *Acta Otolaryngol (Stockh).* 538 (suppl), 70–77.

Schairer KS, Keefe DH. (2005) Simultaneous recording of stimulus-frequency and distortion-product otoacoustic emission input-output functions in human ears. *J Acoust Soc Am.* 117, 818–832.

Schmiedt RA. (1986) Acoustic distortion in the ear canal. I. Cubic difference tones: effects of acute noise injury. *J Acoust Soc Am.* 79, 1481–1490.

Schmiedt RA, Adams JC. (1981) Stimulated acoustic emissions in the ear canal of the gerbil. *Hear Res.* 5, 295–305.

Shera CA. (2004) Mechanisms of mammalian otoacoustic emission and their implications for the clinical utility of otoacoustic emissions. *Ear Hear.* 25 (2), 86–97.

Shera CA, Guinan JJ. (1999) Evoked otoacoustic emissions arise by two fundamentally different mechanisms: a taxonomy for mammalian OAEs. *J Acoust Soc Am.* 105, 782–798.

Shera CA, Guinan JJ. (2003) Stimulus-frequency-emission group delay: a test of coherent reflection filtering and a window on cochlear tuning. *J Acoust Soc Am.* 113, 2762–2772.

Siegel JH, Kim DO. (1982) Efferent control of cochlear mechanics? Olivocochlear bundle stimulation affects cochlear biomechanical nonlinearity. *Hear Res.* 6, 171–182.

Sininger YS, Cone-Wesson B, Folsom RC, Gorga MP, Vohr BR, Widen JE, et al. (2000) Identification of neonatal hearing impairment: auditory brain stem responses in the perinatal period. *Ear Hear.* 21, 383–399.

Spivak L, Dalzell L, Berg A, Bradley M, Cacace A, Campbell D, et al. (2000) The New York State Universal Newborn Hearing Screening Demonstration Project: inpatient outcome measures. *Ear Hear.* 21 (2), 92–103.

Stavroulaki P, Vossinakis IC, Dinopoulou D, Doudounakis S, Adamopoulos G, Apostolopoulos N. (2002) Otoacoustic emissions for monitoring aminoglycoside-induced ototoxicity in children with cystic fibrosis. *Arch Otolaryngol Head Neck Surg.* 128 (2), 150–155.

Stover L, Gorga MP, Neely ST, Montoya D. (1996) Toward optimizing the clinical utility of distortion product otoacoustic emission measurements. *J Acoust Soc Am.* 100, 956–967.

Stover L, Neely ST, Gorga MP. (1999) Cochlear generation of intermodulation distortion revealed by DPOAE frequency functions in normal and impaired ears. *J Acoust Soc Am.* 106 (5), 2669–2678.

Stover L, Norton SJ. (1993) The effects of aging on otoacoustic emissions. *J Acoust Soc Am.* 94, 2670–2681.

Strickland AE, Burns EM, Tubis A. (1985) Incidence of spontaneous otoacoustic emissions in infants and children. *J Acoust Soc Am.* 78, 931–935.

Talmadge CL, Long GR, Murphy WJ, Tubis A. (1993) New off-line method for detecting spontaneous otoacoustic emissions in human subjects. *Hear Res.* 71, 170–182.

Talmadge CL, Long GR, Tubis A, Dhar S. (1999) Experimental confirmation of the two-source interference model for the fine structure of distortion product otoacoustic emissions. *J Acoust Soc Am.* 105 (1), 275–292.

Vinck BM, Van Cauwenberge PB, Corthals P, De Vel E. (1998) Multi-variant analysis of otoacoustic emissions and estimation of hearing thresholds: transient evoked otoacoustic emissions. *Audiology.* 37, 315–334.

Vohr BR, Carty LM, Moore PE, Letourneau K. (1998) The Rhode Island Hearing Assessment Program: experience with statewide hearing screening. *J Pediatr.* 133, 353–359.

Warr WB, Guinan JJ, White JS. (1986) Organization of the efferent fibers: the lateral and medial olivocochlear systems. In: Altschuler RA, Hoffman DW, Bobbin RP, eds. *Neurobiology of Hearing: The Cochlea.* New York: Raven Press; pp 333–348.

Weinstein MC, Fineberg HV. (1980) *Clinical Decision Analysis.* Philadelphia, PA: Saunders.

Widen JE, Folsom RC, Cone-Wesson B, Carty L, Dunnell JJ, Koebsell K, et al. (2000) Identification of neonatal hearing impairment: hearing status at 8 to 12 months corrected age using a visual reinforcement audiometry protocol. *Ear Hear.* 21, 471–487.

Zheng J, Shen W, He DZ, Long KB, Madison LD, Dallos P. (2000) Prestin is the motor protein of cochlear outer hair cells. *Nature (London).* 405, 149–155.

Zwicker E. (1983) Delayed evoked otoacoustic emissions and their suppression by Gaussian-shaped pressure impulses. *Hear Res.* 11, 359–371.

CHAPTER 20

Clinical Neurophysiology of the Vestibular System

Erin G. Piker and Douglas B. Garrison

INTRODUCTION TO THE VESTIBULAR SYSTEM

The vestibular system serves the basic function of translating movement of the head into an electrical signal. The vestibular system detects head movement and responds with compensatory reflexive eye movements and postural adjustments that allow us to maintain clear vision and prevent us from falling. Spatial orientation is maintained because of the vestibular system's complex role of driving the reflexes that stabilize our vision and balance. Unlike other sensory systems (e.g., auditory, visual), most individuals are unaware of the vestibular system during routine activities, that is, until the system ceases to function normally. A sudden loss of function from one vestibular end organ can result in a profound disability because of vertigo, imbalance, nausea, and vomiting.

Vestibular function is difficult to assess directly in humans. Current clinical tests of vestibular function evaluate secondary motor responses (i.e., reflexes) used to maintain eye position or postural control during movement. Because there is no direct sensory potential clinically available, the accurate interpretation of current vestibular diagnostic tests requires knowledge and understanding of the anatomy and physiology of the peripheral vestibular system and its central connections. There are entire volumes of books on this complex subject, and the reader should refer to those for a more comprehensive description (Baloh and Kerber, 2011). The purpose of this chapter is to provide a basic background in the anatomy and clinical physiology needed to understand and assess the vestibular system.

OVERVIEW OF VESTIBULAR ANATOMY AND PHYSIOLOGY

Peripheral Vestibular System

The vestibular end organs are housed in a series of tunnels located in the petrous portion of the temporal bone referred to as the *bony labyrinth* (Figure 20.1). The bony labyrinth is a carved out portion of the temporal bone that creates space for the membranous labyrinth. The membranous labyrinth is filled with *endolymph* fluid (higher concentration of potassium and lower in sodium) and is suspended within the bony labyrinth by *perilymph* (lower concentration of potassium and higher in sodium) and supportive connective tissue. Embedded within the membranous labyrinth are the five vestibular sensory organs: three *semicircular canals* (SCCs) and two *otolith end organs* (Figure 20.2).

Semicircular Canals

The SCCs convert angular acceleration and deceleration into electrical signals that are transmitted through the vestibular nerve to the vestibular nucleus. There are three SCCs in each inner ear, one horizontal and two vertical, extending from the utricle (Figure 20.2). They are known as the *lateral/horizontal, anterior/superior,* and the *posterior/inferior* SCCs. Each SCC responds best to angular motion in one plane and they are roughly orthogonal to each other such that they can sense any rotation in three-dimensional space. Additionally, the canals in the right and left inner ears are arranged in complementary coplanar plains. The lateral SCCs from the right and left inner ears lie in the same plane, whereas the plane of each anterior canal is roughly coplanar to that of the posterior canal of the opposite side (Figure 20.3C).

Each SCC is filled with endolymph and forms a closed ring with a shared cavity in the utricular sac. The lateral SCC communicates at both ends with the utricle. The vertical canals (anterior and posterior) communicate with the utricle at one end and join together at the other end. Each SCC is dilated at one end closest to the utricle forming the ampulla. The ampulla is the location of the *crista*—the sense organ of balance.

Otolith Organs

Whereas SCCs respond to angular acceleration in specific directions, the hair cells in the *saccule* and *utricle* respond to linear acceleration and deceleration (i.e., changing velocity, not constant velocity as with a train). The saccule is oriented vertically and responds to linear vertical (up/down) translation whereas the utricle senses tilt and linear

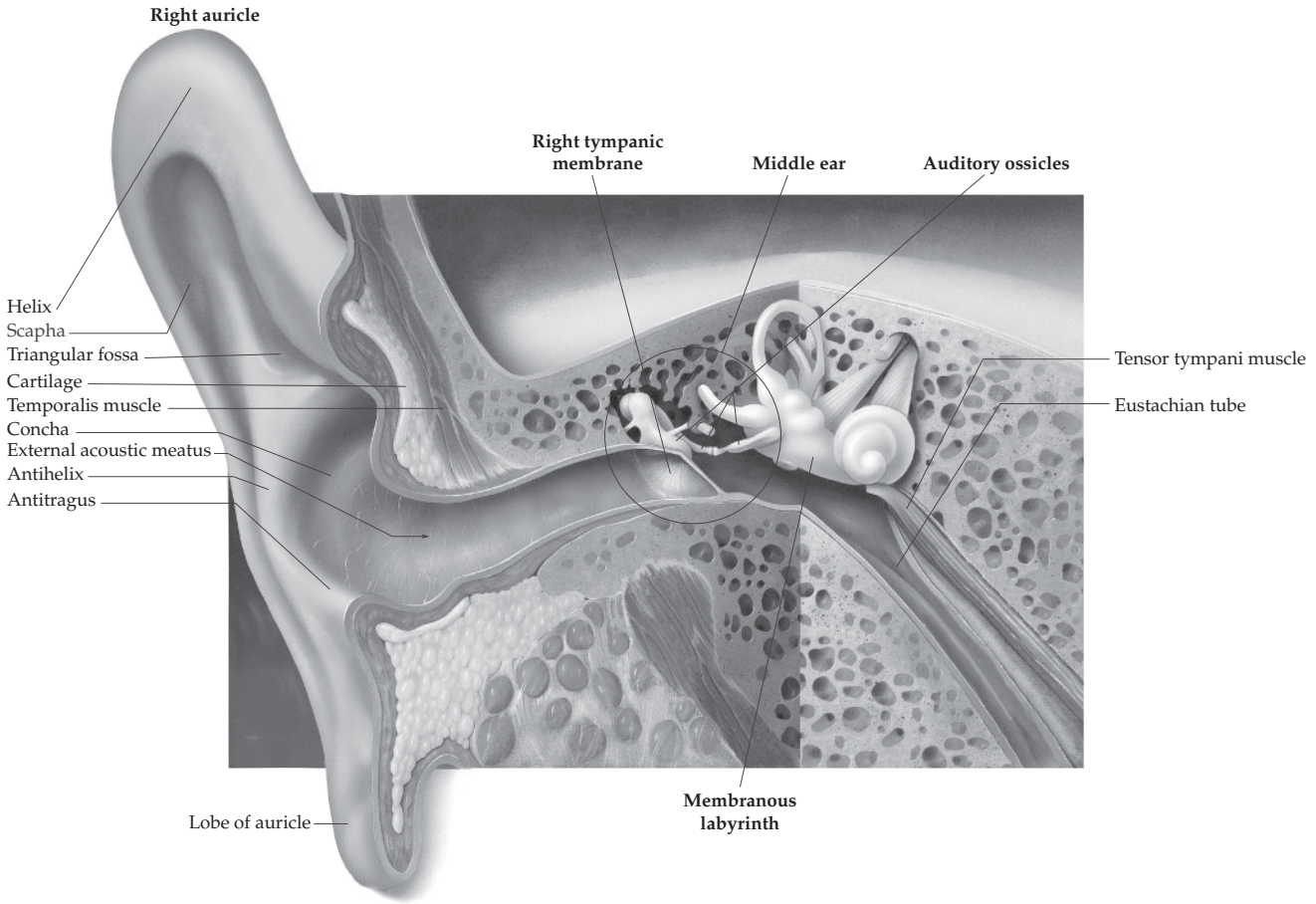


FIGURE 20.1 Outer, middle, and inner ear, including bony labyrinth. From American Chart Company.

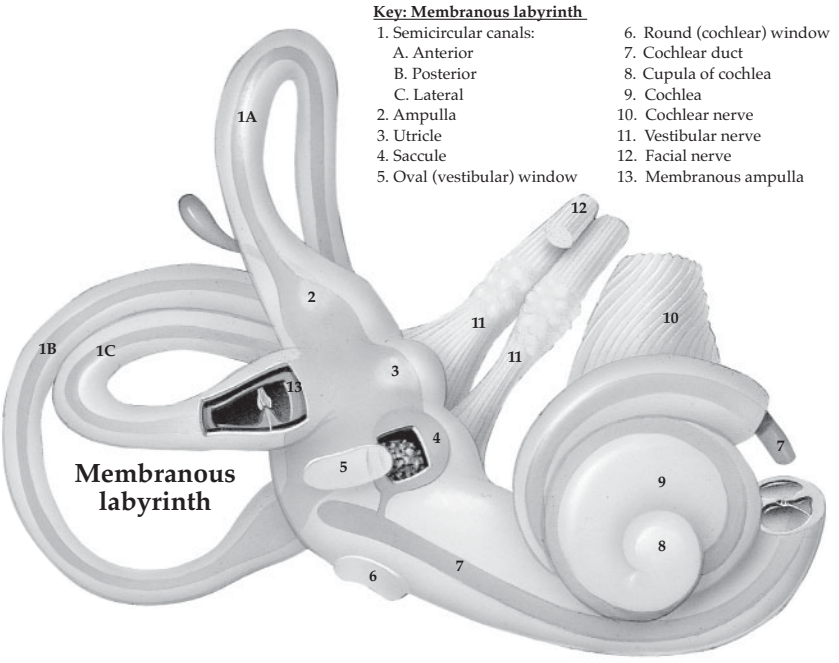


FIGURE 20.2 Membranous labyrinth. From American Chart Company.

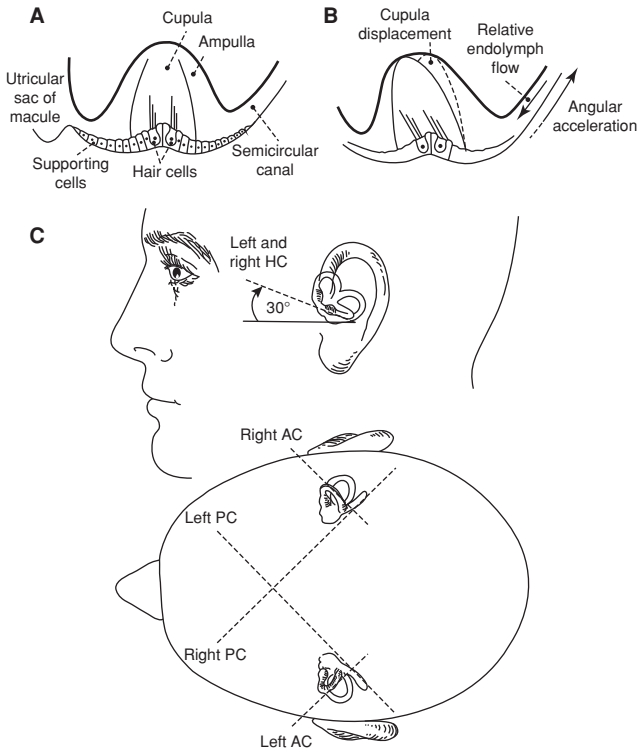


FIGURE 20.3 The crista. **A:** Structural organization of the crista of the lateral SCC. **B:** Mechanism of hair cell activation with angular acceleration. **C:** Orientation of the three SCCs within the head. AC, anterior SCC; HC, horizontal SCC; PC, posterior SCC. [Baloh RW, Honrubia V. (2001) *Clinical Neurophysiology of the Vestibular System*. New York, NY: Oxford University Press; Figure 2.6, p. 30, by permission of Oxford University Press, USA.]

horizontal translations (side/side, front/back). Figure 20.4 shows a three-dimensional representation of otolith orientation. The utricle is located above the saccule in the elliptical recess and is approximately parallel to the plane of the lateral SCC. The saccule is located on the medial wall of the vestibule and is approximately perpendicular to the plane of the utricle (Baloh and Kerber, 2011). The saccule and utricle are filled with endolymph and each house a sensory organ called the *macula.*

Structure of Hair Cells

Both the SCCs and the otolith organs utilize specialized hair cells much like the ones in the auditory system. It is the hair cells that transduce mechanical force into nerve action potentials. There are two types of hair cells in mammalian vestibular systems (Figure 20.5). *Type I* cells are globular or flask shaped (i.e., the cell body is narrower at the apex and wider at the base) with a large nerve terminal surrounding the base. *Type II* cells are cylindrical with multiple nerve terminals at their base, including direct contact with efferent nerve endings. On the apical end of each hair cells is a *cuticular plate,* from which *stereocilia* protrude.

Each hair cell possesses several shorter stereocilia and a single tall *kinocilium* at one margin of the cell (Figure 20.5). These ciliated sensory hair cells contain vesicles that possess neurotransmitters. The tips of the cilia are connected by *tip links* that open and close mechanosensory channels (Vollrath et al., 2007). Deflection of the cilia toward the kinocilium opens the mechanosensory channels at the tips causing an influx of potassium effectively depolarizing (i.e., exciting) the cell (Figure 20.6B; Hudspeth, 2005). This opens voltage-gated calcium channels releasing neurotransmitters

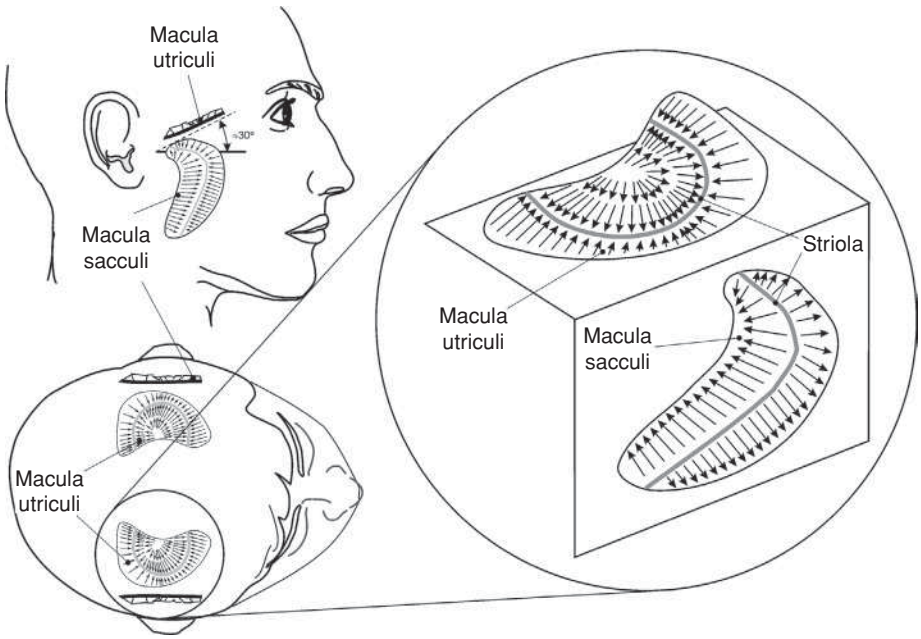


FIGURE 20.4 Orientation of the utricle and saccule. The direction of the excitatory responses of the hair cells is indicated by the *arrows.* [Katz, 6th ed. Figure 19.10, p. 440].

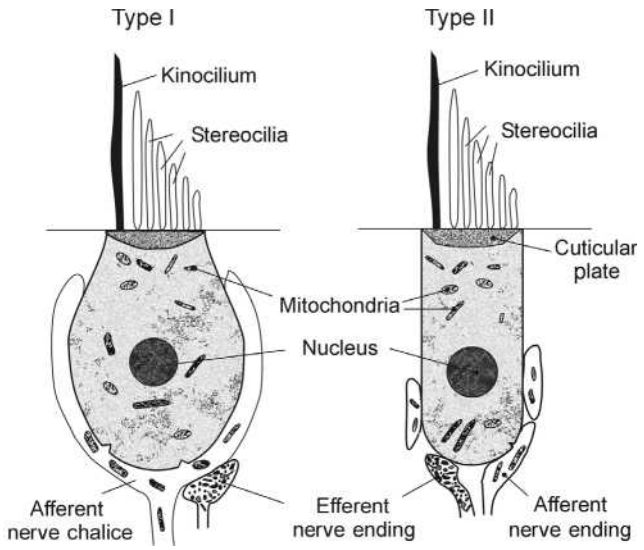


FIGURE 20.5 Type I and Type II hair cells in the vestibular system [Katz, 6th ed. Figure 19.5, p. 436].

from the hair cell and thus increasing the firing rate of the vestibular nerve associated with the hair cell. Deflection of cilia away from the kinocilium results in hyperpolarization (i.e., inhibition) of the cell causing a decrease in neural firing (Figure 20.6C). The hair cells release neurotransmitters even when they are not stimulated (Figure 20.6A; Baloh and Honrubia, 1995). In other words, the axons in the vestibular nerve are always firing at a baseline rate but can be adjusted to fire more or less depending on the direction of head movement.

The Cristae and the Macules

Within each SCC is a dilation called the *ampulla.* Each ampulla contains a *crista,* the receptor organs of the SCCs. The upper surface of the crista contains ciliated sensory hair cells that are embedded in a gelatinous material called the *cupula* (Figure 20.3A; Dohlman, 1971). The cupula is akin to a swing-door device that extends to the top of the ampulla and separates the endolymph of the SCC from the

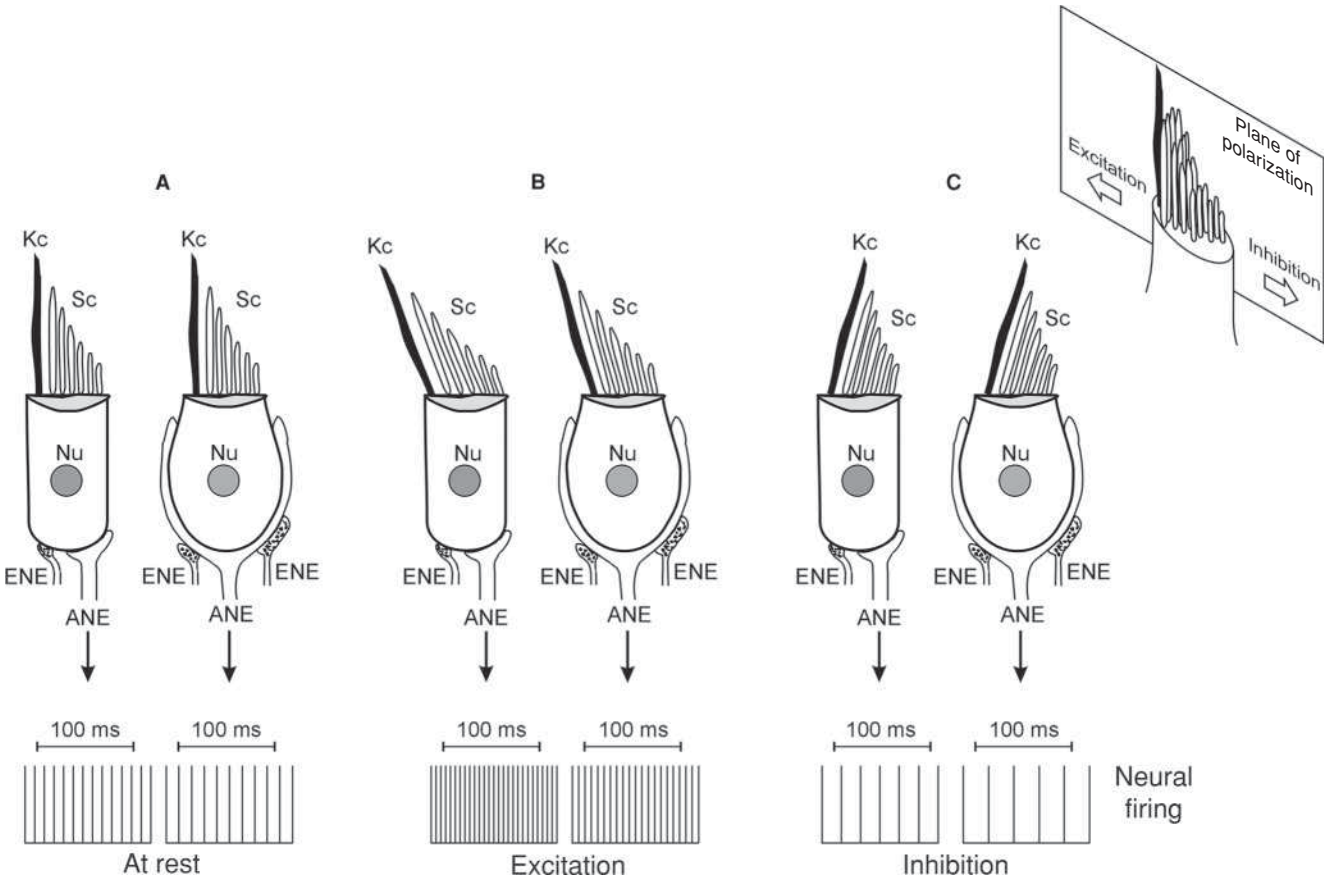


FIGURE 20.6 Firing rate of the primary afferent neurons as a function of stereocilia displacement. **A:** Neurons at rest. **B:** Depolarization as a result of bending toward the kinocilium (excitatory response). **C:** Hyperpolarization as a result of bending away from the kinocilium (inhibitory response). Kc, kinocilium; Sc, stereocilia; ANE, afferent nerve ending; ENE, efferent nerve ending; Nu, nucleus [Katz, 6th ed. Figure 19.6, p. 437].

endolymph of the utricle. The hair cells of the crista, which project into the cupula, are oriented with their kinocilia in the same direction. Deflection of the stereocilia toward the kinocilium results in an increase in the firing rate of the vestibular fiber associated with the hair cell, whereas deflection away from the kinocilium results in a decrease in the firing rate of the vestibular fiber. In the lateral SCC the kinocilia are directed toward the utricular side of the ampulla (as shown in Figure 20.3A), so the firing rate increases when endolymph moves toward the utricle and ampulla (*ampullopetal;* Figure 20.3B). In contrast, the kinocilia of the posterior and superior SCCs are directed toward the canal side of the ampulla so that the firing rate increases when endolymph flows away from the utricle and ampulla (*ampullofugal*).

The cupula has the same specific gravity as the surrounding endolymph (Money et al., 1971). Because of this, the cupula is not displaced by gravitational force. The forces that are associated with angular head acceleration displace the cupula and bend the hair cells of the crista within each of the SCCs. Head acceleration in the plane of a SCC naturally causes movement of the bony labyrinth in that plane. Because of inertia, the endolymph within the membranous labyrinth of that canal lags behind structures within the ampulla so that the endolymph moves in the opposition direction relative to the head (Figure 20.3B). Inside the ampulla, pressure exerted by the endolymph deflects the cupula which results in a shearing of the stereocilia and either excites or inhibits the hair cells. Stimulation of the SCC produces eye movements in the plane of that canal.

The utricle and saccule are sac-like structures that contain a patch of sensory hair cells called the *macula.* The hair cells in the macula of the otolith organs, which are similar to those in the cristae, are embedded in the *otolith membrane,* a gelatinous structure that contains a large number of hexagonal prisms of calcium carbonate called *otoconia* (Figure 20.7A; Lundberg et al., 2006). The distribution of Type I and II hair cells in the macula are such that polarization is along a line that bisects the end organ. This bisected, curved area is called the *striola.* Hair cells and their stereocilia are oriented in opposite directions on each side of the striola such that the kinocilium of the hair cells within each macula are oriented in all possible directions (Figure 20.7C).

Unlike the cupula in the SCCs, the density of the otoconia is much greater than the surrounding endolymph (Money et al., 1971). Because of this, the otolith membrane is displaced by the force of gravity or linear acceleration (Figure 20.7B). Such displacement bends the stereocilia and, depending on the polarity of the cell, either causes an increase or a decrease in the number of impulses in the associated vestibular nerve fiber. As shown in Figure 20.7C, the striola of the utricle divides the macula into a medial 2/3 and a lateral 1/3. Hair cells on either side are polarized so that stereocilia deflection toward the striola is excitatory. The striola of the saccule divides the macula roughly in half and the stereocilia are oppositely polarized (i.e., away from the striola; Figure 20.7C).

Cranial Nerve VIII

The vestibular division of the vestibulocochlear nerve (cranial nerve VIII) arises from Scarpa's ganglion and consists of bipolar neurons organized into inferior and superior branches (Figure 20.2). These fibers travel from the membranous labyrinth through the internal auditory canal and terminate in the vestibular nuclei (VN) at the pontomedullary junction (Baloh and Honrubia, 1995). The vestibular nerve is highly organized. The inferior portion of Scarpa's ganglion comprises the nerve fibers from the crista of the posterior SCC and the macula of the saccule (Figure 20.2). The superior portion comprises the nerve fibers from the anterior SCC, lateral SCC, and utricle (Figure 20.2). The nerve fibers are organized into small bundles and travel together allowing the organization of the sensory epithelium to be retained in the vestibular nerve much like the tonotopic organization seen throughout the auditory system.

The vestibular nerve consists of ~15,000 single nerve fibers that discharge spontaneously at rates from 10 to 100 spikes/second (Barin and Durrant, 2000). This means, at any moment, there can be >1,000,000 spikes/second passing through the central vestibular system. There is a range of spontaneous firing rates and the primary vestibular afferents are classified as having a regular or irregular spontaneous discharge rate (Goldberg et al., 1987).

Blood Supply

The blood supply to the membranous labyrinth is shown in Figure 20.8. The *labyrinthine artery,* the main source of blood supply to the membranous labyrinth, originates from the *anteroinferior cerebellar artery* (AICA; Lee et al., 2004). After entering the inner ear, the labyrinthine artery divides into two main branches: the *common cochlear artery* and the *anterior vestibular artery.* The cochlear artery also divides into two branches: the *posterior vestibular artery* (blood supply to the inferior part of the saccule and the ampulla of the posterior SCC) and the *main cochlear artery* (blood supply to the cochlear structures). The anterior vestibular artery, the other branch of the labyrinthine artery, is the blood supply to the utricle, superior portion of the saccule, and the ampulla of the anterior and lateral SCCs. The vestibular system is susceptible to ischemic events because the arteries lack collateral connections with other major arterial branches. It has been noted that a 15-second interruption in blood supply can cause

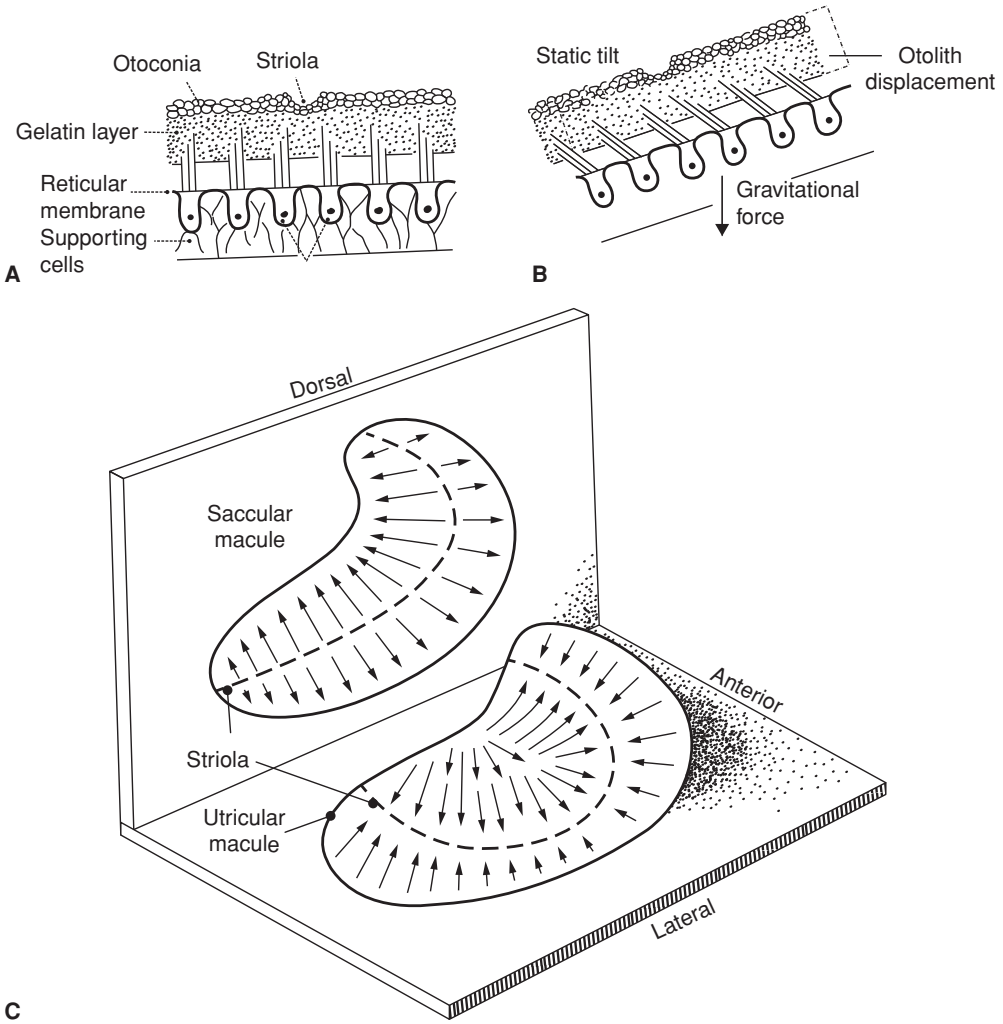


FIGURE 20.7 Macule. **A:** Structural organization of the utricular macula. **B:** Mechanisms of hair cell activation with static tilt. **C:** Orientation of saccular and utricular macules. *Arrows* indicate the direction that the kinocilia point toward. [**A** and **B:** Kiernan JA. (2004) *Barr's The Human Nervous System.* Philadelphia, PA: Lippincott Williams & Wilkins. **C:** Baloh RW, Honrubia V. (2001) *Clinical Neurophysiology of the Vestibular System.* New York, NY: Oxford University Press; Figure 2.9, p. 34, by permission of Oxford University Press, USA; Adapted with permission from Barber HO, Stockwell CW. (1976) *Manual of Electronystagmography.* St. Louis, MO, CV Mosby.]

impairment of the vestibular sensory receptors, and different sensory receptors can be selectively damaged (Kusakari et al., 1981).

Central Vestibular System

Vestibular Nuclei

There are four main *VN* within the brainstem: superior, lateral, medial, and inferior/descending (Figure 20.9; Straka et al., 2005). The VN receive primary input from the vestibular portion of cranial nerve VIII, but they are innervated by multiple nerve fibers, not just direct vestibular connections. For example, the VN receive visual and proprioceptive afferent information in addition to the primary vestibular signals (Angelaki and Cullen, 2008). Outputs from the VN project to the contralateral VN, ipsilateral and contralateral abducens, trochlear nuclei, and oculomotor nuclei and to the motor spinal cord via the medial vestibulospinal tract (MVST), lateral vestibulospinal tract (LVST), and the reticulospinal pathway (Figure 20.9; Baloh and Kerber, 2011). The VN also send projections to the cerebral cortex for perception of motion and to cerebellar pathways to coordinate compensatory eye and head movements and postural changes.

Cerebellum

Afferents from the medial and inferior VN project to the flocculus, nodulus, uvula, and fastigial nucleus of the

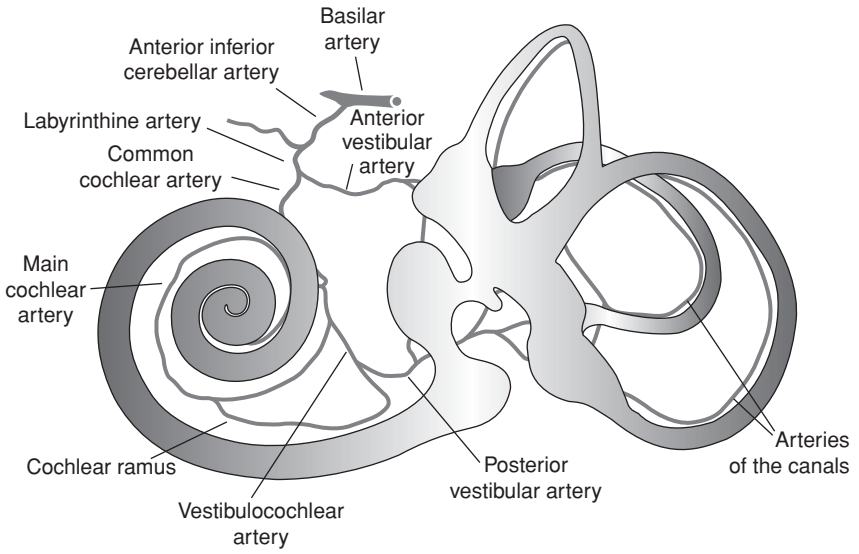


FIGURE 20.8 Blood supply to the membranous labyrinth. (From Schuknecht HE. (1974) *Pathology of the Ear*. Cambridge, MA: Harvard University Press; Figure 2.63, p. 62.)

cerebellum (Baloh and Kerber, 2011). These areas of the cerebellum are collectively known as the *vestibulocerebellum*. The efferent cerebellovestibular pathway extends from the vermis, flocculus, and fastigial nuclei and terminates on the lateral VN. Stimulation of this pathway results in an inhibition of VN activity (Ito, 1993). This pathway is implicated in vestibulo-ocular reflex (VOR) suppression and central vestibular compensation.

ROLE OF THE VESTIBULAR SYSTEM

The vestibular end organs sense head rotation and linear accelerations and send that information to secondary neurons in the VN. Secondary vestibular neuron signals diverge to many areas of the central nervous system and serve as

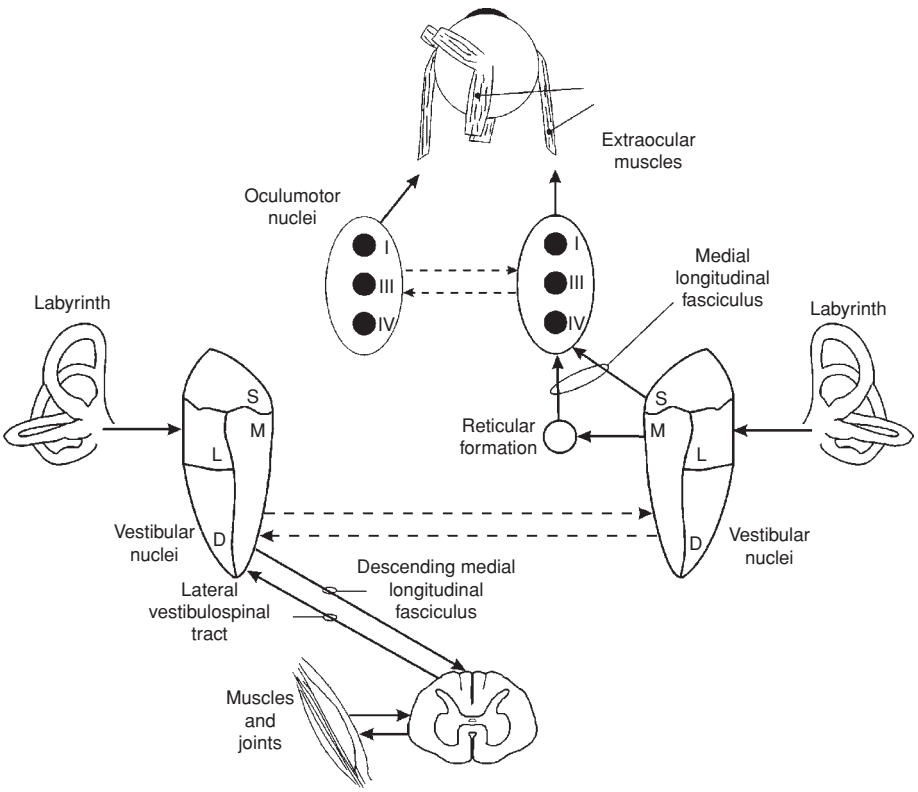


FIGURE 20.9 VCR and VOR pathways. S, superior vestibular nucleus; L, lateral vestibular nucleus; M, medial vestibular nucleus; D, descending vestibular nucleus. (From Canalis RF, Lambert PR. (2000) *The Ear: Comprehensive Otology*. Philadelphia, PA Lippincott Williams & Wilkins; Figure 12, p. 125.)

a key relay in at least two important vestibular reflexes: the *VOR* and the *vestibulospinal reflex* (VSR; Figure 20.9). Neurons encode head movement and form synapses with ocular motor nuclei that generate patterns of extraocular muscle (EOM) contraction and relaxation required for driving the VOR, which stabilizes gaze. These neurons also synapse with spinal motor neurons that drive the VSR, which is used to stabilize posture and adjust gait. Additional central nervous system areas also receive this input including (1) autonomic centers, which receive input regarding posture with respect to gravity resulting in an adjustment of hemodynamic reflexes to maintain cerebral perfusion; (2) cerebellum, which is required for coordination and adaptation of vestibular reflexes when abnormal changes occur such as injury to the vestibular system; and (3) cerebral cortex, which is used to mediate perception of movement and orientation. Thus, the 10 vestibular sensory organs (five on each side) provide input to multiple areas of the central nervous system resulting in an intricate network used to maintain balance.

Extraocular Muscles and Ocular Motility

To understand the VOR, it is important to understand the anatomy of the six EOMs that control eye movement and the connections from the peripheral vestibular system to the ocular motor neurons (Figure 20.10). The lateral rectus and medial rectus control horizontal movement; the superior rectus and inferior rectus control vertical movement; and the superior oblique and inferior oblique control torsional movement (Table 20.1). These EOMs are often described by paired agonist and antagonist counterparts where the contraction of one muscle occurs concomitantly with the relaxation of its muscle pair (e.g., contraction of the lateral rectus results in relaxation of the medial rectus). The orientation of the SCCs matches the plane of movement controlled by one pair of eye muscles. For example, as shown in Table 20.2, the lateral canal has an excitatory connection with the ipsilateral medial rectus and contralateral lateral rectus as well as inhibitory connections with the contralateral medial rectus and ipsilateral lateral rectus. Figure 20.11 shows an illustration of the excitatory and inhibitory connections between the SCCs and the EOMs.

Three ocular motor nuclei innervate the eye muscles. Signals to the lateral rectus are routed through the abducens (sixth) nucleus (Figures 20.11B and 11E). Signals to the superior oblique are routed through the trochlear (fourth) nucleus (Figures 20.11C and 11D). The remaining EOMs are innervated by the oculomotor (third) nucleus (Figure 20.11). The three ocular motor nuclei receive signals from the VN via the medial longitudinal fasciculus (MLF), ascending track of Dieters, or the reticular formation.

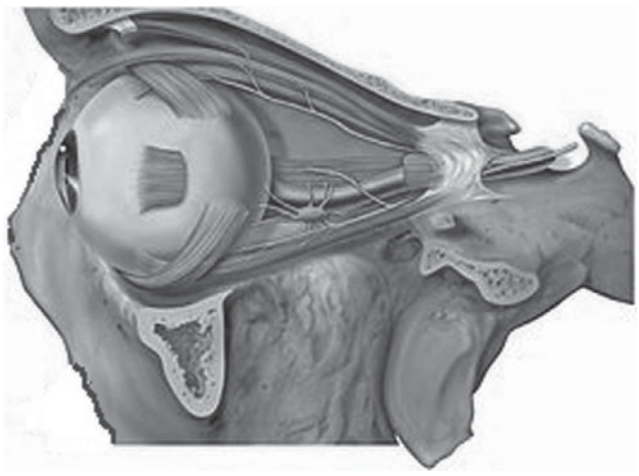


FIGURE 20.10 The six extraocular muscles of the eye. [Courtesy of Patrick Lynch, Yale University School of Medicine.]

TABLE 20.1

Extraocular Muscle Pairs and their Plane of Action

Muscle Pairs	Primary Plane of Action
Lateral rectus and medial rectus	Horizontal
Superior rectus and inferior rectus	Vertical
Superior oblique and inferior oblique	Torsional

TABLE 20.2

Semicircular Canals and their Connections to the EOMs

Semicircular Canals	Excited Extraocular Muscles	Inhibited Extraocular Muscles
Horizontal	Ipsilateral medial rectus	Contralateral medial rectus
	Contralateral lateral rectus	Ipsilateral lateral rectus
Anterior	Ipsilateral superior rectus	Ipsilateral inferior rectus
	Contralateral inferior oblique	Contralateral superior oblique
Posterior	Ipsilateral superior oblique	Ipsilateral inferior oblique
	Contralateral inferior rectus	Contralateral superior rectus

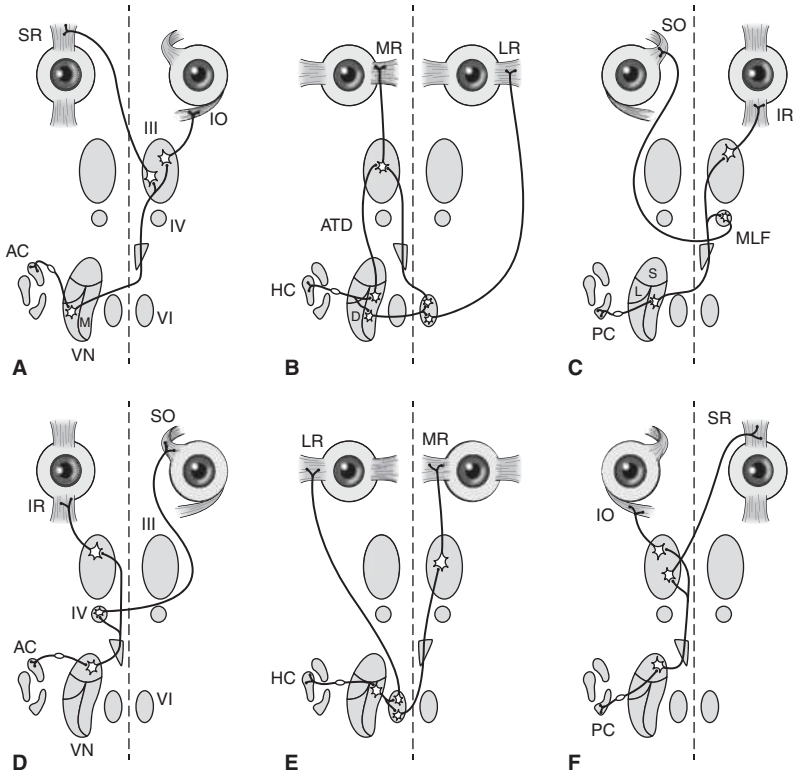


FIGURE 20.11 Excitatory (**A–C**) and inhibitory (**D–F**) pathways between the individual SCCs and eye muscles. AC, anterior SCC; ATD, ascending tract of Dieters; HC, horizontal SCC; III, oculomotor nucleus; IO, inferior oblique; IR, inferior rectus; IV, trochlear nucleus; L, lateral vestibular nucleus; M, medial vestibular nucleus; D, descending vestibular nucleus; LR, lateral rectus; MLF, medial longitudinal fasciculus; MR, medial rectus; PC, posterior SCC; S, superior vestibular nucleus; SO, superior oblique; SR, superior rectus; VI, abducens nucleus; VN, vestibular nuclei. (Baloh RW, Honrubia V. (2001) *Clinical Neurophysiology of the Vestibular System*. New York, NY: Oxford University Press; Figure 3.6, p. 64, by permission of Oxford University Press, USA.)

Oculomotor Control Systems That Interact with the VOR

The purpose of the VOR is to stabilize gaze during head movement. Similarly, the primary purpose of the ocular motor system is to stabilize gaze by moving the eyes to view an item of interest. Objects are viewed most clearly at the center of the retina, known as the *fovea,* because of the higher concentration of sensory cells in that area. The center of the fovea, the foveola, provides even greater resolution. The interested reader is referred to Leigh and Zee (2006) for a more detailed description of the anatomy and physiology of the ocular motor system.

Saccades are rapid eye movements whose function is to place the image of interest on the fovea. Saccades are different from other eye movements because they move at very high velocity and vision is impaired during the movement. Generation of horizontal saccades involves the paramedian pontine reticular formation (PPRF) as well as multiple sites within the central nervous system. Impairment of saccadic eye movements often localizes to lesions of the brainstem or cerebellum (Leigh and Zee, 2006).

The saccadic system interacts with the vestibular system during head movement. When the head turns, the VOR initiates a slow eye movement (i.e., the "slow phase" of nystagmus) in the opposite direction of head movement. The saccadic system is responsible for bringing the eyes back to midline (i.e., the "fast phase" of nystagmus) after the vestibular system has driven the eyes from primary position.

Smooth pursuit eye movements are intended to keep an object of interest on the fovea by matching target velocity with eye velocity. The smooth pursuit system generally requires a moving target to be activated, but is unable to reach high velocities so the system makes predictions about where the target will be and programs accordingly (Leigh and Zee, 2006). If this fails, or if the object is simply moving too quickly, the saccadic system is used to refocus the target on the fovea. The smooth pursuit system relies on multiple sites within the central nervous system, including the pons and cerebellum.

The smooth pursuit system is used for tracking moving objects while the head is stationary. It is also activated when one tries to fixate on a stationary object during head movement. Specifically, head movements activate the VOR effectively moving the eyes resulting in the stationary image of interest moving across the fovea. This act triggers the smooth pursuit system to override the VOR, which suppresses the slow phase of vestibular nystagmus and allows one to fixate on the object of interest. This is referred to as *fixation suppression.*

Optokinetic nystagmus (OKN) is a repetitive series of fast and slow eye movements responsible for maintaining stable vision during movement of the visual surround. OKN and pursuit are both tracking responses and are often activated at the same time. OKN shares pathways with other eye movements, but the nucleus of the optic tract plays an important role (Leigh and Zee, 2006).

The OKN system contributes to gaze stabilization when an image moves across the retina. This system is important

because the peripheral vestibular system does not provide accurate information during very low frequency–sustained movement. For this type of movement, the OKN system is activated and attempts to stabilize a moving image by moving the eyes in the same direction. Together, optokinetic and vestibular nystagmus are combined to provide stable vision across a wide frequency range of movement.

Vestibulo-ocular Reflex

The VOR contributes to ocular stability when the head is in motion. The eye movements induced by the vestibular system (i.e., the VOR) are compensatory (Leigh and Zee, 2006). That is, they oppose head movements or changes in head position and act to keep the fovea of the retina on an object of interest. To put things in perspective, a quick turn of the head to the left sends input through multiple ocular motor nuclei resulting in a compensatory reflex pulling the two eyes to the right. The horizontal VOR (hVOR) is the focus of most clinical vestibular testing and is what we will discuss here.

A simplified description of the hVOR pathway is shown in Table 20.3. When the head turns to the left, endolymph lags behind the head movement because of inertia and the left cupula in the horizontal SCC is deflected toward the kinocilium whereas the right cupula is deflected away from the kinocilium. Thus, discharge from the left SCC increases and discharge from the right SCC decreases. Increase in activity from the left SCC results in an increase in activity in the left VN, causing an excitatory output to the right lateral rectus muscle and the left medial rectus muscle (i.e., pulling the eye to the right) while inhibiting the right medial rectus and left lateral rectus (i.e., the paired agonist and antagonist eye muscle counterparts; Table 20.2). Since the right cupula is deflected away from the kinocilium, the right SCC sends a signal decreasing the activity of the right VN, causing a decrease of excitatory output to the left lateral rectus and the right medial rectus (Table 20.2). The end result is movement of the eyes to the right (to compensate for the head turn to the left) with a fast saccade back to the left (i.e., left-beating nystagmus; Figure 20.12).

TABLE 20.3

Horizontal Vestibulo-ocular Reflex (hVOR)

1. Receptors (cristae of the horizontal/lateral SCC)
2. Afferent pathway (first-order neurons at Scarpa's ganglion in the superior vestibular nerve)
3. Central connection (second-order neurons at the VN)
4. Efferent pathway (medial longitudinal fasciculus)
5. Third-order neurons at the motor nucleus of cranial nerves III and VI
6. Effector muscles (medial and lateral rectus)

Nystagmus is a biphasic, rhythmic, repetitive movement that has a well-defined slow and fast phase (Markham, 1996). Conventionally, it is named (i.e., right or left) by the direction of the eye during the fast phase. The vestibular system mediates the slow phase of the nystagmus; as such vestibular nystagmus (i.e., nystagmus provoked by head movement) is typically quantified by its maximum slow-phase velocity (SPV). Following the slow phase of the nystagmus, the PPRF neurons produce a saccade bringing the eyes back to midline (i.e., the fast phase of nystagmus).

Neural Integrator and Velocity Storage

The vestibular system responds to input frequencies between 0.003 and 5 Hz (e.g., 0.01 Hz is a very slow postural sway and 5 Hz is akin to an active head turn during walking). Because of the mechanical properties of the cupula, the system is less sensitive to input frequencies less than 0.8 Hz and greater than 5 Hz. However, the vestibular system has a brainstem-mediated process for enhancing the sensitivity to low frequencies, called the *neural integrator,* located in the VN (specifically the caudal pons) that is under the control of the cerebellum (i.e., nodulus and uvula).

The time taken for the slow-phase eye velocity to decline to 37% of its initial value after the onset of a velocity test stimulus is called a *time constant* (TC). In response to continual earth axis rotation, cranial nerve VIII produces neural activity until the cupula in the lateral SCC returns to neutral position (TC = 6 seconds). The nystagmus intensity of the VOR shows rise similar as the VIIIth nerve; however, the nystagmus duration is considerably longer (TC = 16 seconds). In other words, the eye response of the VOR persists for a longer period of time than the drive from the peripheral end organ. The prolongation of nystagmus beyond the duration of the peripheral drive is attributed to the neural integrator. The neural integrator acts to extend the low-frequency response of the VOR by one order of magnitude, a function that has been referred to as *velocity storage* (Raphan et al., 1979). Loss of velocity storage affects low-frequency sensitivity of the vestibular system and is identified in clinical assessment by a change in the timing properties of the VOR (i.e., decreased TCs on trapezoidal testing or increased phase on sinusoidal harmonic testing).

The central pathways of velocity storage are not fully understood but there appears to be both "direct" and "indirect" pathways driving the VOR. The "direct" pathway is mediated by the electrical signal originating from the lateral SCC, passing through the first-order neurons in the VIIIth cranial nerve, to the second-order neurons leaving the medial VN and synapsing on the ocular motor nuclei and abducens nuclei, and finally the third-order neurons terminating on the ocular motor muscles. The "indirect" pathway travels from the vestibular nerve through a series of neural connections and terminates on the midline cerebellum. The

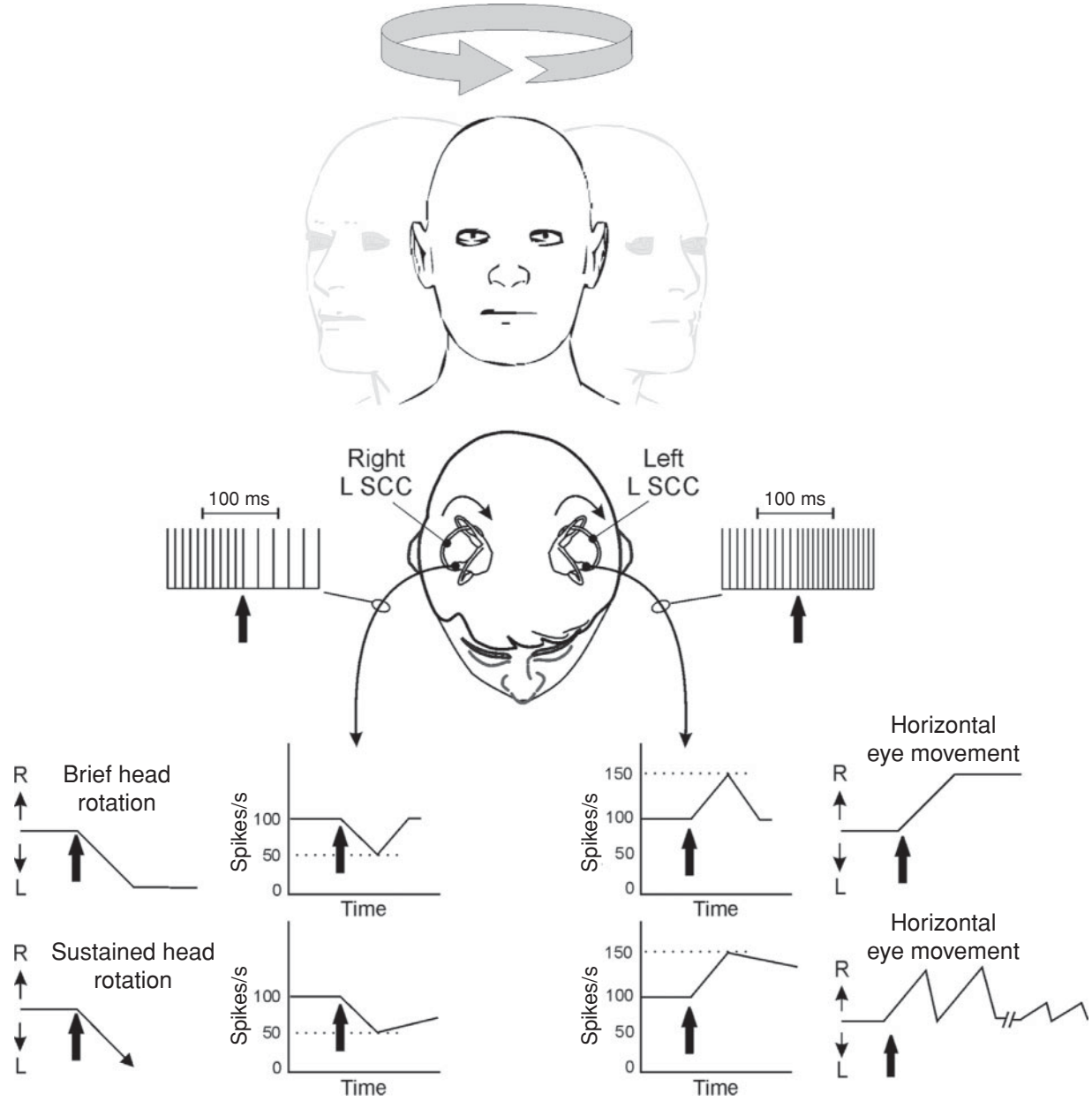


FIGURE 20.12 Head turn to the left results in left beating nystagmus. [From Canalis RF, Lambert PR. [2000] *The Ear: Comprehensive Otology*. Philadelphia, PA: Lippincott Williams & Wilkins; Figure 13, p. 127.]

cerebellum plays an important role in velocity storage function, specifically the efferent portion of the "indirect" pathway extending from the midline cerebellum (i.e., nodulus and uvula) and projecting to regions of the superior and medial VN. Output from the "indirect" pathway persists for a longer period of time than the input it receives from the periphery (Galiana and Outerbridge, 1984). Both the "direct" and "indirect" velocity storage pathways are integrated centrally by a commissural network originating in the brainstem.

In summary, velocity storage is dependent on the electrical drive from the peripheral vestibular end organ, the integrity of the VN, commissural fibers in the brainstem, and connections between the cerebellum and VN. Under normal circumstances, it serves to widen the dynamic range of the vestibular response. In the case of vestibular dysfunction, abnormal velocity storage is the most sensitive parameter on rotary chair testing and is used in conjunction with ear-specific measures to provide valuable information as to the status of the peripheral and central vestibular system.

Vestibulospinal Reflex (VSR)

The VSR is used to produce transitory contractions of muscles to maintain posture, equilibrium during movement, and muscular tone. Whereas the effector organs of the VOR

are the EOMs, the effector organs of the VSR are the extensors of the neck, trunk, and extremities. Stimulation of the SCCs and the otolith receptors leads to a variety of patterns of activation of neck and body muscles. A push–pull mechanism exists between the extensor muscles (e.g., triceps) and flexor muscles (e.g., biceps). The VSR requires a coordinated action of both extensor and flexor muscles to respond to postural disturbances.

Connections from the VN to the spinal cord are through three main pathways, the MVST, the LVST, and the reticulospinal tract. The MVST and LVST provide direct connections to neck motorneurons and indirect connections through spinal interneurons. The reticulospinal tract has indirect connections with the VN through the reticular formation.

The MVST receives input from both the saccule and utricle via the medial VN, but the saccular input is more prominent. The MVST also receives afferent input from all SCCs, though the input from the lateral and superior SCCs is more prominent than the posterior SCC input. The MVST pathway descends bilaterally through the MLF into the spinal cord (Barmack, 2003).

The main afferent input to the LVST is from the utricle and posterior SCC via the lateral VN. Fibers from the LVST descend into the ipsilateral central funiculus of the spinal cord (Barmack, 2003). Both the MVST and LVST send fibers to the spinal cord, but the MVST is more important for coordinating the muscles and providing tonic input to motor neurons that stabilize the head position in space (i.e., neck and cervical muscles). The LVST results in the activation of extensor motor neurons and inhibition of flexor neurons in the upper and lower extremities.

The *vestibulocollic reflex* (VCR), part of the VSR, is the focus of most clinical vestibular testing as it is what we assess with the cervical vestibular–evoked myogenic potential (cVEMP) test. The basic pathway is shown in Table 20.4. The saccule sends an electrical signal through the inferior vestibular nerve to the medial vestibular nucleus. A signal is then sent to the spinal cord motor neurons via the MVST, resulting in an increase in the ipsilateral activation of the extensor motor neurons and inhibition of the flexor motor neurons in the neck and cervical region. Clinically, the cVEMP measures the inhibition of the flexor motor neurons through a skin electrode over the sternocleidomastoid muscle (SCM).

TABLE 20.4

Vestibulocollic Reflex (VCR)

1. Receptors (macula of the saccule)
2. Afferent pathway (first-order neurons at Scarpa's ganglion in the inferior vestibular nerve)
3. Central connection (second-order neurons at the VN)
4. Efferent pathway (MVST)
5. Motor nucleus cranial nerve XI
6. Cranial nerve XI (spinal accessory)
7. Effector muscle (sternocleidomastoid muscle)

LESIONS OF THE VESTIBULAR SYSTEM

What Happens to the VOR in Unilateral Impairment

The severity of signs and symptoms of vestibular impairment is predicated on the degree of damage, the speed of onset, and whether the damage is unilateral or bilateral. In an acute unilateral impairment, vertigo symptoms are usually severe. With gradual unilateral loss, it is possible that the symptoms associated with an acute event never occur because incremental changes in vestibular function are offset by incremental compensation in the central nervous system. Similarly, a patient with gradual bilateral loss may also not experience vertigo, but instead report oscillopsia and ataxia under dynamic conditions.

This section will examine what happens in an acute unilateral impairment of one vestibular end organ, in this case the right side (Figure 20.13B). A right-sided impairment results in a decrease in the electrical drive from the right end organ to the nerve and to the VN on that side. The result is in an electrical code similar to that created during a leftward rotational (counterclockwise) head movement resulting in a slow-phase eye movement toward the right, followed by fast-phase movement to the left (i.e., motion-induced left-beating nystagmus shown in Figure 20.13A). In the case of an acute impairment, the nystagmus will continue whereas the asymmetry between sides remains. In this example, the lesion of the right vestibular nerve will result in a continual left-beating nystagmus creating the illusion of movement of the surroundings, often perceived by the patient as vertigo. In the acute period, the nystagmus will be direction fixed and occur in all directions of gaze and will follow Alexander's law (i.e., amplitude of nystagmus increases when gazing in the direction of the fast phase; Jacobson et al., 2008). The nystagmus augments when vision is denied.

Central Compensation

Acute unilateral peripheral vestibular impairment yields an asymmetry in neural firing causing a persistent nystagmus and retinal slip that triggers a central compensation process (Zee, 1994). Static compensation is complete when the tonic electrical activity of the two VN is restored. Keeping with the example of right peripheral vestibular damage, the compensation process would begin within hours of the initial lesion to the right side. The first step is to eliminate the tonic asymmetry in the firing rate of vestibular neurons. The asymmetry in neural firing rates and the spontaneous nystagmus is reduced when the cerebellum downregulates

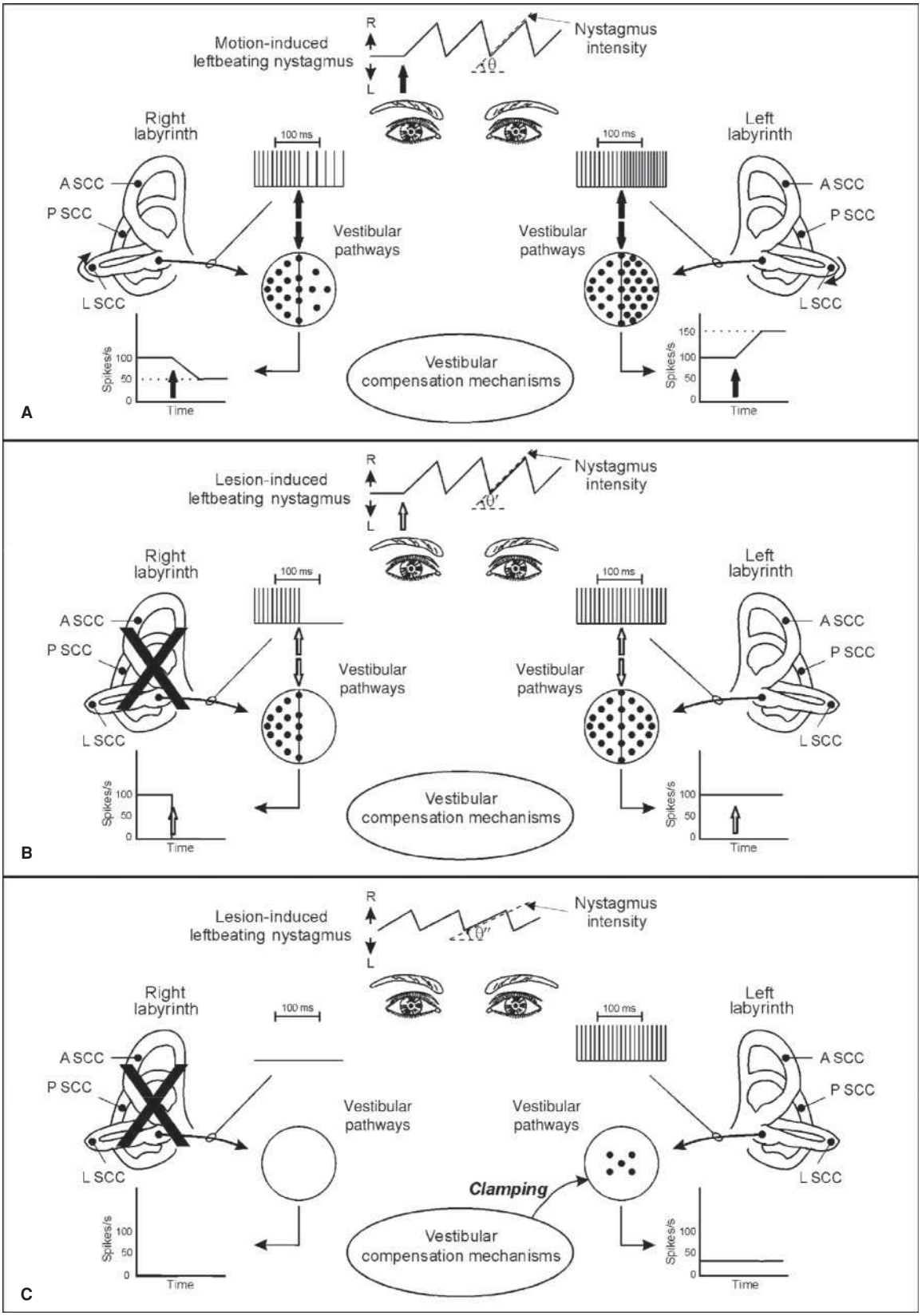


FIGURE 20.13 Vestibular compensation after lesion to the right peripheral vestibular lesion. **A.** Normal physiology following leftward head rotation. **B.** Acute loss of vestibular function on right side. **C.** Cerebellar clamping reduced acute symptoms. **D.** Release of cerebellar clamping. **E.** Static compensation is complete. [Canalis RF, Lambert PR. [2000] *The Ear: Comprehensive Otology*. Philadelphia, PA: Lippincott Williams & Wilkins; Figure 19, pp. 136–137.] [*continued*]

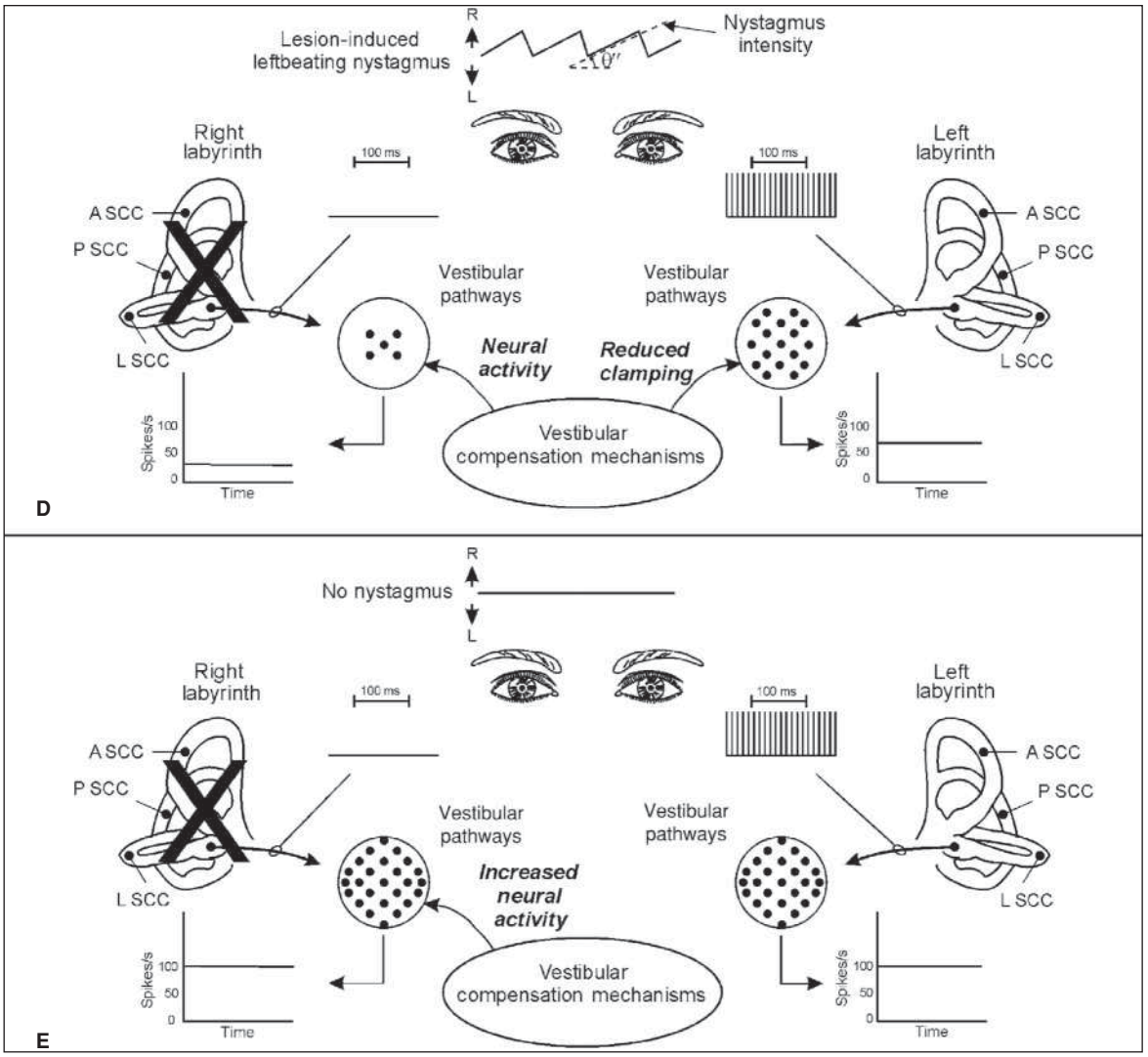


FIGURE 20.13 [*Continued*]

the electrical activity of the VN on the left (intact) side (i.e., "cerebellar clamping"; Figure 20.13C). Typically, within 1 week of the lesion, neural activity returns to the right VN (Figure 20.13D). The activity does not originate from the right vestibular end organ or nerve; it originates from central sources (i.e., vestibulocerebellum). The activity in the left VN then begins to increase as the downregulation from the cerebellum ceases (i.e., a release from clamping; Figure 20.13D). After 3 weeks, static compensation should be complete. The electrical firing returns to its prelesion level on the left side and neural tone is restored to the VN on the right side (Figure 20.13E). The central nervous system is "guessing," based on the electrical input from the left side, what activity it would have received had the right peripheral vestibular system been intact. When static compensation is complete, the patient's vertigo stops, spontaneous nystagmus disappears, and postural control is restored. Retinal slip still occurs during rapid head movements and the patient may report some oscillopsia.

Dynamic compensation occurs when the gain of the vestibular pathway is modified to accommodate unilateral loss of input during head movement. Continuing with the previous example, after static compensation has occurred, the tonic resting rate at the level of the VN is equal between the right and left side. Head rotation generates the neural asymmetry at the level of the VN required for awareness of rotation. However, the size of the asymmetry is half of that generated by two functioning inner ears (since, in our example, only the left vestibular end organ is functioning) and as such the velocity of the slow phase of nystagmus is too low to match the head movement resulting in retinal slip. During dynamic compensation, the central vestibular pathways are "recalibrated" (Zee, 1994). That is, the gain of the VOR pathway must essentially be doubled to generate the same compensatory eye movement generated before the lesion. The retinal slip that occurs with rapid head movements drives the neural integrator to decrease velocity storage (i.e., increase electrical flow). The increase in outflow of

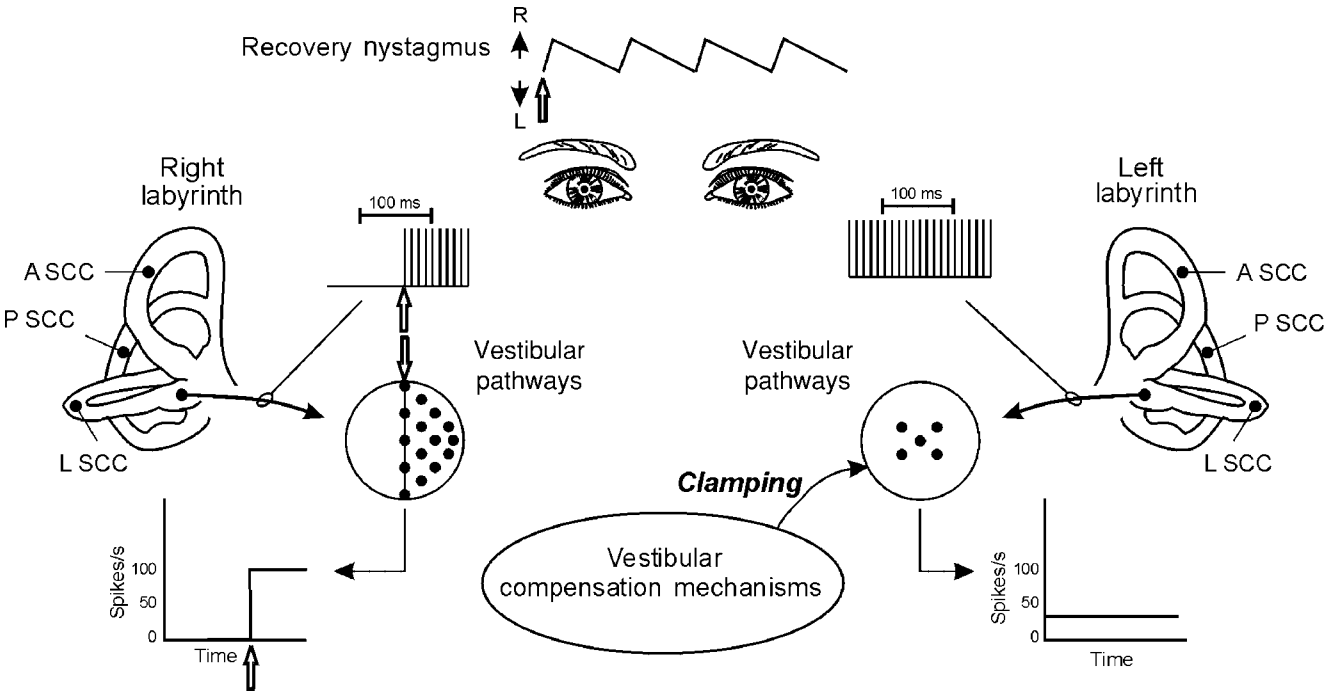


FIGURE 20.14 Recovery nystagmus [Katz, 6th ed. Figure 19.20, p. 455].

the velocity storage mechanism increases the gain (electrical output) of the neural integrator and increases the gain of the VOR so that compensatory eye movements occur during dynamic head movements (Figure 20.13F). Consequently, diminishing the storage capacity of the neural integrator degrades low-frequency VOR performance (i.e., recall the purpose of the neural integrator is to enhance the vestibular system's sensitivity to low frequencies). Complete dynamic compensation results in the elimination of VOR asymmetry and oscillopsia (retinal slip); however, degradation of low-frequency VOR performance often remains (Leigh and Zee, 2006).

It is possible, during central compensation, for a spontaneous nystagmus to paradoxically beat toward the lesioned ear. This is called *recovery nystagmus* or "Bechterew's nystagmus" (Figure 20.14) and usually occurs in individuals with fluctuating vestibular impairments (e.g., Meniere's disease; Leigh and Zee, 2006). Recall, during "cerebellar clamping" the firing rate of the VN on the intact side is reduced to lessen the asymmetry between ears. Typically during this phase of compensation the level of neural activity at the VN is bilaterally reduced. In a stable impairment, static compensation will continue as described above. With a fluctuating lesion, the damaged side can spontaneously recover function. If this occurs during compensation, then an asymmetry once again exists between the sides; only this time the VN of the damaged ear shows greater neural activity relative to the intact ear (Figure 20.14). As a result, the clinician may observe a recovery nystagmus. For this reason, a spontaneous nystagmus should not be used in isolation to localize the site of lesion.

NEW TRENDS

Do blood perfusion and neural innervation patterns make it possible to have isolated end organ impairments that might be detected through quantitative testing? Figure 20.15 is a simplified block diagram showing the basic blood supply and neural innervation to the membranous labyrinth. Injury to the *superior division* of the vestibular nerve blocks the electrical signals originating from the utricle, anterior SCC, lateral SCC, and small portion of the saccule (not shown in the figure). Injury to the *inferior division* of the vestibular nerve blocks the electrical signals originating from the saccule and posterior SCC. The different neural innervation patterns of each end organ can help explain how a neuritis or ischemia can produce a focal lesion affecting only the saccule and/or posterior SCC (i.e., inferior neuritis) or the utricle, lateral SCC, and/or anterior SCC (i.e., superior neuritis).

A review of the blood supply to each end organ can provide clues as to how thrombus or emboli can produce a specific impairment. If the arterial support from the basilar artery, AICA, or labyrinthine artery is cut off the result is profound hearing loss and total loss of peripheral vestibular system function (i.e., the entire labyrinth including the cochlear and vestibular end organs loses function). If the blood supply from the common cochlear artery is interrupted the result is a profound hearing loss and loss of saccule (i.e., abnormal *cVEMP*) and posterior SCC function only (i.e., *caloric* testing and *ocular VEMP* (oVEMP) will be normal). If a lesion occurs in the main cochlear artery, the result would be a profound hearing loss and an intact peripheral vestibular system. If the posterior vestibular artery is cut off, the result would be a loss

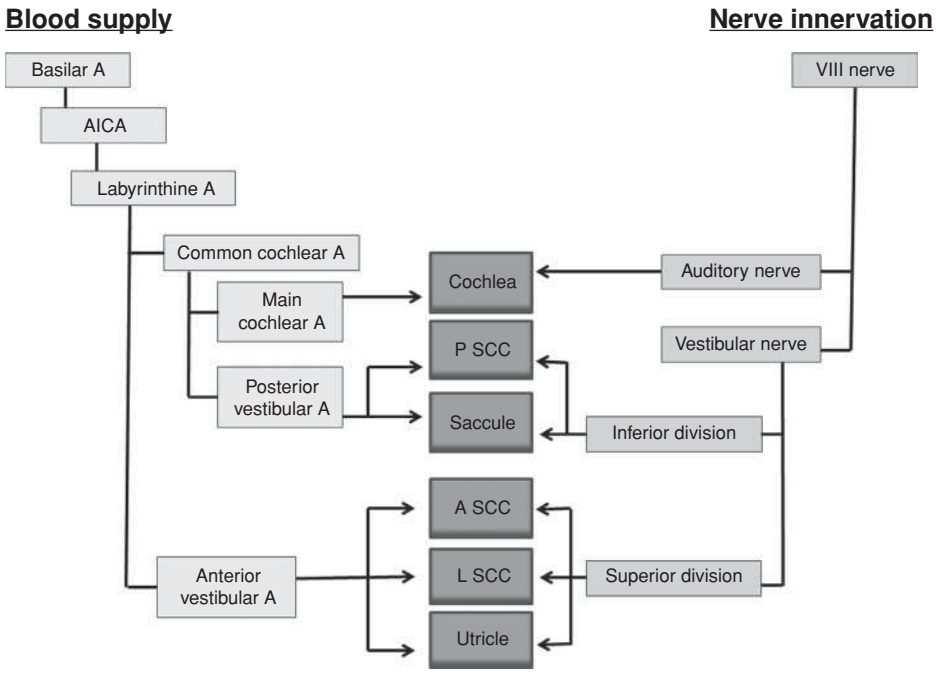


FIGURE 20.15 Block diagram showing schematic of blood supply and neural innervation to the membranous labyrinth. A, artery; AICA, anterior inferior cerebellar artery; P SCC, posterior semicircular canal; A SCC, anterior semicircular canal; L SCC, lateral semicircular canal. [Courtesy of Gary Jacobson, Vanderbilt University.]

of function in the posterior SCC and saccule (i.e., abnormal *cVEMP*) with both hearing and the remainder of the vestibular system intact. Finally, if the anterior vestibular artery is cut off, we see loss of function in the anterior SCC, lateral SCC (i.e., abnormal *caloric test*), and utricle (i.e., abnormal *oVEMP*) with normal hearing, normal saccule (i.e., normal *cVEMP*), and normal posterior SCC function.

A number of recent papers have been published suggesting that the caloric test, cVEMP, and oVEMP can vary independently and provide topologic information regarding vestibular impairments (e.g., Jacobson et al., 2011). For example, one may have an impaired saccule or inferior vestibular nerve (i.e., as assessed with a cVEMP) but the remainder of peripheral vestibular system is normal (i.e., normal caloric test and normal oVEMP). So the cVEMP, oVEMP, and caloric test results may vary independently for patients with various peripheral vestibular system impairments. Ongoing research in the utility of the video head impulse test (vHIT) may allow us to assess the posterior and anterior SCCs independently as well. In the near future, it may be possible to test all aspects of the peripheral vestibular system (i.e., all 10 end organs and superior and inferior divisions of the vestibular nerve). Thus, the combination of results from cVEMP, oVEMP, caloric, and vHIT has the potential to provide localizing information about the specific site or sites of peripheral vestibular system impairment.

FOOD FOR THOUGHT

1. The cupula is a gelatinous material that divides the SCCs from the vestibule and senses angular acceleration. Why can't the cupula sense gravity (i.e. like the macula within the saccule)?
2. Describe the vestibulo-ocular reflex (VOR) pathway.
3. If the neural connections between the cerebellum and vestibular nuclei are not intact, can central compensation following an acute vestibular lesion be completed? Why or why not?

REFERENCES

Angelaki DE, Cullen KE. (2008) Vestibular system: The many facets of a multimodal sense. *Annu Rev Neurosci.* 31, 125–150.

Baloh RW, Honrubia V. (1995) *Physiology of the Vestibular System.* 2nd ed. St. Louis, MO: Mosby.

Baloh RW, Kerber KA. (2011) *Clinical Neurophysiology of the Vestibular System.* New York, NY: Oxford University Press.

Barin K, Durrant JD. (2000) *Applied Physiology of the Vestibular System.* Philadelphia, PA: Lippincott Williams and Wilkins.

Barmack NH. (2003) Central vestibular system: Vestibular nuclei and posterior cerebellum. [Review]. *Brain Res Bull.* 60 (5–6), 511–541.

Dohlman GF. (1971) The attachment of the cupulae, otolith and tectorial membranes to the sensory cell areas. *Acta Otolaryngol.* 71 (2), 89–105.

Galiana HL, Outerbridge JS. (1984) A bilateral model for central neural pathways in vestibuloocular reflex. *J Neurophysiol.* 51 (2), 210–241.

Goldberg JM, Highstein SM, Moschovakis AK, Fernandez C. (1987) Inputs from regularly and irregularly discharging vestibular nerve afferents to secondary neurons in the vestibular nuclei of the squirrel monkey. I. An electrophysiological analysis. *J Neurophysiol.* 58 (4), 700–718.

Hudspeth AJ. (2005) How the ear's works work: Mechanoelectrical transduction and amplification by hair cells. *C R Biol.* 328 (2), 155–162.

Ito M. (1993) Neurophysiology of the nodulofloccular system. *Rev Neurol (Paris).* 149 (11), 692–697.

Jacobson GP, McCaslin DL, Kaylie DM. (2008) Alexander's law revisited. [Case Reports]. *J Am Acad Audiol.* 19 (8), 630–638.

Jacobson GP, McCaslin DL, Piker EG, Gruenwald J, Grantham SL, Tegel L. (2011) Patterns of abnormality in cVEMP, oVEMP, and caloric tests may provide topological information about vestibular impairment. *J Am Acad Audiol.* 22 (9), 601–611.

Kusakari J, Kambayashi J, Kobayashi T, Rokugo M, Arakawa E, Ohyama K, Kaneko Y. (1981) The effect of transient anoxia upon the cochlear potentials. *Auris Nasus Larynx.* 8 (2), 55–64.

Lee H, Ahn BH, Baloh RW. (2004) Sudden deafness with vertigo as a sole manifestation of anterior inferior cerebellar artery infarction. *J Neurol Sci.* 222 (1–2), 105–107.

Leigh RJ, Zee DS. (2006) *The Neurology of Eye Movement.* Philadelphia, PA: F.A. Davis.

Lundberg YW, Zhao X, Yamoah EN. (2006) Assembly of the otoconia complex to the macular sensory epithelium of the vestibule. *Brain Res.* 1091 (1), 47–57.

Markham CH. (1996) *How Does the Brain Generate Horizontal Vestibular Nystagmus?* New York, NY: Oxford University Press.

Money KE, Bonen L, Beatty JD, Kuehn LA, Sokoloff M, Weaver RS. (1971) Physical properties of fluids and structures of vestibular apparatus of the pigeon. *Am J Physiol.* 220 (1), 140–147.

Raphan T, Matsuo V, Cohen B. (1979) Velocity storage in the vestibulo-ocular reflex arc (VOR). *Exp Brain Res.* 35 (2), 229–248.

Straka H, Vibert N, Vidal PP, Moore LE, Dutia MB. (2005) Intrinsic membrane properties of vertebrate vestibular neurons: Function, development and plasticity. *Prog Neurobiol.* 76 (6), 349–392.

Vollrath MA, Kwan KY, Corey DP. (2007) The micromachinery of mechanotransduction in hair cells. *Annu Rev Neurosci.* 30, 339–365.

Zee DS. (1994) *Vestibular Adaptation.* Philadelphia, PA: F.A. Davis.

CHAPTER 21

Evaluation of the Patient with Dizziness and Balance Disorders

Troy Hale, Henry Trahan, and Tabitha Parent-Buck

INTRODUCTION

Balance disorders are common and can occur in patients of all ages, constituting a significant personal and public healthcare burden. It is often reported that dizziness is the third most common complaint among outpatient medical visits and the single most common complaint among elderly patients. Dizziness and balance disorders may result from abnormalities in a variety of organ systems including the vestibular system, central or peripheral nervous system, cardiovascular system, and cerebrovascular system. Additionally, medications taken by patients can contribute to symptoms of dizziness. When a patient presents to the balance clinic, the primary goal of the healthcare provider is to investigate the symptoms and conduct evaluations to narrow the differential diagnosis. Although most patients with dizziness have a benign condition, a small percentage may have a potentially life-threatening underlying cause involving the brain, heart, or the circulation of blood necessitating more immediate medical management. In many cases the patient with an acute balance disorder will recover spontaneously requiring only short-term treatment for the symptoms. However, patients demonstrating more chronic symptoms may require significant intervention from healthcare providers to evaluate and manage the dizziness.

More than 4 out of 10 Americans will experience an episode of dizziness sometime during their lives significant enough for them to seek medical care (NIH News in Health, 2012). Data suggest that one in five patients over 65 years of age experience problems with balance or dizziness annually. A review of data from the US Census Bureau's National Health Interview Survey, collected in 2008, provided alarming statistics on balance issues in the elderly population (Lin and Bhattacharyya, 2012). For patients 65 years of age and older, 20% or 7 million people in the United States reported experiencing dizziness or a balance problem in the previous 12 months. With respect to quality of life and functional impact, of those individuals who experienced dizziness or balance problems, 27% reported that the balance issues prevented them from participating in normal activities. The restriction in activities included not engaging in exercise (61%), social events (46%), driving (47%), and participation in work/school (38%). Additionally, for 26% of those with balance issues, simple activities of daily living such as bathing, dressing, and eating were also reported as affected.

Epidemiologic studies relate balance disorders to the increased incidence of falls in the aging population. The Centers for Disease Control and Prevention in the United States report that roughly one-third of adults 65 years of age and older fall each year, resulting in injuries that limit activities and independent living. Of patients who fall, 20% require medical attention (Gillespie et al., 2013). In addition, falls among older adults, especially those resulting in fractures, are a leading cause of injury-related deaths. Patients suffering from dizziness or imbalance may report unsteadiness, light-headedness, vertigo, dysequilibrium, or a feeling of fainting/pre-syncope. Such conditions may ultimately lead to serious medical, physical, emotional, and social consequences such as loss of independence, isolation, and injuries from falls. Because of the impact of dizziness on the quality of life of patients and the increased risk of falls, a number of falls prevention programs have been investigated and found to provide effective methods for reducing falls (Gillespie et al., 2013).

Given the vast number of acronyms commonly used in relation to vestibular screening and diagnostic measures, Table 21.1 provides a list that will aid in recognizing many terms and their abbreviations used throughout this chapter.

CLINICAL PRESENTATION OF THE DIZZY PATIENT

Our innate ability to maintain balance and navigate safely through the environment depends on sensory information that is gathered from the visual, vestibular, and somatosensory (proprioceptive) receptors of the body. A properly functioning balance system allows us to maintain stable vision with movement, orient ourselves with respect to gravity, and make automatic postural adjustments for maintenance

TABLE 21.1

List of Screening and Diagnostic Vestibular Tests or Measures with Commonly Used Acronyms

Acronym	Test Name or Measurement
AHR	Active head rotation
BPPV	Benign paroxysmal positional vertigo
CDP	Computerized dynamic posturography
CTSIB	Clinical Test of Sensory Integration of Balance
DHI	Dizziness Handicap Inventory
DP	Directional preponderance
DVA	Dynamic visual acuity
ENG	Electronystagmography
HIT	Head impulse test
HSN	Headshake nystagmus
HVT	Hyperventilation test
OKN	Optokinetic nystagmus
SHA	Sinusoidal harmonic acceleration
SCV or SPV	Slow-phase component eye velocity/ slow-component eye velocity or slow-phase velocity
UW	Unilateral weakness (may also be referred to as reduced vestibular response [RVR])
VFX	Visual fixation
VNG	Videonystagmography
VOR	Vestibulo-ocular reflex
VSS	Vertigo Symptom Scale
VVOR	Visually enhanced vestibulo-ocular reflex

of stability. When a person is complaining of imbalance or dysequilibrium, the first task of the audiologist is to have the patient describe the symptoms in detail. This information forms the basis for beginning to determine which sensory input system, or which combination, is contributing to the patient's complaints.

Signs and Symptoms

Many patients will report their presenting symptom as "being dizzy." The term "dizzy" is not particularly specific because it broadly refers to some perception of imbalance, disorientation in space, or false feelings of movement. Patients may describe symptoms with terms such as dizziness, dysequilibrium, spinning, swimming, light-headedness (pre-syncope), floating, turning, and perhaps even vertigo. Although the symptoms reported by patients can provide valuable information for formulating an assessment plan, caution must be taken because the terms used are subjective and can be misleading. The importance of gathering more detailed information to further characterize the reported symptoms will be emphasized below under the topic of the neurotologic case history. Dizziness is often classified into distinct categories based on quality or character of presentation. Various authors have suggested from four to six general categories with the most common descriptors being vertigo, dysequilibrium, pre-syncope, light-headedness, and gait instability. Each of these is briefly described here.

Vertigo has been described as a hallucination or illusion of movement because of an imbalance in the central or peripheral vestibular system. The spinning or tilting illusion of movement can be of self, of surroundings, or a combination of both. Vertigo is often associated with nystagmus, oscillopsia (blurring or oscillation of the visual field), postural imbalance, and autonomic symptoms (e.g., sweating, pallor, nausea, and/or vomiting).

Dysequilibrium is a state of altered static (standing/sitting) or dynamic (ambulating) postural balance without vertigo. Patients presenting with dysequilibrium often complain of visual disturbance, unsteadiness, imbalance, and/or falls. Typically, this condition reflects a failure or mismatch of sensory integration among body systems (e.g., visual, vestibular, muscular, or neurologic) or a disturbance of motor control. Dysequilibrium may be further subdivided into psychologic and ocular subcategories with the former characterized by feelings of anxiety, fear, or confusion and the latter with visual integration deficits often leading to motion sickness.

Pre-syncope is a syndrome characterized by a sensation of impending loss of consciousness (as opposed to syncope, which is actual fainting) and may be associated with a cardiovascular abnormality, malaise, generalized weakness, diaphoresis, visual disturbances, nausea, facial pallor, and/or epigastric (abdominal) distress. Orthostatic hypotension is a common cause of pre-syncope, but arrhythmias, postural orthostatic tachycardia syndrome, hyperventilation, panic attacks, and other conditions can produce the sensation as well. Episodes of pre-syncope are generally relieved with lying down or reclining.

Light-headedness is another common descriptor and is generally meant to imply a head sensation that is not vertiginous or pre-syncopal and not related to ambulation. Light-headedness can be transient or persistent and is often associated with anxiety or hyperventilation syndrome. Patients who complain of this sensation often say that they feel like they are floating, they are giddy, or their head is somehow detached from their body. Because light-headedness is such a vague and variable term, it may actually reflect other types of dizziness that are not properly described.

Gait instability or ataxia is an unsteadiness or inability to perform coordinated muscle movements. Gait unsteadiness can arise when there is a deficit in the central nervous system or either the central or peripheral vestibular systems. Symptoms with this type of presentation are generally constant and sometimes progressive, with patients

reporting that they feel like they are intoxicated during ambulation.

The Neurotologic Case History

Most vestibular disorders cannot be distinguished from one another based solely on laboratory studies. Additionally, laboratory studies tell us little about a particular patient's functional disability as a result of his/her condition. As such, balance function tests are best interpreted in conjunction with an in-depth neurotologic case history. The neurotologic case history begins with the acquisition of a full depiction of the patient's self-described symptoms. Several aspects must be investigated including, but not necessarily limited to, the feeling created by the symptoms, any precipitating events, the onset (e.g., when did they begin), the frequency (e.g., how often they occur), and the duration (e.g., how long do they last).

The general health condition preceding the onset of symptoms should be determined (e.g., any minor or major health issues such as a cold, gastrointestinal or respiratory problems, or head trauma). It is equally important to gather a complete drug history, including information about any medical or nonmedical care the patient has sought for the dizziness. This should include questions about the use of recreational drugs; the use of prescription medications; and the use of homeopathic or naturopathic herbs, supplements, and vitamins.

To the extent possible it is also important to identify any associated symptoms, recognizing that some of these symptoms may occur with more than one type of dizziness. Headache, for example, can occur with vertigo (e.g., migraine, brainstem ischemia), motor dysequilibrium (e.g., cerebellar infarct or hemorrhage), pre-syncope, and psychosomatic dizziness (often associated with anxiety, sleep deprivation, and caffeine overuse/withdrawal) (Sowerby et al., 2010). A careful evaluation of the patient's complaints and the answers to several simple questions can lead the audiologist to suspect whether the patient is suffering from a peripheral vestibular disorder, a central disorder, or a multifactorial disorder, which may alter the order or types of tests with which an individual is evaluated. Table 21.2 provides a summary of key areas to address during the case history process.

Although obtaining the neurotologic case history can be time-consuming, the collective sum of the patient's responses is used to initially separate out suspected vestibular versus nonvestibular disorders. This may be indicated by the patient's descriptions of onset, duration, and provoking factors of the dizziness. If the patient complains of the sensation that the room is spinning for about 1 minute after rolling over in bed or looking up, the clinician should suspect that a peripheral disorder may exist, specifically benign paroxysmal positional vertigo (BPPV). If the vertigo is continuous without fluctuation for long periods of time, such as weeks or more, a central cause should be suspected.

TABLE 21.2

Key Points to Address in the Neurotologic Case History Related to Dizziness

Area of Questioning	Probes (Preferably Asking Open-Ended Questions)
Description of the feeling/ experience	What does the sensation feel like? Why are you here?
Severity	Is it mild, moderate or severe?
Onset	It is new, ongoing or changing since onset?
Duration	Is it short [seconds to minutes], intermediate [minutes to hours], long [>24 hours]?
Frequency	How often? [i.e., single episode vs. multiple and daily vs. weekly vs. occasionally]
Provoking factors	When do the attacks occur? Does anything make the dizziness occur [i.e., change in head, neck or body position; loud noise, exertion, diet, visual stimuli]?
Associated symptoms	Do you also have hearing loss, tinnitus, aural fullness, pain, headache, visual disturbances, numbness, nausea, or vomiting?
Other medical history/ general health	Did any illness or health changes occur near the onset?
Drug case history	Any new medications or changes in prescriptions?
Level of disability or impact on function	No restrictions to activities to completely unable to engage in routine activities

In other instances, the preceding factors play a key role in highlighting a suspected origin for the dizziness or imbalance. For instance, if the patient reports the possibility of a viral illness prior to the sudden onset of dizziness and changes in hearing, this may point towards a viral labyrinthitis. In such cases, a hearing evaluation should be administered along with balance function assessment. Trauma to the head and/or neck may indicate neurologic involvement or, once again, BPPV. Medications that were prescribed for existing medical conditions, or to ameliorate symptoms of dizziness, could result in side effects and/or vestibulotoxicity and impaired balance function. Table 21.3 provides an overview of how the qualitative details gathered from appropriate questions can contribute to differentiating between suspected vestibular and nonvestibular sites of origin.

TABLE 21.3

Differentiating Vestibular and Nonvestibular Dizziness on the Basis of Patient Complaints

Component	Vestibular	Nonvestibular
Common descriptive wording used by patients	Off-balance, spinning (usually the environment moves), tilting, feels like car sickness, too much to drink, unsteady	Light-headed, floating on a cloud or swimming sensation, out-of-body, self-spinning but world is steady
Time course	Distinct attacks (episodic)	All day long (constant)
Common antecedent or exacerbating factors	Change in body position or quick head movements up or down or side to side	Stressful situations, heavy mouth-breathing (hyperventilating), heart thumping
Common symptoms	Nausea, vomiting, unsteadiness, ringing in ear(s), decreased hearing, blurred vision or bouncing vision	Syncope, difficulty concentrating, numbness, tension in body with or without accompanying headache

Self-Assessment Inventories

To assess the functional limitations of an individual's balance problems many health professionals employ self-assessment inventories. These can help to ascertain the patient's functional state or, stated otherwise, how the physiological problems affect the patient's quality of life. Several such self-assessment scales are reported in the literature; two of the common ones are described below.

The Vertigo Symptom Scale (VSS) (Yardley et al., 1992) was developed to assess the symptoms and the relationship between vertigo, anxiety, and emotional disturbances in an effort to examine the relative influence of vertigo and anxiety on reported handicap and distress. The VSS consists of 36 items describing common symptoms often reported by or observed from patients with vertigo. With the VSS, the patient is asked to rate the frequency of his/her experience over the preceding 12 months, and the results are summed and analyzed by the examiner in relation to vertigo and anxiety/autonomic symptom subscales.

The Dizziness Handicap Inventory (DHI) (Jacobson and Newman, 1990) is a standardized measure of self-reported limitations of daily life imposed by the patient's symptoms. The DHI consists of 25 questions that pertain to dizziness or unsteadiness and are to be answered by the patient with "yes," "no," or "sometimes." There are three subscales which investigate functional, physical, and emotional impacts on disability. Scoring ranges from 0 to 100 with those scoring above 10 indicating at least a mild handicap.

"Bedside" Screening Procedures

Evaluating patients with dizziness and/or balance disorders takes time. Often, when a patient is referred for evaluation, it may not be feasible or appropriate to perform a full vestibular test battery. Fortunately, simple "bedside" screening tools are available and, in the vast majority of patients, produce results that coincide with the more extensive laboratory studies. Appropriately applied bedside measures can assist the clinician in the identification of site of lesion and qualification of functional impairment and, in some cases, are a useful counseling tool for patients. Screening measures, however, have limited validity and reliability. It is important to remember that a negative result on a bedside screening does not necessarily mean that the patient does not have the disorder being assessed. Sensitivity and specificity of screening measures vary widely and some have not been thoroughly investigated. In almost all cases, bedside tests should be used to direct patient management and should be corroborated by more comprehensive evaluations.

Determining which bedside tests to employ for a particular patient can be challenging. A typical screening battery may be driven by patient symptoms or may be more general, consisting of multiple measures to evaluate central ocular-motor control, as well as vestibulo-ocular and vestibulospinal reflex function. Bedside tests need not be limited to the initial patient interview either. Some screening procedures such as the swivel chair study and the Clinical Test of Sensory Integration of Balance (CTSIB) can be combined with electronystagmography (ENG) or videonystagmography (VNG) to expand the laboratory investigation, particularly when equipment such as rotary chair and computerized posturography may not be readily available. Although a comprehensive list of bedside testing is beyond the scope of this chapter, some of the common tests are briefly described below.

Ocular Motility

The ocular-motor and vestibular systems share many close anatomic and physiological connections. For this reason, eye movement can provide considerable information to assist in a preliminary evaluation. Certain patterns of nystagmus can be associated with either central neurologic or peripheral labyrinthine disorders. Prior to any examination, a quick

assessment of ocular-motor control should be performed by the clinician. This can be done quickly at the bedside using a finger or a probe. The clinician should examine the relative position of each eye within the orbit during center gaze, establish ocular range of motion, and assess the pursuit and saccadic mechanisms to get an idea of the patient's control of volitional eye movement. Central nervous system disorders or other ophthalmoparetic disorders have the potential to skew vestibular interpretation so it is advisable to identify these ahead of time. Any notable abnormalities may also indicate possible central nervous system involvement, necessitating additional referral.

Head Impulse/Head Thrust

The head impulse test (HIT) or head thrust test utilizes rapid passive head movements to evaluate a patient's functional vestibulo-ocular reflex (VOR) and identify the side of lesion in cases of unilateral or bilateral peripheral vestibular loss (Halmagyi and Curthoys, 1988). The patient is stationed in front of the examiner with his/her head tilted forward-down, approximately 30 degrees, to position the lateral semicircular canals coplanar (parallel) to the ground. The patient is then instructed to fixate on a target, typically the clinician's nose or forehead, and to maintain fixation on that target throughout the duration of the procedure. The examiner gently grasps the patient's head on both sides and passively guides it through a brief, unidirectional, high acceleration thrust. The movement must be rapid (>3,000 degree/s^2), unanticipated by the patient, and not greater than a 20- to 30-degree displacement. The clinician monitors the patient's eye movements to see whether visual fixation is maintained throughout the entire excursion or whether the patient loses visual contact and must make quick corrective eye movements (termed "catch-up saccades") to reacquire the target. Patients with a significant unilateral or bilateral vestibular loss will have their eyes slip off the target when the head is thrust in the direction of the impaired labyrinth, resulting in a corrective saccade during or after the thrust. For example, a patient with a significant unilateral vestibular loss on the left side will experience a loss of visual fixation with leftward head thrust and therefore produce a catch-up saccade to the right to reacquire the target. This process occurs because of decreased neural contribution to the VOR from the ipsilateral ear and because the inhibitory signal from the contralateral ear is not sufficient to stabilize gaze during rotation. Therefore, the eyes initially travel with the head and a refixation saccade is necessary to reacquire the target. If there is no significant peripheral vestibular loss and the VOR is normal or near normal on the side of the movement, the eyes will remain fixed on the point of interest and no catch-up saccade will be necessary. Horizontal head movement in this manner assesses the lateral semicircular canals, whereas the anterior and posterior canals can also be evaluated by rotating the head diagonally within the respective planes (Aw et al., 1996). The HIT procedure is typically repeated multiple times in an unpredictable fashion, bidirectionally within the plane of interest, until a proper assessment has been made. The head thrust exam has been shown to have good specificity (>82%) but variable sensitivity (45% to 84%) in detecting unilateral or bilateral vestibular hypofunction, confirmed with caloric irrigation (Perez and Rama-Lopez, 2003; Schubert et al., 2004). Some measure of this variability between the head thrust test and caloric irrigation may be attributable to the vastly different stimuli employed by both tests, particularly as it relates to stimulus frequency. Most of the literature does suggest that the overall sensitivity of the head thrust increases as the degree of peripheral vestibular system impairment increases, particularly for those with caloric weaknesses greater than 40% (Perez and Rama-Lopez, 2003). Clinical utility of the HIT may be strengthened even further with new computerized diagnostic systems that are now available on the market.

Dynamic Visual Acuity

The dynamic visual acuity (DVA) test evaluates a patient's ability to perceive objects correctly while actively moving his/her head. Normally, losses in visual acuity are minimized during head movement by the VOR which helps maintain gaze on a fixed target by driving the eyes in the equal but opposite direction of head movement. When the VOR is impaired, as is common in patients with bilateral vestibular hypofunction and various central disorders, visual acuity is degraded during head movement resulting in oscillopsia. The DVA test compares a patient's static visual acuity to his/her active visual acuity, measured during head rotation. The patient is seated in front of a Snellen eye chart at the appropriate distance and asked to read the letters or figures to the lowest line possible to establish the baseline static visual acuity. The examiner then gently grasps the patient's head from behind on both sides and passively guides it back and forth at a frequency between 2 and 7 Hz and with less than 20 to 30 degrees of displacement in the yaw (horizontal) plane. The patient is again asked to read the lowest line possible while the head is being moved. The lowest line at which the patient is unable to correctly identify at least half of the letters or figures is noted. Subjects with normal DVA will have no change or just a single-line change from baseline in their visual acuity during head rotation. Those with abnormal DVA will experience significant blurring in the visual field and will have line changes of two or greater during head movement (Longridge and Mallinson, 1984). Care must be taken by the clinician to avoid pausing at the turnaround points, and the patient should alternate the direction of line reading to control for letter or figure memorization. The sensitivity and specificity of the DVA test has been shown to be quite variable in the literature largely because of differing techniques and clinician experience.

Headshake

The headshake test evaluates a patient's central velocity storage mechanism. When the head is shaken vigorously back and forth for 20 to 30 seconds and then stopped, a transient vestibular nystagmus may emerge in patients with central and peripheral vestibular system disorders. This nystagmus is called headshake nystagmus (HSN) and is believed to reflect a dynamic asymmetry within the VOR. The patient is fitted with Frenzel lenses or VNG goggles (with vision denied to prevent VOR suppression) and stationed in front of the examiner with his/her head tilted forward-down, approximately 30 degrees, to position the lateral semicircular canals coplanar to the ground. The patient is then instructed to shake his/her head back and forth (actively) approximately 30 to 45 degrees from the center at a frequency of at least 2 Hz for 20 to 30 seconds. Alternatively, the head can be moved passively by the examiner. Immediately following cessation of the headshake, the patient is instructed to open his/her eyes and the clinician notes if any nystagmus is observed. In subjects with normal vestibular function, no nystagmus will be present and the test is deemed negative. In subjects with unilateral vestibular loss, a brief period of horizontal nystagmus may emerge and the test is considered positive. A test is generally said to be positive if at least five beats of nystagmus are noted within 20 seconds post-headshake (Guidetti et al., 2006).

Typically, the nystagmus will beat toward the more neurally active side, but other patterns have been described. This is believed to occur because of asymmetric neural firing of the central velocity storage integrator (Hain et al., 1987). The headshake test has been shown to have poor sensitivity for mild to moderate unilateral vestibular impairment, but with increasing degrees of vestibular hypofunction, there is general agreement in the literature that the sensitivity of the headshake test increases. Patients with bilateral vestibular impairment typically do not exhibit any post-HSN because the central neurons are not receiving asymmetric input. The presence of a vertical nystagmus after horizontal or vertical headshake has also been identified in some studies and may indicate a lesion affecting the central vestibular pathways, specifically the brainstem or cerebellum (Zee and Fletcher, 1996). The headshake test can be performed as a screening tool or as an addition to the typical ENG/VNG battery.

Romberg

The Romberg is a test of static balance that assesses the function of lower spinal reflexes by forcing the subject to rely solely on vestibular and proprioceptive inputs to maintain upright posture. The patient is instructed to stand with feet together and arms at the sides or folded across the chest while maintaining a quiet stance with minimal swaying. If the patient is able to do this effectively with eyes open, the stance is repeated with eyes closed. Each condition is held for approximately 30 seconds or until a fall becomes imminent. Qualitative observations of postural adjustment, including direction and amplitude of sway, and any "falls" are made by the clinician. The test can be further modified (i.e., "sharpened") to increase the difficulty by having the patient stand in the tandem position (toe to heel) or on a foam pad. The Sharpened Romberg reduces the amount of proprioceptive feedback, therefore increasing the reliance on vestibular information. Regardless of the method, the clinician should always maintain close proximity to the patient while performing the Romberg to prevent a fall if the patient loses his/her balance. If the patient can maintain the standing position with eyes closed for 30 seconds without falling or without substantial increase in sway, the test is negative. The test is considered positive when there is increased sway or a "fall" with eyes closed. Increased sway or falls are associated with proprioceptive loss from the lower extremities. Patients with acute vestibular deficits and cerebellar dysfunction may also show a pattern of increased sway, although the latter is not typically affected by eye closure. Patients with acute uncompensated vestibular impairment may show a tendency to fall toward the side of lesion with eyes closed, but patients with compensated or chronic vestibular loses will often perform normally on the Romberg test.

Fukuda Stepping

The Fukuda step test is a screening measure used to identify the presence of a peripheral vestibular impairment that manifests as an asymmetry in lower extremity vestibulospinal reflex function. The Fukuda test should only be performed on patients who are able to maintain balance during eyes-closed Romberg testing. The patient is instructed to stand with eyes closed, arms extended outward in front of the body (palms down), and march in place for 50 steps. The angle, direction, and deviation from the original starting point are observed by the clinician after the stepping is complete. The patient should be able to complete this task without significant angular deviation (less than 45 degrees) and without significant linear translation (less than 1 m). A rotation of greater than 45 degrees in any direction is considered abnormal (Fukuda, 1959; Furman and Cass, 2003).

As it was originally described, patients with significant unilateral vestibular impairment would have a tendency to deviate (rotating greater than 45 degrees) in the direction of the affected labyrinth. This was a commonly held assumption for many years, but more recent investigations (Honaker and Shepard, 2012) have suggested that this may not be universally true and that the direction of rotation may be pathology dependent. For clinical purposes, the Fukuda step test should be considered positive if the angle of rotation exceeds 45 degrees or if excessive sway is noted. However, it should not be used in isolation to attempt to lateralize or localize lesions of the vestibular system as the type of pathology may produce results contradictory to the direction of rotation.

Hyperventilation-Induced Nystagmus

The hyperventilation test (HVT) is used to help identify disorders of the vestibular system and/or cranial nerve VIII. It may also be useful in identifying individuals who suffer from anxiety-related dizziness. When a patient is asked to voluntarily overbreathe for an extended period of time, a transient nystagmus may emerge in individuals with various central or peripheral vestibular disorders. This nystagmus is called hyperventilation-induced nystagmus and is believed to be caused by a change in blood gas concentration and nerve conduction. The patient is fitted with Frenzel lenses or VNG goggles (with vision denied), stationed in front of the examiner, and asked to take deep rapid breaths for a period of 30 to 90 seconds at a rate of about 1 breath per second (Minor et al., 1999). The clinician maintains close contact with the patient at all times to increase awareness of any change in patient postural stability and to minimize the risk of falling as this screening may bring about dizziness and reduced equilibrium. The examiner observes eye movement before and after the hyperventilation task and notes any evoked nystagmus or change in existing nystagmus. If the patient exhibits no nystagmus, the test is considered negative. Patients may report dizziness, a light-headed sensation, or decreased equilibrium as the result of this exam but this, in isolation, should not be interpreted as a positive result. The appearance of a new nystagmus or reversal of an existing nystagmus is considered a positive HVT. Peripheral vestibular lesions typically produce horizontal nystagmus with fast phases beating toward the side of lesion. However, in cases of total unilateral loss (e.g., vestibular nerve section), the nystagmus will typically beat away from the side of lesion (Chee and Tong, 2002). Hyperventilation-induced nystagmus may also be observed in patients with central lesions such as demyelination because of multiple sclerosis or cerebellar ischemia because of an infarct. In such cases, the direction of the nystagmus is unpredictable. If no nystagmus is provoked but the patient becomes severely symptomatic within the first 20 to 30 seconds of overbreathing, anxiety-related dizziness or dysautonomia may be suspected (Choi et al., 2007). Hyperventilation is more likely to provoke dizziness, light-headedness, autonomic symptoms, and acute anxiety attacks in patients with certain anxiety disorders than in the general population. It may also provoke light-headedness and autonomic arousal without significant anxiety in patients with autonomic dysfunction, such as hyperventilation syndrome. Sensitivity and specificity of the HVT vary widely in the literature so it should only be used in conjunction with other laboratory testing and not interpreted in isolation.

Valsalva-Induced Nystagmus

Increased middle-ear and intracranial pressure, as the result of Valsalva maneuver, has been shown to induce eye movements and dizziness or vertigo in patients with abnormalities of the craniocervical junction and disorders of the middle/inner ear. These abnormalities may include Arnold–Chiari malformation, perilymphatic fistula, cholesteatoma, superior canal dehiscence, and anomalies of the ossicles, oval window, round window, and the saccule. To test for Valsalva-induced nystagmus, the patient is fitted with Frenzel lenses or VNG goggles (with vision denied) and asked to perform the Valsalva maneuver in two different methods. In the first approach, the subject is instructed to take a deep breath, pinch his/her nose, and then blow against a tightly closed mouth for 10 to 15 seconds as if attempting to equalize pressure in the ears while diving. This method increases air pressure within the sinuses, pharynx, and middle ear (Walker and Zee, 2000). The clinician examines the patient for any evoked nystagmus during or after the procedure. Additionally, any subjective dizziness, vertigo, or visual changes by the patient should be noted. After sufficient recovery time, the second method is attempted. For the second method, the patient is instructed to take a deep breath and strain against a closed glottis and lips for 10 to 15 seconds, as if pressurizing the lungs while attempting to lift a heavy object. This method effectively increases intracranial pressure by inducing elevated central venous pressure (Walker and Zee, 2000). Eye movement is again observed during and immediately following pressurization and relaxation. The normal patient will exhibit little to no eye movement and will experience no substantial dizziness or vertigo. The abnormal patient may exhibit a conjugate nystagmus with fast phases directed toward the affected ear. Horizontal evoked nystagmus typically indicates involvement of the lateral semicircular canals, whereas torsional and downbeating vertical nystagmus or torsional and upbeating vertical nystagmus indicates involvement of the anterior canals and posterior canals, respectively (Davies, 2004). The direction of the torsion (e.g., the top of the eyeball rotating toward or away from the nose) may also provide information regarding the laterality of the lesion. Fast phases of the torsional nystagmus will typically beat in a clockwise direction for lesions of the left ear and counterclockwise direction for lesions of the right ear. Sensitivity and specificity of the Valsalva test is variable and pathology dependent. In most cases, however, the presence of a positive Valsalva test is a good clinical indicator of an abnormal junction between either the middle ear and inner ear or the middle ear and intracranial space.

LABORATORY STUDIES OF BALANCE SYSTEM FUNCTION

Clinicians commonly utilize information gathered from a patient's medical history, physical examination, and "bedside" screenings as a basis for ordering laboratory studies. The role of the majority of the vestibular laboratory studies is to ascertain the extent and/or site of lesion within the peripheral or central vestibular systems and to aid in the

characterization of any functional impairment. A thorough evaluation of the patient with dizziness and balance disorders may therefore require administration of one or more of the following types of procedures. Not every patient that presents in the dizziness/balance clinic receives a complete battery of all tests. The administration of tests should be limited to those that are minimally necessary to make decisions regarding management. Each of the major types of laboratory studies (ENG/VNG, active head rotation [AHR], rotational chair, otolith function testing, and computerized posturography) is discussed in this section. It is important to note that although the clinical utility of each will be discussed in turn, a detailed description of the origins, methods of delivery, and interpretations are beyond the scope of this chapter.

Electronystagmography and Videonystagmography

Nystagmography (ENG/VNG) is the most commonly practiced method of vestibular assessment. Because of the close physiological connections between the vestibular apparatus and the visual system, eye movements have classically been used to infer functional status of the peripheral vestibular organ and its associated central VOR pathways. ENG utilizes electro-oculography as a means to indirectly track eye movements as a function of time. Changes in eye position are indicated by the polarity of the corneo-retinal potential relative to surface electrodes placed around the eye. The corneo-retinal potential is the difference in electrical activity between the cornea (positively charged front portion of the eye) and the retina (negatively charged back portion of the eye). A typical ENG setup consists of at least five electrodes with one placed at the lateral canthus of each eye to record horizontal movement, one placed above and below at least one eye to record vertical movement, and a common or reference electrode placed on the forehead (Figure 21.1). When the eye is moved within the orbit, there is a corresponding increase in the electrical potential in the direction of eye movement and decrease in the opposite direction. This occurs because the positively charged cornea is now closer to the electrode in the direction of movement. ENG recordings can be performed with eyes open or eyes closed and in either a lighted or darkened environment.

VNG is a more modern and widely administered method of vestibular assessment. VNG utilizes infrared video-oculography as a means to directly record eye movements as a function of time. With VNG, the eyes are illuminated with infrared light and cameras located within the goggles track eye location and movement using the center of the pupil as a guide (Figure 21.2).

Significant differences exist between ENG and VNG systems pertaining to calibration, artifact, test environment, recording of torsional nystagmus, and associated costs. Whereas all of these areas are not discussed in detail here, it is important to note a few of the more important differences. Both ENG and VNG systems utilize two-dimensional recording techniques to track eye movements in the horizontal (yaw) and the vertical (pitch) planes. The video component of VNG, however, allows for qualification in three-dimensional space by allowing the examiner to directly monitor eye movements within all planes simultaneously. This can be advantageous for identifying rotary or torsional nystagmus such as in BPPV variants. This advantage of VNG is limited, however, to the extent that the examiner observes the video of the eye movements during testing, as the actual post-exam tracings are two-dimensional for both

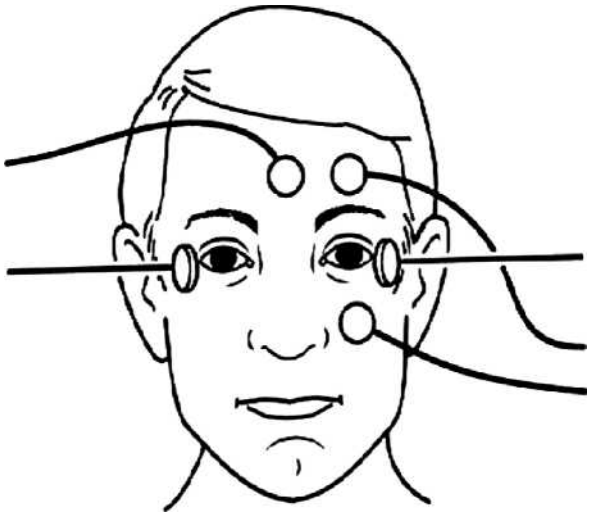


FIGURE 21.1 Typical electrode montage for monitoring eye position as a function of time in both the lateral and vertical dimensions. Note that given the dipole nature of the eyes and the use of electrodes lateral and vertical to the anterior–posterior axis of the eye, this type of configuration is not responsive to torsional movements of the eyes. [Reprinted with permission from Shepard NT, Telian SA. (1996) *Practical Management of the Balance Disorder Patient.* San Diego, CA: Singular Publishing Group.]

A **B**

FIGURE 21.2 VNG goggles: [**A**] With vision allowed; [**B**] with vision denied. [Courtesy of A.T. Still University, AFA Balance and Hearing Institute.]

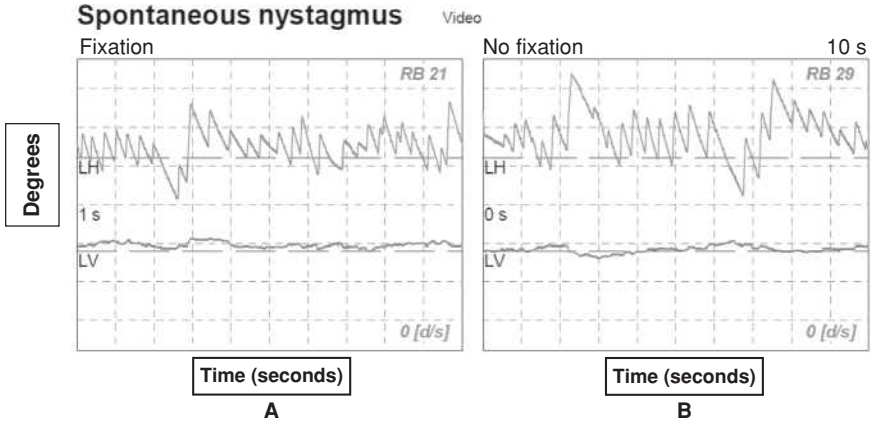


FIGURE 21.3 Plot of horizontal and vertical eye position in degrees as a function of time. The *top lines* were recorded from the horizontal channel (showing right-beating nystagmus) and the *bottom lines* were recorded from the vertical channel (showing no nystagmus). Each panel represents 10 s of tracing. [**A**] Spontaneous right-beating nystagmus at 21 degrees/s [*RB 21*], with fixation. [**B**] Spontaneous right-beating nystagmus at 29 degrees/s [*RB 29*], without fixation. *RB,* right beating; *LH,* left horizontal channel; *LV,* left vertical channel. [Courtesy of A.T. Still University, AFA Balance and Hearing Institute.]

systems. Another primary advantage of VNG is the presence of less recording artifact. VNG is much less susceptible to contamination from myogenic activity and eye blinks which are common pitfalls with ENG systems. Although VNG can be used on the vast majority of patients, there are some situations where ENG is better suited. Those patients for whom ENG may be better suited include (1) patients with claustrophobia or who will not participate in vision-denied conditions, (2) young children who often will not tolerate goggles or for whom the goggles do not fit appropriately, and (3) patients with dark areas on or around the eye that make infrared recording difficult because of the inability of the system to differentiate the pupil from other parts of the eye (e.g., those with permanent makeup or dark areas on the sclera).

Both ENG and VNG systems are designed to record and measure nystagmus (as well as other types of eye movement, discussed later). Nystagmus is a type of eye movement that is oscillatory, that is, it moves in one direction (e.g., rightward or leftward) and then in the opposite direction (see Chapter 20). Nystagmus, in most cases, also typically follows a defined temporal pattern with slow drift in one direction and rapid return in the other direction. By convention, nystagmus is labeled by the direction of the faster phase but its strength is measured by the velocity of the slower phase (in degrees per second). Thus, a nystagmus that has a slow phase to the right and a fast phase to the left is termed a "left-beating" nystagmus. Conversely, a nystagmus that has a slow phase to the left and a fast phase to the right is termed a "right-beating" nystagmus.

Figure 21.3 shows a typical nystagmography tracing which could be recorded from either electrodes or video recording techniques. In this figure, the rapid upward deflection of the tracing (fast-phase component of the nystagmus) is shown, followed in each case with a slower downward drift of the tracing (slow component of the nystagmus). By convention, upward deflections in the recording signify rightward (horizontal trace) or upward (vertical trace) eye movements whereas downward deflections signify leftward (horizontal trace) or downward (vertical trace) movements. For torsional eye movements, there will likely be activity reflected in both the horizontal and vertical tracings simultaneously but given the two-dimensional nature of ENG/VNG tracings (discussed previously), torsional nystagmus is best identified by direct observation. Torsional eye movements are generally referred to by the direction of the beat of the nystagmus in the horizontal plane (e.g., right-torsional nystagmus represents eye movements with the fast phase beating horizontally to the patient's right, together with the eyes beating to the patient's right in the roll plane). Alternatively torsional nystagmus may be referenced as clockwise or counterclockwise based on the direction of the rotation as a whole, but this convention can be somewhat confusing. Regardless of description, the principal parameter measured in the analysis of most types of nystagmus is the slope or slow-phase component eye velocity (SCV) (Figure 21.4).

The typical ENG/VNG exam consists of a series of subtests designed to assess portions of the central and peripheral vestibular systems. ENG/VNG is limited with respect to the peripheral system in that results are based predominantly on measurements of horizontal semicircular canal function with only minimal information garnered from the vertical canals or otolithic organs. As such, broad

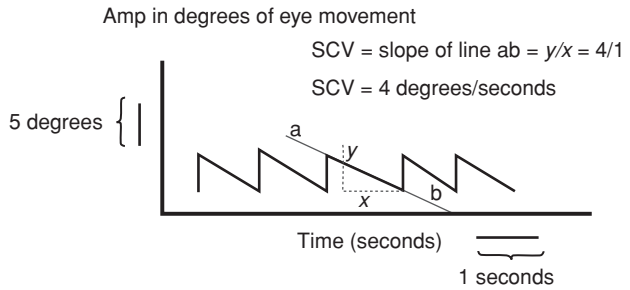


FIGURE 21.4 Right-beating nystagmus is illustrated along with the calculation of slow-component eye velocity (*SCV*), the slope of the line (ab). Upward trace deflections represent rightward eye movement with downward indicating leftward eye movement.

generalizations about vestibular function based on ENG/VNG, in isolation, are not recommended. The typical ENG/VNG exam consists of the following subtests: Ocular-motor evaluation, dynamic positioning, static positional tests, and caloric irrigations.

Ocular Motility Tests

The first portion of the ENG/VNG exam is the ocular-motor evaluation. Tests of ocular motility are useful for investigating the central pathways that are required for the function of the VOR. Abnormalities in these tests typically indicate a central lesion or neurologic disorder. These tests may also be affected by pre-existing nystagmus and can be abnormal in patients with severe fatigue, medication effects, or general inattention. The typical ocular motility battery includes tests of gaze stability, smooth pursuit tracking, saccades, and optokinetic stimulation. Each of these subtests are recorded and quantified according to the eye movements generated during the task and compared to established normative data.

Gaze Stability

Gaze stability testing involves measuring the patient's ability to hold the eyes in a fixed direction of gaze without drifting off the target. Gaze is usually fixed in a central (neutral) position or at points of horizontal and vertical displacement, 20 to 30 degrees from center. The gaze-holding task is performed both with and without visual fixation and each position is typically held for at least 30 seconds. Care must be taken to ensure that the subject's head remains still at all times and that the angle of gaze never exceeds 30 degrees. A normal physiologic endpoint nystagmus could be recorded and misinterpreted if the patient's gaze exceeds 30 degrees past center. Abnormalities of gaze fixation typically suggest brainstem or cerebellar dysfunction.

Smooth Pursuit

Smooth pursuit testing measures the patient's ability to track a moving target and maintain that target of interest on the fovea with continuous fluid eye movements. These movements should follow a smooth (sinusoidal) pattern without any jerking or "catch-up" corrections. During smooth pursuit the target typically moves rightward and leftward or up and down at speeds varied between 0.2 and 0.8 Hz. As the frequency of the stimulus increases so does the difficulty of the task. The recorded eye movements will cause the appearance of a sinusoid on the tracings (Figure 21.5). There are three parameters typically analyzed for smooth pursuit: Velocity gain, symmetry, and phase. Smooth pursuit is the most sensitive of the ocular-motor tests but has poor site of lesion localization because multiple pathways are involved in the response generation. Abnormalities of pursuit are

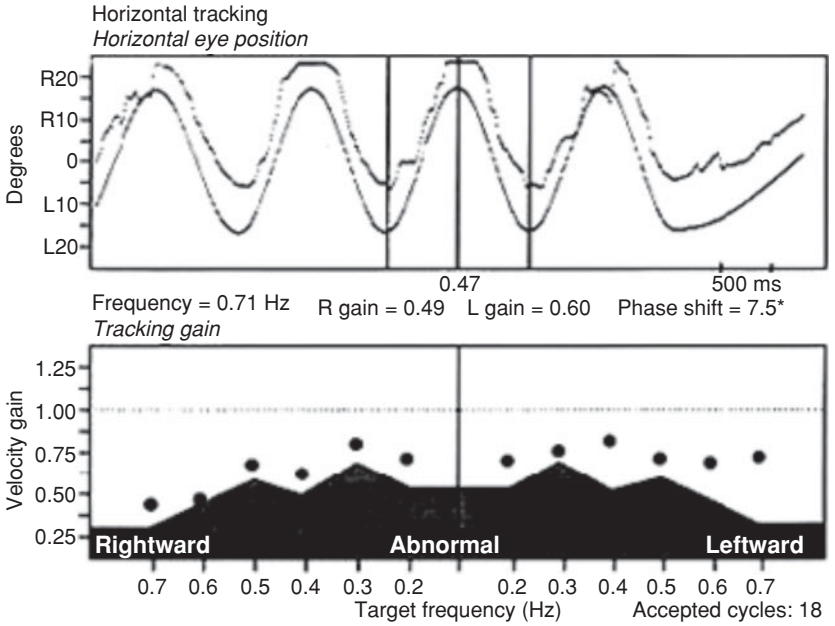


FIGURE 21.5 Smooth pursuit eye movement recording with the target at 0.71 Hz. The **top panel** is a plot of horizontal eye position as a function of time (500 ms time mark shown) for sinusoidal tracking (*dotted trace*). In the same plot (*smooth line*) the target position in degrees as a function of time is given. The panel below the eye and target position plots gives the value of velocity gain as a function of frequency of target movement. The *shaded region* represents abnormal performance.

usually indicative of vestibulocerebellar dysfunction as this area is common to all of the pursuit pathways.

Saccade Testing

Saccades are rapid eye movements designed to bring a target of interest onto the fovea of the eye. They are the fastest eye movements generated by humans and can be either reflexive or voluntary. Saccade testing therefore evaluates the ability of the patient to make quick eye movements in response to a moving target and to maintain that target on the fovea for maximum visual acuity. Typically there are two types of saccadic tests: One is a fixed position saccade and the other is a random position saccade. The time intervals between saccades can also be fixed or random. During evaluation the patient is instructed to locate the target as soon as it moves from one position to the next. The patient is asked to do this as quickly as possible without head movement and without anticipating location. The three parameters of saccadic eye movement that are analyzed include latency (how long it takes for the eyes to react), velocity (how fast the eyes move to the target), and accuracy (how well the eyes acquire the position of the target). A typical tracing is displayed in Figure 21.6. Saccade testing is not as sensitive as pursuit testing but can provide information to aid in the identification of involvement of the brainstem versus the vermis in the posterior cerebellum.

Optokinetic Testing

Optokinetic nystagmus (OKN) tests measure reflexive jerk nystagmus eye movements created by repeated visualization of objects moving horizontally or vertically across the patient's visual field. The test stimuli utilized to produce the OKN response is typically lighted bars or vertical stripes and must occupy at least 80% of the visual field. This process is typically repeated with increasing stimulus velocities (20 and 60 degree/s) and the subject is generally instructed to watch or count the stimuli as they pass. Velocity gain is measured by comparing the speed of the stimulus with the slow-phase velocity (SPV) of the eye recordings. Optokinetic testing is the least sensitive of the ocular-motor tests. At present, OKN best serves as a cross check when abnormalities are seen during pursuit or saccade testing. It is also important to note that light bar stimulus arrays do not provide sufficient visual field stimulation. Therefore, responses generated under such testing conditions amount to nothing more than another form of smooth pursuit testing and not true optokinetic testing.

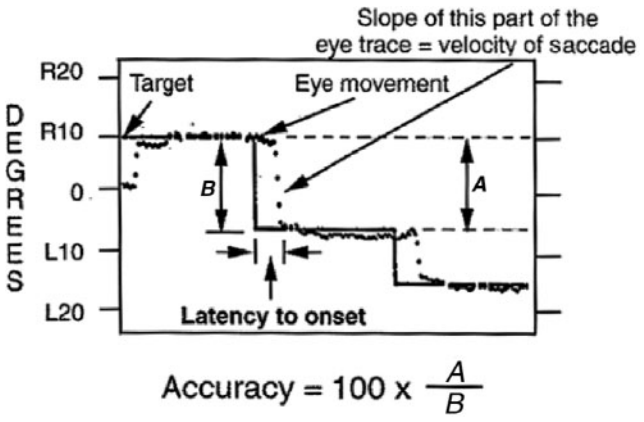


FIGURE 21.6 Schematic of eye and target position in degrees, as a function of time, demonstrating the calculations of velocity, latency, and accuracy of the saccade eye movement for analysis. [Reprinted with permission from Shepard NT, Telian SA. (1996) *Practical Management of the Balance Disorder Patient*. San Diego, CA: Singular Publishing Group.]

Dynamic Positioning

Dynamic positioning requires actively transitioning the patient from one position to another. It is most commonly utilized to assess for the presence of BPPV, which is the most common cause of vertigo in the adult population. Positioning evaluations typically include the Dix–Hallpike maneuver, the horizontal head roll maneuver, or other similar modifications. Because many different BPPV variants exist, an accurate description of any evoked nystagmus present during dynamic positioning is critical for proper identification. Each semicircular canal produces unique eye movements (see Chapter 20) which can be characterized to help localize the disorder.

The Dix–Hallpike maneuver (Figure 21.7) is the most common positioning evaluation used to determine if BPPV of the posterior semicircular canal or the anterior semicircular canal is present. The patient is seated on an examination table so when placed in a supine position (lying on the back) he/she will occupy the length of the table. The patient's head is rotated by the examiner 45 degrees to the right or to left depending on which side is being tested. The examiner stands behind or in front of the patient and briskly brings him/her down into a supine position with the head positioned to the side and slightly lower than the shoulders. Positive Dix–Hallpike responses produce a complex nystagmus with torsional (roll plane) and vertical eye movements. There is also a minor horizontal component (yaw plane) to the nystagmus with most cases of anterior–posterior canal BPPV. The fast component of the torsional nystagmus is generally directed toward the involved ear. This is typically, but not always, the underneath ear. To adequately detect this type of nystagmus, the eye movements must be visualized by the examiner or recorded with video equipment because the rotary component of the nystagmus will not show up in standard electro-oculography or video-oculography tracings, since both are two-dimensional in printed output (see earlier discussion). Torsional nystagmus, evoked in this manner, is not typically able to be suppressed by the central nervous system, so the Dix–Hallpike maneuver can be performed with vision allowed.

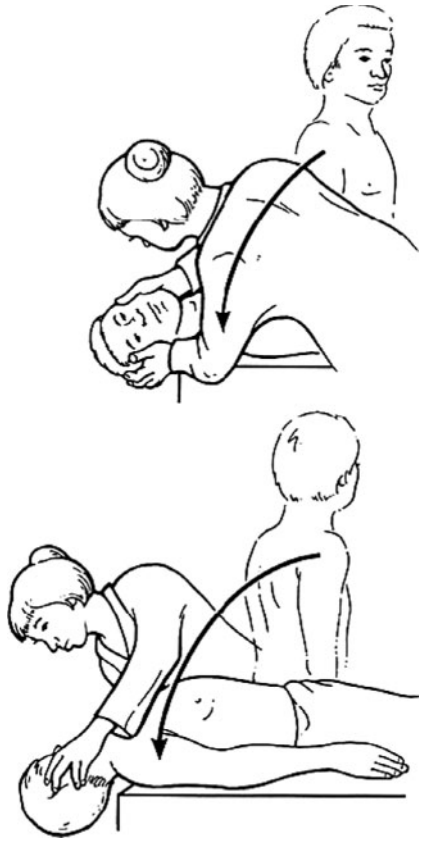


FIGURE 21.7 Illustrations of the technique for the Dix–Hallpike maneuver, for right side (**top**) and left side (**bottom**). Note that the patient's eyes are open and fixating on the examiner. (Reprinted with permission from Shepard NT, Telian SA. (1996) *Practical Management of the Balance Disorder Patient*. San Diego, CA: Singular Publishing Group.)

The horizontal head roll maneuver is used to determine if BPPV of the horizontal semicircular canal is present. The patient begins in the supine position with head at center, slightly elevated, and supported by the examiner. The patient's head is rotated 90 degrees to the right or to the left and held in that position for at least 30 seconds, or if nystagmus is observed, until it abates or is properly identified. The head is then turned back to the center position, pausing briefly, and then rotated in the opposite direction. Positive head roll responses produce horizontal geotropic (nystagmus that beats toward the ground) or ageotropic (nystagmus that beats away from the ground) responses. It is important to note that because the horizontal semicircular canals are coplanar, nystagmus should exist in both directions of head turn. The general convention is that the position of the head with the stronger response reflects the side of involvement for canalithiasis with loose otoconia moving within a canal, and the reverse is true for cupulolithiasis in which otoconia debris is adhered to the cupula of one of the canals. Various modifications of the Dix–Hallpike maneuver and horizontal head roll maneuver exist for those who have difficulty moving into the supine position, such as pregnant women and those with low back pain, obesity, or arthritis.

Static Positional Testing

The primary purpose of the positional evaluation is to identify nystagmus evoked during specific head and body positions relative to gravity. Some clinicians employ these tests in both the sitting and supine positions whereas others employ only the latter. Possible patient orientations include sitting head turned right, sitting head turned left, supine, supine head turned right, supine head turned left, and the "caloric" position (head elevated by 30 degrees up from the horizontal plane). If nystagmus is observed during either the head turned right or head turned left positions, or if the patient is unable to fully rotate his/her head, then side-lying right (body right) and side-lying left (body left) are also investigated. Additionally, in cases where no cervical region injuries or active pathologies are reported, use of head hanging (neck hyperextended) positions may also be added. Regardless of orientation, eye movements are monitored while the patient is held stationary and denied visual fixation (i.e., performed in darkness). Some have suggested that static positional testing should be performed both with and without visual fixation, particularly if any nystagmus is present, as the effect of fixation is the single most useful factor in differentiating nystagmus of central origin from other types. When testing in the vision-denied condition, it is also prudent to appropriately engage the patients by giving them mental "tasks" (e.g., counting or naming objects) to ensure they are alert.

A positional nystagmus may be direction-fixed (always beating in the same direction) or direction-changing (more than one direction observed during the exam). Direction-changing nystagmus is often subclassified into geotropic or ageotropic variants as discussed previously in the dynamic positioning section. Positional nystagmus is effectively a nonlocalizing finding (i.e., can be produced by either central or peripheral lesions); therefore a review of ocular-motor studies in combination with static results are necessary to help differentiate between the two. Generally speaking, the presence of a positional nystagmus is considered to be indicative of peripheral system involvement (assuming normal ocular motility tests) except in the case of (1) direction-changing nystagmus observed in a single head position or (2) if persistent vertical nystagmus is noted with no accompanying horizontal component. Both of these conditions could suggest central pathway involvement. Additionally, if a spontaneous, direction-fixed nystagmus is present and no significant change in that nystagmus occurs during static positional evaluation, then the nystagmus is taken to be reflective of the spontaneous findings and not considered as positional. This situation would suggest little or no influence of the otolith organs in the modulation or generation of the nystagmus but would not otherwise alter the general interpretation of peripheral system involvement.

Positional nystagmus is the most common abnormality described during ENG/VNG evaluations. The exact quantification of static positional nystagmus remains somewhat controversial. The following is one set of conservative criteria which relies on a combination of average SPV, direction, and number of positions in which nystagmus is present to determine if the nystagmus should be reported as an abnormal finding (Barber and Stockwell, 1980):

- SCV >5 degree/s in single position
- SCV <6 degree/s but persistent in four or more of the 8 to 11 positions
- SCV <6 degree/s—sporadic in all positions tested
- Direction-changing within a given head position

Caloric Irrigation

The caloric irrigation is used to help lateralize peripheral lesions by cross-comparing nystagmus responses between the two labyrinths. The process involves timed irrigation of the external auditory canal by one of three delivery methods: Closed-loop water (water circulated inside a thin latex balloon inside the external auditory canal), open-loop water (water circulated in and out of the external auditory canal continuously), or air flow (air circulated in and out of the external auditory canal continuously). Open-loop water systems are typically the preferred method but can be contraindicated in patients with tympanic membrane perforations or when pressure equalization tubes are present. In such cases the closed-loop water irrigation or air method is a suitable alternative. For all of these delivery systems, the irrigating medium (water or air) is set to specific temperatures above or below that of the body, typically temperatures of 44°C for warm and 30°C for cool are used for water systems and 50°C for warm and 24°C for cool are used for air systems. Irrigating the ear in this manner generates a vestibular nystagmus that can be measured and analyzed. The most widely accepted theory about the physiologic origins of the caloric response involves gravity and density changes that occur within the endolymph of the horizontal semicircular canal as a result of temperature change.

To perform the caloric irrigation test, the patient is placed in the supine position with the head elevated at 30 degrees above the horizontal plane. This position, termed the "caloric position," places the horizontal semicircular canal in an almost perpendicular orientation to the ground making it most susceptible to endolymph movement. Air or water is then delivered into the external auditory canal causing an increase or decrease in the temperature of the tested labyrinth. During a warm irrigation, the less dense fluid of the horizontal canal in the endolymphatic space attempts to rise upward. Since the fluid cannot flow around the canal secondary to the cupula, the change in fluid density results in a pressure differential across the cupula that produces a deviation of the cupula toward the utricle, thereby causing stimulation of the horizontal canal. The reverse action occurs for the more dense area of cooled fluid, causing inhibition. This response pattern results in a directional mnemonic known as "COWS" indicating that Cool irrigations will produce a nystagmus beating in the Opposite direction of the irrigated ear and Warm irrigations will produce a nystagmus that will beat in the Same direction as the irrigated ear. Thus, a cool irrigation of the left ear will produce a right-beating nystagmus whereas a warm irrigation of the left ear will produce a left-beating nystagmus.

Caloric responses are interpreted by using a relative comparison of the maximum slow-phase eye velocity for right irrigations versus that of the left irrigations. These values are used to provide a percent comparison of response magnitude (termed unilateral weakness [UW] or reduced vestibular response) and directional bias of eye movement (termed directional preponderance [DP]) (Figure 21.8). A fixation index or fixation suppression of caloric evoked nystagmus may also be included by comparing the maximum SPV response without fixation to that of the minimum SPV response with fixation. Each of these parameters is discussed in greater detail below. It should also be noted that although four irrigations are typical for calculation purposes, there are situations where "ice water" (4°C) may also be utilized and situations where only two monothermal irrigations may be sufficient (Shepard and Telian, 1996).

Unilateral Weakness (Reduced Vestibular Response)

By comparing the relative strength of individual ear responses to the sum of responses for both ears, a UW can be determined using Jongkees' formula:

$$\text{Unilateral Weakness (UW)} = \frac{(RW + RC) - (LW + LC)}{RW + RC + LW + LC} \times 100 \qquad \text{(Eq. 21.1)}$$

where RW is right warm, RC right cool, LW left warm, and LC left cool. The results of this formula will show the relative strength of each horizontal canal, and thus it can be inferred if one side is "weaker" than the other. The SPV values of each response should be taken from the area of greatest magnitude on the tracings (e.g., the 10-second period that yields the strongest responses following irrigation). There are no universally accepted norms for classification of UW. Each laboratory should establish its own normative data. Some laboratories use a 20% cutoff. If one ear is 20% stronger than the other ear, a UW is said to be present in the weaker ear. Other laboratories use values that vary from 20% to 30%. This presents a statistical weakness in determining what is normal and what is not. However, the UW calculation is used by many as the most robust indicator of a peripheral vestibular disorder. It is important to remember that if a spontaneous nystagmus has been found in the preceding gaze stability tests, then the direction and degree of that spontaneous nystagmus must be corrected for when

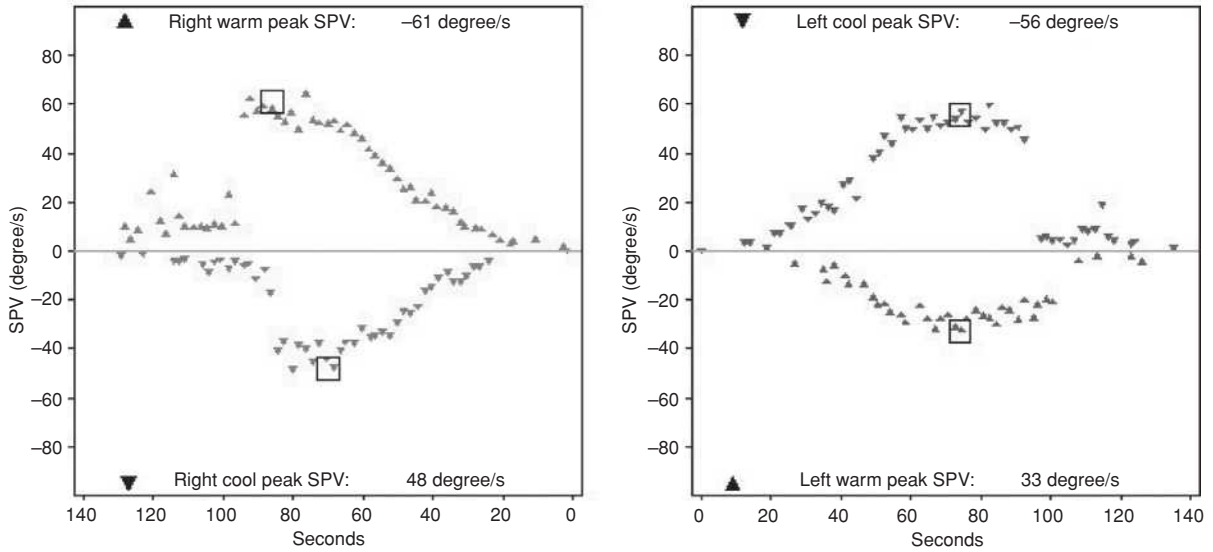


FIGURE 21.8 Plots of slow-phase velocity (SPV) from nystagmus provoked during caloric irrigations as a function of time. The *plotted triangles* represent SPV measurements of the eye movements from the nystagmus tracings. Data for the right ear, warm caloric irrigation, are shown at the **top left**. Data for the right ear, cool irrigation, are shown at the **bottom left**. Data for the left ear, cool, are shown at the **top right**. Data for the left ear, warm, are shown at the **bottom right**. The orientation of the triangular data points represent cool (30°C), ▼, or warm (44°C), ▲, irrigations. The velocity values provided in each box represent the average maximum SPV calculated from the peak areas of nystagmus shown by the *square markers* over the plotted triangle data points. Unilateral weakness equals 10% in the left ear and directional preponderance equals 18% to the right, both of which are within normal limits. (Courtesy of A.T. Still University, AFA Balance and Hearing Institute.)

calculating caloric results to avoid an artifactual error that leads to an incorrect interpretation.

Directional Preponderance

DP is perhaps the weakest of the three caloric parameters. Instead of looking at the overall response strength of each ear as done for the UW parameter, the DP calculation is used to detect the presence of a directional bias of the caloric-induced nystagmus. Since two irrigations are expected to produce right-beating nystagmus and two irrigations are expected to produce left-beating nystagmus, there should be equal values of right- and left-beating nystagmus generated. If the responses in one direction are stronger than in the other direction, a DP exists, indicating a bias within the system enabling nystagmus in one direction to be produced more easily than in the other direction. In determining if a DP exists Eq. 21.2 is used:

$$\text{Directional Preponderance (DP)} = \frac{(RW + LC) - (LW + RC)}{RW + RC + LW + LC} \times 100 \qquad \text{(Eq. 21.2)}$$

where RW is right warm, RC right cool, LW left warm, and LC left cool.

As with the calculation of a UW there are no accepted norms for determining a DP, and it is necessary for each lab to generate normative data. Note that what is being compared is the strength of all right-beating nystagmus to that of all left-beating nystagmus. It is generally accepted that a difference of 30% or more is an indication of a DP in the direction of the larger value. The presence of a DP is considered a nonlocalizing finding in that it can be seen in both central and peripheral disorders, but can also be indicative of failure to correct for a spontaneous nystagmus.

Fixation Index/Fixation Suppression

A well-known feature of caloric-induced nystagmus is that in individuals with intact central function, the evoked nystagmus SPV can be strongly reduced with visual fixation. Typically, once the maximal caloric response has been identified, the patient is instructed to visually fixate on a small stationary target, which necessitates that the eyes be kept open. During this period of visual fixation, measurements of the SPV of the nystagmus are again taken. The values are compared to the strength of the SPV of the nystagmus immediately preceding the visual fixation (peak caloric response), which should be the same area measured for UW and DP. Usually, the strongest nystagmus produced by visual fixation occurs 60 to 90 seconds from the onset of the irrigation. It is recommended that fixation suppression be obtained for all four caloric irrigations and that it is ascertained that the patient is actually attempting to fixate. Some patients experience vertigo to an extent that they cannot, or will not,

make an attempt to perform the task. The patient should fixate for a duration of at least 15 seconds. A comparison of the SPV at the peak of the caloric response is compared to values during visual fixation. As with other measurements of caloric-induced nystagmus there is no general agreement as to what values are considered abnormal, but most clinicians use a 50% to 60% reduction in caloric-induced nystagmus as a sign of normalcy. Failure to adequately suppress caloric-induced nystagmus with visual fixation is a sign of a central vestibular lesion (Baloh and Kerber, 2011). Once again, it is strongly advised that individual clinic norms be established for the proper interpretation of these results.

It is also worth noting that caloric testing, like most tests, does have some inherent limitations. Caloric stimulation is not a true physiologic response. The normal vestibular system operates in a complimentary arrangement, such that if one side is stimulated, the opposite side is inhibited simultaneously. A caloric response is therefore not analogous to normal stimulation. Another limitation of the caloric test is that it only provides information about horizontal semicircular canal function and simulates very low frequencies (0.002 to 0.004 Hz) which are well below the normal operational range of the VOR. Therefore, absence of a caloric response to warm, cool, or ice water irrigations cannot be taken as an indication of complete lack of vestibular function. Rotational chair evaluation would be necessary in this case to better define the true extent of any bilateral peripheral vestibular loss. Despite these inadequacies, the caloric test remains a critical part of the traditional ENG/VNG battery.

Active Head Rotation

AHR evaluates the VOR at higher frequencies, typically 2 to 6 Hz. AHR is considered an "active" test because volitional movement by the patient is required for the stimulus. This is in contrast to rotational chair testing (discussed later) which is deemed a "passive" test because of the fact that the stimulus is delivered by a motor-driven chair and not by the patient. The equipment necessary to perform AHR testing includes infrared goggles or electrodes, an accelerometer attached to the goggles or to a headband, a computer with VOR analysis software, and a stationary visual target. It is important to note that the goggles or headband must be secured tightly to the patient's head during AHR to minimize slippage of the motion sensor which could adversely affect data collection. The patient is seated in front of the stationary target with goggles open (vision allowed) and instructed to move his/her head back and forth, in rhythm with an audible stimulus. During this task, the subject is also instructed to keep his/her eyes focused on the stationary target. The AHR test can be performed in the horizontal or vertical planes, allowing for assessment of multiple semicircular canals. The audible stimulus begins at a slow frequency interval and gradually increases to a faster rate. Several trials are typically performed to obtain sufficient data points. Gain, symmetry, and phase parameters of the VOR response are recorded and compared to normative data for each frequency. Different response patterns have been observed for various pathologies and are not described in detail here because it is beyond the scope of this chapter. AHR is generally well tolerated by most patients, but challenges can arise if the individual cannot tolerate rapid head rotation.

For head movements below 2 Hz, the smooth pursuit system is generally considered the dominant contributor to visual stabilization. For frequencies greater than 2 Hz, however, the VOR functions almost exclusively for that purpose (Grossman et al., 1989). As a result, the AHR test can be performed with vision allowed (goggles open) rather than with vision denied (goggles closed). Caloric irrigations and rotational chair testing evaluate frequencies below 2 Hz and can therefore be influenced by the central ocular-motor system; thus these tests are usually performed in darkness to prevent any visual contribution. AHR more closely approximates natural head movement and should therefore be better suited to characterize VOR function than the nonphysiologic caloric response which simulates speeds well below the operational range of the VOR, and rotational chair which only approaches the low end of natural head movement. The clinical use of AHR systems has been limited up to this point, however, because of concerns regarding sensitivity and specificity in identifying vestibular abnormalities as well as poor test–retest reliability (Fife et al., 2000).

Rotational Chair

Rotational chair testing involves stimulation of the vestibular system by turning the patient in an angular fashion around an earth-vertical axis (Figure 21.9). This is typically accomplished using a sinusoidal or constant velocity paradigm with and without incorporation of visual stimuli. Because rotary chair uses a physiologic stimulus and can be performed at a range of frequencies, it is often used to expand our evaluation of the peripheral vestibular system. Rotational studies can be useful in determining site of lesion, confirming clinical suspicion of diagnosis, counseling the patient, and evaluating rehabilitation potential. They may also be useful for special populations that cannot undergo traditional caloric testing or those with interaural caloric responses that cannot be reliably compared (e.g., young children, individuals with external or middle-ear pathology, individuals with perforations). It is important to note, however, that whereas rotary chair testing does provide some distinct advantages over nystagmography (ENG/VNG), in general it is viewed as complimentary and in most cases not likely to significantly alter patient management, except in the case of a bilateral vestibular loss.

A review of 2,266 patients was performed by Shepard and Telian (1996) investigating the clinical utility of rotary chair testing in the evaluation of peripheral vestibular system

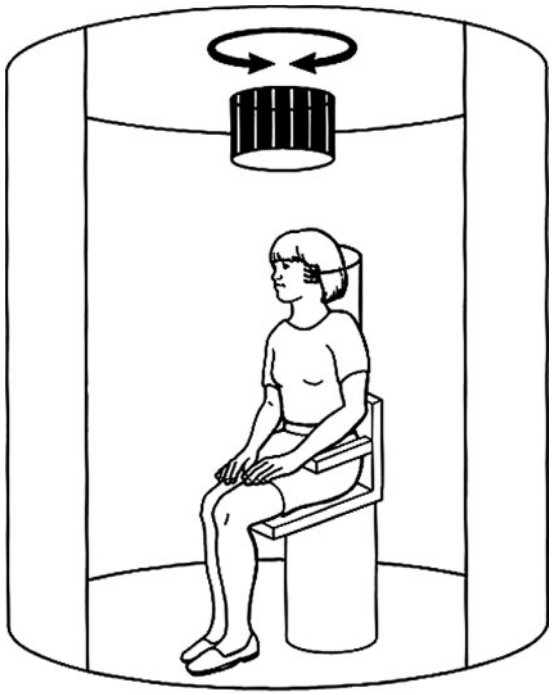


FIGURE 21.9 Generic rotational chair setup. The chair is on a computer-controlled motor within an enclosure and can be rotated in either direction. A device for holding the head to the chair is shown. A means for producing optokinetic stimulation is shown as the drum in the ceiling. [Reprinted with permission from Shepard NT, Telian SA. (1996) *Practical Management of the Balance Disorder Patient.* San Diego, CA: Singular Publishing Group.]

function. Based on that review, there appears to be good evidence for obtaining, at minimum, low-frequency rotational data in certain patient populations with suspected peripheral vestibular system dysfunction. Based on current research, general criteria for when rotational chair testing may be of significant clinical use is given below (Ruckenstein and Shepard, 2000; Shepard and Telian, 1996):

- When either the ENG is normal and ocular-motor results are normal or observed abnormalities would not invalidate rotational chair results. Chair testing is used here to expand the investigation of peripheral system involvement and compensation status.
- When the ENG suggests a well-compensated status (no spontaneous or positional nystagmus), despite the presence of a clinically significant unilateral caloric weakness and ongoing symptom complaints. Chair testing is used here to expand the investigation of compensation in a patient with a known lesion site and complaints suggesting poor compensation.
- When warm and cool caloric irrigations are below 10 degree/s bilaterally, when caloric irrigations cannot be performed, or when results in the two ears may not be compared reliably because of anatomic variability. Chair testing is used in these cases to verify and define the extent of a bilateral weakness or to further investigate the relative responsiveness of the peripheral vestibular apparatus in each ear when caloric studies are unreliable or unavailable.
- When a baseline is needed to follow the natural history of the patient's disorder (e.g., possible early Ménière's disease) or for assessing the effectiveness of a particular treatment, like that of chemical ablation of one or both peripheral vestibular systems.

When utilizing rotational protocols, it is important to remember that the peripheral vestibular system has a complimentary "push–pull" arrangement, so that if one side is stimulated with angular or linear acceleration, the opposite side is inhibited in its neural activity. Therefore, the primary disadvantage of rotational chair testing is its relative inability to yield much information regarding laterality of a lesion. Also, rotational equipment is very costly and takes up considerable space in the clinic so it is not always readily accessible.

A typical rotational chair protocol will likely include sinusoidal harmonic acceleration (SHA) tests performed at several frequencies, step velocity tests, and in some cases visual–vestibular interaction tests. These protocols are discussed in greater detail below. Before performing any rotational study, all equipment should be calibrated and the patient evaluated for any spontaneous or gaze-evoked nystagmus as the presence of which may bias the test results. Electro-oculography or video-oculography is used to monitor and record all jerk nystagmus that is generated in response to the angular chair acceleration stimulus. The slow component of the evoked nystagmus is the VOR and, as with ENG/VNG, is the portion of the eye movement for which velocity is calculated and used for analysis purposes.

Sinusoidal Harmonic Acceleration

The SHA test evaluates the VOR over a range of frequencies, typically harmonics between 0.01 and 1.28 Hz. SHA testing is performed in complete darkness (vision denied) with the patient seated in the upright position and the head tilted forward-down, approximately 30 degrees, to achieve maximum stimulation of the horizontal semicircular canals. The patient is secured tightly at the head, torso, and legs prior to any rotation. Proper restriction of the patient's head and body is critical because the rotational stimulus is being delivered to the vestibular system via rotation of the entire body, thus both the head and body must be fixed for their movement to be congruent. The subject is brought to a constant velocity, typically 50 to 60 degree/s, at different rates and rotated sinusoidally for multiple cycles at each frequency. Either video or electrode recording technique is used to capture the SPV of the eye movements from each cycle of stimulation. The SPV responses from each cycle are added together and then divided by the total number of

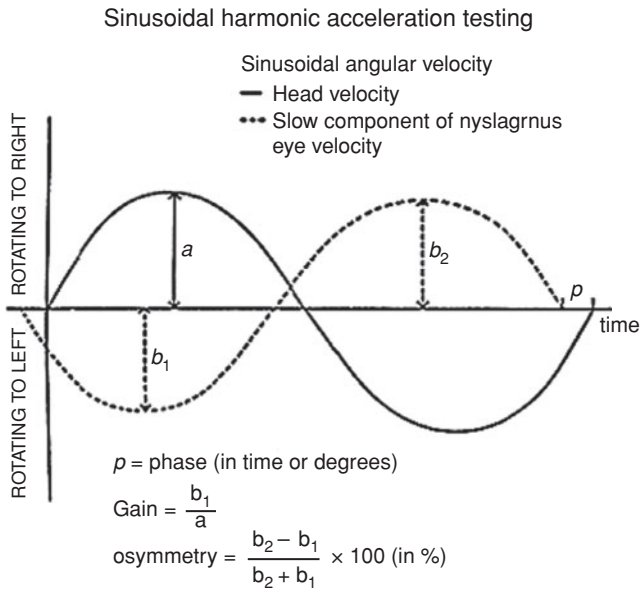


FIGURE 21.10 Pictorial and formulated definitions of the three major parameters used for analyzing sinusoidal harmonic acceleration testing.

cycles performed to get an average response for the tested frequency. The frequency is then changed and the entire process repeated. Protocols may vary from lab to lab but the more cycles performed at a given frequency, the more reliable the data. Pragmatically however, the very slow cycles take a considerable amount of time to complete and therefore performing more than two or three cycles becomes time prohibitive. Unfortunately, the very low frequencies (<0.08 Hz) also produce the weakest response from the VOR and, therefore, have the poorest signal-to-noise ratio. In general, the very low frequencies are also the most likely to produce any unpleasant symptoms such as nausea. The frequencies from 0.16 Hz and above can be completed quickly and easily, however, allowing responses from many cycles to be averaged. This is because the peak chair velocity is fixed for each rotation, so that as the frequency is increased, the subject experiences increasing acceleration with decreasing excursion of the chair.

Three parameters are measured for SHA testing: Gain, symmetry, and phase (Figure 21.10). Some systems may also include a spectral purity measure which details the quality of the data collected. Gain describes the amount of eye movement relative to head movement, symmetry is a comparison measure of rightward rotation to leftward rotation, and phase can be thought of as the reaction time of the eyes in response to head movement. Each measure is described in more detail below. The results of the SHA parameters are plotted by frequency and then compared with manufacturer or clinical normative data (Figure 21.11).

Gain

Gain represents a ratio of eye velocity to chair/head velocity and tells us about the overall responsiveness of the peripheral vestibular system (Figure 21.10). Patients with a unilateral loss of vestibular function often display a reduction in gain in the low frequencies with normalization in the higher frequencies. The principal use of gain measures, however, is to identify and quantify the extent of a bilateral reduction in peripheral vestibular function. Individuals with partial bilateral vestibular weakness may exhibit gain patterns similar to those of unilateral losses, whereas those with complete bilateral hypofunction will exhibit patterns that are reduced across all frequencies. The gain value and the phase component (discussed below) help to verify that severely reduced or absent caloric irrigation responses accurately reflect a true bilateral vestibular weakness and are not the result of an artifact or some other technical error. Gain measures may also play a significant role in determining the course of rehabilitation or prognosis for therapy success. It is important to

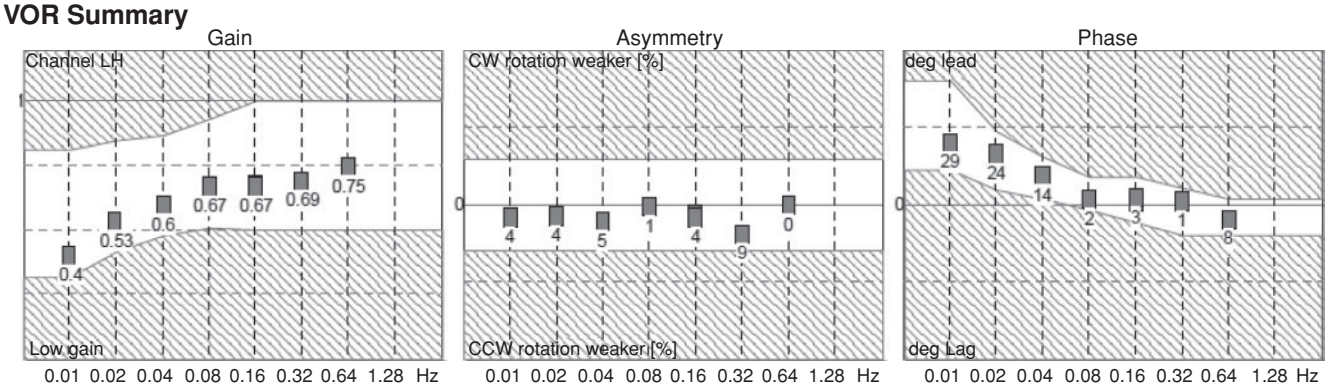


FIGURE 21.11 Normal rotational chair results. The plot on the **left** shows gain (eye velocity divided by head velocity) as a function of frequency of chair sinusoidal stimulation. The plot in the **center** shows symmetry data in percentage as a function of frequency. The plot on the **right** shows phase angle in degrees as a function of frequency. The *darkened areas* represent abnormal performance based on a two standard deviation range above and below the mean. (Courtesy of A.T. Still University, AFA Balance and Hearing Institute.)

note that gain measurements can be negatively influenced by patient alertness, calibration, or technical errors such as light in the booth. Figure 21.11 shows normal results for gain as a function of sinusoidal harmonic stimulation.

Symmetry

Symmetry refers to the difference between the peak right-beating and peak left-beating slow-phase eye velocities divided by the total peak SPVs (Figure 21.10). Symmetry values are calculated by comparing eye velocity while rotating to the right (eye movement to the left) versus eye velocity while rotating to the left (eye movement to the right). This may seem contradictory, but it is important to remember that symmetry values are calculated and named by the direction of the eye movement that is produced by the VOR, which is the slow component. The situation is reversed when discussing DP of caloric irrigations. DP values are calculated by slow-component velocity but, by convention, are named by the direction of the fast phase of the nystagmus. Therefore, a patient who exhibits a right-beating DP (left slow-component velocity greater than right slow-component velocity) may show a left asymmetry on rotational chair testing, indicating that during chair testing, left slow-component velocity was greater than right slow-component velocity. Abnormal symmetry values, under most circumstances, should correspond with any direction-fixed spontaneous nystagmus and correlate with the DP of the caloric response. Asymmetric responses are typically indicative of an uncompensated peripheral vestibular weakness on the side of the stronger SPV response or an irritative lesion on the opposite side. In circumstances where the DP and asymmetry do not agree, the bias may be because of a peripheral lesion with incomplete dynamic compensation or, less likely, the presence of an uncompensated lesion in the central pathways. Figure 21.11 shows a normal result for the symmetry measurement.

Phase

Phase represents the timing relationship between the stimulus (chair/head velocity) and the response (eye velocity) (Figure 21.10). It is the value in degrees to which compensatory eye movements lead or lag movement of the head. Shepard and Telian (1996) report that this parameter has the greatest clinical value because of its ability to accurately identify peripheral system dysfunction but it is also the least intuitive of the three VOR metrics. If eye movement is perfectly compensatory to head movement, then the eyes will be 180 degrees out of phase with the head resulting in zero phase lead. The VOR, however, is a nonlinear mechanism so this only occurs for a limited range of frequencies. During the slower frequencies (<0.16 Hz) the compensatory eye movement will typically lead the head movement and at the faster frequencies the eyes will lag behind the head movement. Abnormally increased phase leads are common in individuals with peripheral vestibular disorders, especially when viewed in conjunction with reduced VOR gain. Increased phase leads may also be seen in persons with damage to the central vestibular nuclei within the brainstem; therefore, increased phase leads should not be looked at exclusively to localize lesions to the labyrinth or cranial nerve VIII. The significance of an abnormally decreased phase lead is still not fully understood. Low phase leads may suggest a lesion in the nodulus region of the cerebellum, an area that influences the velocity storage integrator (Waespe et al., 1985), but they have also been reported in individuals with migraine-related dizziness and those with a history of motion sensitivity (Brey et al., 2008). Figure 21.11 shows a plot of phase angle as a function of frequency of rotation in a subject with normal SHA findings.

Velocity Step Testing

A second type of rotational method in common clinical practice is the velocity step or impulse acceleration test. For this test, the patient is seated in complete darkness (vision denied), head tipped forward-down 30 degrees, and secured in the same manner as the SHA protocol. The subject is quickly accelerated or "stepped" up to a constant velocity between 60 and 240 degree/s with an impulse acceleration of approximately 100 degree/s^2. Once the desired velocity has been obtained, the rotation continues for a period of 45 to 60 seconds at that speed. The VOR response to the initial acceleration is known as per-rotary nystagmus and the slow-component peak eye velocity (gain) is recorded. Over time, the per-rotary nystagmus will begin to decay and the subject will falsely perceive a slowing of the chair. The time (in seconds) at which the nystagmus SPV component decays to 37% of its original peak value is also recorded and is known as the vestibular time constant. Time constants are parameters that characterize the timing relationship between head movement and subsequent eye movement. After the constant velocity interval of 45 to 60 seconds has expired, the chair is stopped, using a rapid deceleration of equal magnitude to initial acceleratory impulse. Although the chair is now stationary, the subject will likely perceive motion in the opposite direction. The post-rotary VOR response will elicit nystagmus beating in the direction opposite to that of the initial acceleration and is known as the post-rotary nystagmus. The gain and time constant of the post-rotary period are measured in the same manner as the per-rotary period. Therefore, there will be two gain and two time constant measurements for a single rotation. After sufficient recovery time, the entire process is then repeated in the opposite direction so that one trial is performed in the clockwise or rightward direction and one in the counterclockwise or leftward direction. Clockwise acceleration stimulates the right horizontal canal and inhibits the left during the per-rotary phase and stimulates the left horizontal canal and inhibits the right during the post-rotary phase. The opposite is true for counterclockwise rotations.

Gain and time constant values obtained from the step protocol are analyzed and compared against clinical or manufacturer norms. Normal gain values typically fall within the range of 0.4 and 0.7 and normal time constants are generally greater than 10 seconds. Abnormally decreased time constants, particularly for 60 degree/s stimuli, may suggest unilateral, bilateral, or central vestibular deficits. Asymmetric or reduced gain values, particularly for 240 degree/s stimuli, may suggest unilateral peripheral vestibular deficits. For uncompensated peripheral lesions, both low and higher velocity step tests may demonstrate asymmetric gain values. It is also important to note that rotational step tests are heavily influenced by noise in the recording or physiologic systems and by the arousal of the patient prior to the acceleration because averaging is not used in this rotational paradigm. As a result, there will be patients for whom the estimates of time constant from the two protocols do not agree. Ideally, the step test and the sinusoidal acceleration tests can be used in parallel to increase the accuracy of estimates of the system time constant, individual periphery gains from the step procedure, and overall gain from the sinusoidal protocol, with possible asymmetrical peripheral responsiveness.

Visual–Vestibular Interaction

Up to this point, all rotational chair studies discussed have been performed in complete darkness (i.e., vision denied) to ensure that only the VOR was being evaluated, without interference from the visual system and without possibility of central VOR suppression. The introduction of visual stimuli to the standard rotary evaluation allows the clinician to make judgments about the central vestibular–ocular relationship and may be useful in identifying patients with migraine-related dizziness, traumatic brain injury, and various central vestibular pathologies. There are two common visual–vestibular studies performed within the balance laboratory: Visual fixation (VFX) and visually enhanced vestibulo-ocular reflex (VVOR) tests.

The VOR suppression test or VFX evaluates a patient's ability to suppress vestibular-induced nystagmus by means of visual fixation. It is somewhat analogous to the fixation suppression (fixation index) that is assessed during caloric irrigations following measurement of peak SPV caloric-induced nystagmus. The patient is seated in the rotary chair, head tipped forward-down 30 degrees, and secured in the same manner as before, but this time with the goggles open rather than closed (vision allowed). Additionally, there is also the introduction of a visual target, typically a laser line or dot that is projected from the chair onto the enclosure or wall in front of the subject. The subject is asked to fixate visually on the target while the chair rotates sinusoidally back and forth. The target travels at the same speed of the chair thereby always remaining in front of the participant. The patient with normal vestibular function should be able to maintain fixation on the lighted target reducing or eliminating any vestibular-induced nystagmus. A normal score typically results in at least 90% suppression of the VOR gain. The patient with abnormal vestibular function will be unable to suppress the vestibular-induced nystagmus and it will persist in large quantities. Failure to suppress (fixate) is a central sign typically associated with cerebellar dysfunction.

The VVOR test evaluates a patient's ability to integrate the visual pursuit and VOR systems effectively. This is the most "real-world" rotational test as it is the manner in which the vestibular system typically operates, combining visual and peripheral sensory inputs. The patient is secured in the same fashion as in the VFX test but this time an OPK stimulus is projected onto the enclosure around or wall in front of the subject. Unlike OPK testing performed during routine ENG/VNG, this OPK stimulus is held fixed (nonmoving). The movement is supplied by the chair which is rotated sinusoidally back and forth, typically at frequencies in the 0.16- to 0.64-Hz range. The goal of the VVOR test is to effectively match eye speed to the speed of rotation while viewing the OPK stimulus. A normal patient will be able to accomplish this with eye speed approaching a gain of 1. Abnormal patients may show different patterns based on pathology. If a patient exhibits abnormally low gain during SHA testing but normal gain during VVOR, it implies that the patient is able to compensate for any defective VOR with voluntary pursuit and thus the central vestibular system is likely intact and the patient has a peripheral hypofunction. If, however, both SHA gain and VVOR gain are low at the same frequency, it is suggestive of central dysfunction most likely affecting the cerebellum or brainstem (Furman and Cass, 1996). More recent investigations using the VVOR protocol have also been used to help identify those with traumatic brain injury or migraine-related dizziness. For example, Arriaga et al. (2005) reported that 71% of patients diagnosed with migraine vestibulopathy had elevated VVOR gain, whereas only 5% of a control group had similar gain. VVOR appears to be the least sensitive of all the rotational tests but it can nevertheless assist in differential diagnosis because its failure demonstrates an inability of the CNS to successfully integrate visual and vestibular information effectively.

Otolith Function Testing

Despite the growing body of knowledge regarding the function of the vestibular labyrinth, clinical assessment of each vestibular end-organ is still somewhat incomplete. The most commonly used laboratory tests in clinical practice today such as caloric irrigation, rotational chair, and head impulse are based predominantly on lateral semicircular canal function. Research of otolith responses over the past three decades has employed different methods of stimulation including off-vertical axis rotations (OVAR), linear track, and parallel swing devices. Only recently have

tools for the investigation of the utricle and saccule begun to appear in more frequent clinical use. Since this area of investigation is still emerging in routine clinical use, the full extent and clinical utilization is not yet entirely established. For organizational simplicity, the two otolith organs will be considered separately though there is presumably some physiologic overlap.

Saccular Evaluation

The primary method for evaluating saccular and inferior vestibular nerve function is by means of the vestibular-evoked myogenic potential (VEMP). VEMPs are short-latency electromyograms (EMGs) produced during application of high-intensity acoustic stimuli recorded via surface electrodes over contracted muscles. Saccular neurons respond not only to linear acceleration but also to sound pressure waves. This latter phenomenon allows for clinical investigation of the saccule by way of the vestibulocollic pathway. When a high-intensity acoustic stimulus is applied to the ear, there is a transient inhibition in tonic muscle activity ipsilateral to the side of stimulation. This temporary release from contraction can be measured via EMG on a commercially available evoked potential unit, allowing for assessment of each saccule independently. Responses can be elicited from different muscle groups but are typically recorded from the sternocleidomastoid (SCM) muscles of the neck and, when performed in this manner, are commonly referred to as cervical VEMPs (cVEMP).

There is currently no standard protocol for VEMP acquisition (Cheng et al., 2003; Li et al., 1999). Generally, VEMPs are obtained using a two-channel recording with the noninverting electrodes placed over the midpoint of the SCM muscles, inverting electrodes placed at the sternoclavicular junctions, chin or dorsum of the hand and a ground electrode placed on the forehead. A single polarity click or low-frequency tone burst at or above 90 dB nHL is delivered monaurally as the stimulus. Sustained contraction of the SCM is necessary to elicit a VEMP response so physical contribution from the patient is required. The SCM ipsilateral to the stimulated saccule must be forced into contraction by having the patient either lift or turn his/her head during stimulus presentation. It is important that the patient maintain sufficient EMG activity of the SCM throughout the test. VEMP responses are proportional to both stimulus level and the level of SCM activity so EMG monitoring with a target range of 30 to 50 mV is a recommended best practice (Akin et al., 2004).

The cVEMP waveform is a biphasic electrical response representing a release from muscle contraction temporally synchronized to the acoustic stimulus presentation. The response markers used to describe the waveform include peaks P13 (designated P1) and N23 (designated N1) which indicate the first positive and negative peaks of the response and their corresponding latencies (Figure 21.12). The

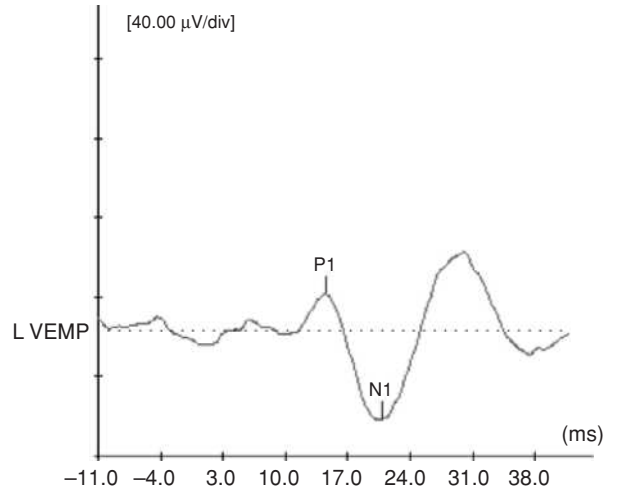


FIGURE 21.12 Normal cVEMP response elicited via air conduction at 95 dB nHL with a 500-Hz tone burst, showing P1 and N1 peaks with their respective latencies. [Courtesy of A.T. Still University, AFA Balance and Hearing Institute.]

response characteristics used to interpret the waveforms include P1–N1 latency, P1–N1 amplitude, threshold, and interaural asymmetry ratio. The cVEMP response is dependent on intact saccular and vestibular nerve function. The cVEMP has been found to be absent in cases of vestibular nerve section, but independent of cochlear function, as it is preserved in subjects with severe to profound sensory/neural hearing loss (Colebtach and Halmagyi, 1992). The VEMP is independent of cochlear function because it is the sound-sensitive neurons of the saccule, not the cochlea, that participate in the response. It should be noted that this holds true only for sensory/neural hearing losses since conductive pathologies can significantly reduce or eliminate VEMP responses because of the reduction in stimulus sound pressure ultimately reaching the inner ear.

The diagnostic utility of the VEMP is being investigated for a wide range of audio-vestibular and neurologic disorders. Response characteristics vary by pathology but certain patterns have begun to be identified. Absent responses and interaural amplitude differences tend to be the most common abnormalities described in vestibular disorders such as vestibular neuritis, vestibular schwannoma, and endolymphatic hydrops. Prolonged latencies have been described in those with central pathologies such as multiple sclerosis (Alpini et al., 2005) and brainstem lesion (Itoh et al., 2001). Of greatest use to date, however, has been the discovery of abnormally low thresholds in patients with superior semicircular canal dehiscence, large vestibular aqueduct syndrome, and Tullio phenomenon (Minor, 2005; Sheykoholeslami et al., 2004).

In recent years, several investigators have described a VEMP response recorded from the extra-ocular muscles of the eye by activating the otoliths with acoustic or vibratory stimuli. This response, termed the ocular VEMP (oVEMP),

requires the patient to sit quietly and fix his/her gaze upward on a stationary visual target. In contrast to the cervical VEMP, which is an ipsilateral inhibitory response, oVEMPs appear to reflect a predominantly contralateral excitation of muscle activity. The oVEMP response characteristics are similar to those of the cVEMP in several ways but the exact anatomic origins are still somewhat debated. Mounting research suggests that oVEMPs may in fact reflect utricular activity thereby providing a means to clinically assess both otolithic organs independently, via the cVEMP and oVEMP measures.

Utricular Evaluation

The primary clinical method at this time for evaluating the function of the utricle is a test of subjective visual vertical (SVV). SVV is a psychophysical measure determined by having the patient adjust a vertical line or light array to what he/she perceives to be perfectly vertical (Böhmer and Mast, 1999; Friedmann, 1970). SVV may be obtained while the patient is seated upright and motionless or while exposed to rotational accelerative stimuli (per-rotary or dynamic SVV). The principle underlying SVV is that an individual's perception of verticality and ability to adjust a visual target to true vertical is because of the detection of the pull of gravity, primarily via the utricles. If a pathologic insult disrupts the peripheral functioning of the utricular organ or the central utricular pathways, a resulting change in the position of the eye in regards to the true horizontal occurs, along with a static ocular counter roll. During an acute insult, an individual may adjust the SVV line off the true vertical as much as 21 degrees, tilted in the same direction as the affected side and ocular counter roll (Böhmer and Rickenmann, 1995). However, as the acute phase of the lesion subsides and becomes more chronic, the individual's performance quickly returns to normal (Vibert et al., 1999). Rotational chair paradigms have been experimented with in recent years in an attempt to allow for investigation of the utricular system under more chronic conditions. This has led to the implementation of eccentric per-rotary SVV protocols. An eccentric displacement paradigm where the patient estimates SVV while exposed to centripetal acceleration, commonly referred to as an off-axis rotation or unilateral centrifugation, was first introduced by Wetzig et al. (1990). For individuals with normal vestibular function, the off-axis SVV tilts symmetrically during unilateral centrifugation. That is, when the subject is placed off-axis to the right, the SVV is tilted toward the left; and when placed off-axis to the left, the SVV is tilted toward the right. Patients with chronic unilateral utricular loss exhibit an SVV asymmetry when measured during unilateral centrifugation. When the lesioned ear is placed off-axis, the SVV does not shift because the utricle does not respond to the gravito-inertial force. Because of the relative newness of this procedure, clinical protocols and normative data are not yet widely distributed.

Computerized Dynamic Posturography

A large percentage of patients that are seen in the balance laboratory require some assessment of postural control. Postural stability can be evaluated in a number of different ways each with its own unique methods and equipment. Because of the breadth of this topic, we will limit our discussion here to one of the most common formal assessment tools utilized in balance clinics today: Computerized dynamic posturography (CDP) as devised by EquiTest®. In general, CDP utilizes dynamic forceplates to detect the horizontal and vertical forces exerted by a subject's feet on the platform on which he/she is standing. The forceplates have the ability to rotate up or down or to translate forward or backward to provoke movement from the subject's center of mass. Rotation can also be initiated by the individual's own anterior–posterior body sway. The forceplates are supplemented by a moveable visual surround which can similarly be made to move either independently or as a result of the subject's movement. The CDP system collects and analyzes center of gravity and sway responses and provides numeric data which can then be compared with established norms. Posturography is not a diagnostic site-of-lesion test but is useful in assessing functional abilities and often as an adjunct in the design and monitoring of vestibular and balance rehabilitation programs. CDP may also be beneficial in the identification of patients who are exaggerating their functional abilities. Generally speaking, CDP consists of three distinct assessment protocols: The sensory organization test (SOT), the motor control test, and the adaptation test (ADT), each described in short below.

Sensory Organization Test

The SOT measures a subject's postural responses to a variety of visual, vestibular, and somatosensory altered conditions by means of patient-initiated floor and visual surround movements. The test consists of six specific conditions following a pattern of progressively more difficult scenarios accomplished by reducing or distorting information used for the maintenance of balance (Figure 21.13). The first three conditions offer uninterrupted, accurate, foot support surface information but with different visual inputs. Condition 1 is performed with eyes open, whereas in condition 2, the eyes are closed. Under condition 3, the eyes are open but the visual surround moves synchronously with the anterior–posterior sway movements of the patient. Condition 3 therefore presents a situation of visual conflict, where visual information is of no significant help in maintaining postural control. Conditions 4, 5, and 6 use the same sequence of visual conditions but with the foot support surface now giving misleading information. As with the movement of the visual surround in condition 3, when testing under conditions 4, 5, and 6, sway movements of the patient in the anterior–posterior

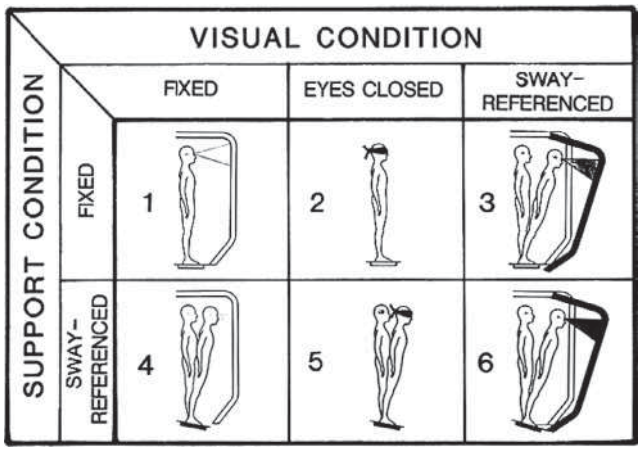


FIGURE 21.13 Six test conditions for the sensory organization portion of dynamic posturography. In the first three conditions, accurate foot somatosensory cues are available to the patient in all of the tests. The first and second conditions are simply eyes open and eyes closed. Condition 3 provides for orientationally inaccurate visual information in that, if the patient sways anterior–posterior, the visual surround moves with the patient (sway referenced). In conditions 4, 5, and 6, inaccurate foot somatosensory cues are provided by tilting the platform equal to the patient's sway in the sagittal plane (sway referenced). Then, for each of these latter three conditions, eyes open, eyes closed, and sway-referenced visual surround are presented, respectively. (From NeuroCom Int., Inc. Instruction Manual, with permission.)

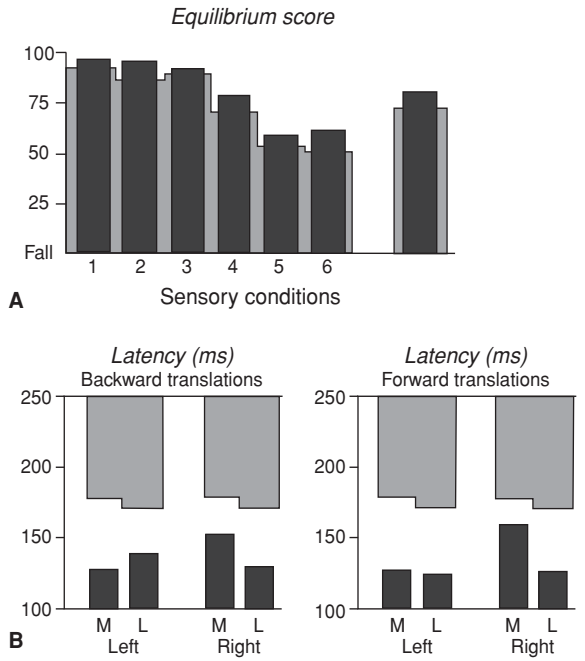


FIGURE 21.14 Results of dynamic posturography testing in a patient with all results interpreted as normal. The bar graph in the top panel (**A**) plots a percentage equilibrium score for each of the six SOT conditions (see Fig. 21.13). A score of 100 indicates no sway in the sagittal plane and a 'Fall' indicates that sway reached a magnitude equal to the theoretical limits of sway for the patient in the sagittal plane. The composite bar on the far right shows the numerical average of scores from the six conditions. The bar graphs in the bottom panel (**B**) show the motor control test results for latency to onset of recovery scores for medium and large forward and backward translations. The latencies are given in milliseconds and shown by the black bars for left leg and right leg for both sizes of platform translations. (Reprinted from NeuroCom Int., with permission.)

plane drive the movement of the support surface. In this way, somatosensory information is of limited use in maintaining balance. Typically, three trials are performed for each condition and the average performance is taken as representative of the patient's postural control ability under that sensory condition. A composite equilibrium score for all trials of all conditions is also generated. Figure 21.14A shows an example of the graphical representation of these results in a patient with normal CDP findings.

In addition to the quantitative equilibrium score, CDP studies are also interpreted using pattern recognition. Abnormal scoring on any of the six conditions is grouped to define a pattern of instability that can be functionally interpreted. Table 21.4 presents the most frequent patterns and a commonly used nomenclature. By far, the most common pattern is the vestibular dysfunction pattern. It is important to recognize, however, that SOT only provides information regarding which sensory system cues the patient is unable to utilize for maintenance of postural control. In other words, it provides a relative measure of the patient's ability to utilize the sensory input cues of vision, vestibular, and somatosensation to maintain balance, but does not provide relative information as to which of the sensory systems has lesions, causing postural control abnormalities. Therefore, SOT information should be interpreted only to reflect which input information the patient is able, or conversely unable, to use for the task at hand.

Motor Control Test

The motor control test (MCT) provides information about patients' ability to react to unexpected displacement of their center of mass. The unexpected displacement is created by abrupt horizontal translations of the support surface on which they are standing. Typically, three small, medium, and large translations are presented in increasing magnitude for both the forward and backward directions. When the support surface translates unexpectedly in this manner, the body center of mass remains approximately stationary becoming offset to the base of support. Rapid automatic postural corrections are then necessary to restabilize the body and prevent a fall. The postural responses are quantified and analyzed based

TABLE 21.4

Abnormalities of Sensory Organization Testing

Pattern	Abnormality	Description
Vestibular dysfunction pattern	Abnormal on conditions 5 and 6 (alternatively condition 5 alone)	Vestibular dysfunction pattern indicates the patient's difficulty in using vestibular information alone for maintenance of stance. When provided with accurate visual and/or foot somatosensory information, stance is within a normal range
Visual vestibular dysfunction pattern	Abnormal on conditions 4, 5, and 6	Visual and vestibular dysfunction pattern indicates the patient's difficulty in using accurate visual information with vestibular information or vestibular information alone for maintenance of stance. When provided with accurate foot support surface cues, stance is within a normal range
Visual preference pattern	Abnormal on conditions 3 and 6 (alternatively condition 6 alone)	Visual preference pattern indicates the patient's abnormal reliance on visual information, even when inaccurate. When provided with accurate foot support surface information together with accurate or absent visual cues or absent vision and vestibular information alone, stance is within a normal range
Visual preference/ vestibular dysfunction pattern	Abnormal on conditions 3, 5, and 6	Visual preference and vestibular dysfunction pattern indicate the patient's difficulty in using vestibular information alone and the patient's abnormal reliance on visual information, even when inaccurate. When provided with accurate foot support surface information together with accurate or absent visual cues, stance is within a normal range
Somatosensory/vestibular dysfunction pattern	Abnormal on conditions 2, 3, 5, and 6	Somatosensory and vestibular dysfunction pattern indicates the patient's difficulty in using foot support surface information with vestibular information or vestibular information alone for maintenance of stance. When provided with accurate visual information, stance is within a normal range
Somatosensory/vestibular dysfunction pattern	Abnormal on conditions 2, 3, 5, and 6	Somatosensory and vestibular dysfunction pattern indicates the patient's difficulty in using foot support surface information with vestibular information or vestibular information alone for maintenance of stance. When provided with accurate visual information, stance is within a normal range
Severe dysfunction pattern	Abnormal on four or more conditions not covered in the above descriptions, for example, conditions 3, 4, 5, and 6; or 2, 3, 4, 5, and 6; or 1, 2, 3, 4, 5, and 6	Severe dysfunction pattern indicates the patient's difficulty with stance independent of the sensory information (vestibular, visual, and/or somatosensory) provided. Note that these situations many times involve a dominant feature such as significantly abnormal conditions 5 and 6 or they may involve equally distributed difficulties on all conditions affected
Inconsistent pattern	Abnormal on conditions 1, 2, 3, or 4, or any combination and normal on conditions 5 and 6	Inconsistent pattern indicates that performance of the patient is difficult to explain with normal or typical pathophysiologic conditions and could imply volitional or nonvolitional exaggerated results

on latency of onset of translation to that of active recovery. Other information commonly gathered from the motor control protocol includes abnormal weight bearing and inability to properly scale the strength of the postural responses to the increasing size of the stimulus.

The MCT protocol is used primarily to evaluate the long-loop pathways of the body. The long-loop pathways begin with the stretch receptors in the lower limbs, project to the motor cortex, and then are relayed to the upper and lower body muscles involved in maintenance of balance. When abnormal latencies from unexpectedly induced sway are noted, then problems in the long-loop pathway should be considered. Prolonged latencies are a relatively nonspecific finding, however, in that they may indicate an abnormality of the afferent or efferent neural pathways but they can also be seen in individuals with various somatosensory disorders and musculoskeletal conditions (Shepard et al., 1993). Figure 21.14B shows an example of the graphical representation from a patient with normal MCT latency findings.

Adaptation Test

The ADT evaluates a patient's ability to adapt to a familiar stimulus specifically, unexpected rotations about the ankle. Five rapid toes-up or toes-down translations are presented to the subject with the expectation that reaction scores should improve on successive trials. For this protocol, as with the SOT protocol, reaction forces detected by the force plates in the foot support surface are measured. The principal parameter examined is the latency from onset of unexpected translation to that of active recovery. Individuals who show poor adaptation over successive trials are likely to be at increased risk for falls.

FUTURE DIRECTIONS

Assessment of patients with dizziness and balance disorders has undergone significant change over the past decade. The introduction of newer and more reliable tests of otolithic function, such as the cervical and ocular VEMP and subjective visual vertical test, has allowed us to expand our clinical investigation of the vestibular apparatus into areas that were previously unattainable. In addition, new commercially available computerized head impulse and DVA systems have increased our ability to objectively quantify VOR function. In fact, when these measures are performed in combination, we now have the ability to clinically assess all three semicircular canals, both otolithic structures, and both branches of the vestibular nerve in each ear, independently. This allows for a more robust functional assessment of the labyrinth as a whole, resulting in better therapeutic guidance and improved outcomes for patients.

Advancing knowledge with concomitant technologies comes at an opportune time, as vestibular assessment is also becoming increasingly more common in patient populations who may not have previously been evaluated. This includes those populations with suspected traumatic brain injury and those with suspected stroke. Several position papers and consensus statements have been released in recent years by groups such as the American Academy of Neurology and the National Athletic Trainers' Association recommending evaluation of balance and vestibular function following concussive head injury. Recent research by Newman-Toker et al. (2013) has suggested that head impulse testing, in conjunction with gaze nystagmus assessment and prism cross-cover tests of ocular alignment, may be more sensitive than MRI for detecting early vertebrobasilar stroke.

Each year, approximately 8 million physician and emergency room visits are attributed to complaints of dizziness and imbalance. When considering future directions in assessment of patients with dizziness and balance disorders; growth in demand, advances in scientific evidence and technology, as well as the impact of healthcare and wellness initiatives will continue to play a role in making this an important area for audiology education and clinical services.

Case studies can be found on thePoint at http://thePoint.lww.com.

FOOD FOR THOUGHT

1. Virtually all newborns in the United States are now screened for hearing loss before leaving the hospital. CDC's National Goals for EHDI Programs still recommend that "hospitals and others [report] information about risk factors for hearing loss to the state, who will monitor the status of children with risk factors and provide appropriate follow-up services." What are the pros and cons of continuing to monitor the status of children with risk factors with respect to issues such as identifying childhood hearing loss, costs, demands on the health care system, and burden for families?
2. Although the percentage of children failing a newborn hearing screening test who are lost to follow-up and or documentation is slowly declining, it remains a very significant issue. What approaches, programs, or initiatives are likely to significantly reduce the percentage of children being lost to follow-up?
3. There continues to be a critical shortage of audiologists who have the expertise, experience, and desire to provide comprehensive audiological services to infants and young children. What can be done to increase the number of fully qualified pediatric audiologists?

REFERENCES

Akin FW, Murnane OD, Panus PC, Caruthers SK, Wilkinson AE, Proffit TM. (2004) The influence of voluntary tonic EMG level on the vestibular evoked myogenic potential. *J Rehabil Res Dev.* 41 (3B), 473–480.

Alpini D, Pugnetti L, Caputo D, Cesarani A. (2005) Vestibular evoked myogenic potentials in multiple sclerosis: a comparison between onset and definite cases. *Int Tinnitus J.* 11 (1), 48–51.

Arriaga MA, Chen DA, Cenci KA. (2005) Rotational chair (ROTO) instead of electronystagmography (ENG) as the primary vestibular test. *Otolaryngol Head Neck Surg.* 133, 329–333.

Aw ST, Haslwanter T, Halmagyi GM, Curthoys IS, Yavor RA, Todd MJ. (1996) Three-dimensional vector analysis of the human vestibuloocular reflex in response to high-acceleration head rotations. I. Responses in normal subjects. *J Neurophysiol.* 76, 4009–4020.

Baloh RH, Kerber KA. (2011) The history of the dizzy patient. In: Baloh RW, Kerber KA, eds. *Clinical Neurophysiology of the Vestibular System.* New York: Oxford University Press; pp 127–147.

Barber HO, Stockwell CW. (1980) *Manual of Electronystagmography.* 2nd ed. St. Louis, MO: C.V. Mosby.

Bohmer A, Mast F. (1999) Chronic unilateral loss of otolith function revealed by the subjective visual vertical during off center yaw rotation. *J Vestib Res.* 9, 413–422.

Bohmer A, Rickenmann J. (1995) The subjective visual vertical as a clinical parameter of vestibular function in peripheral vestibular diseases. *J Vestib Res.* 5, 35–45.

Brey RH, McPherson JH, Lynch RM. (2008) Background and introduction to whole body rotational testing. In: Jacobson GP, Shepard NT, eds. *Balance Function Assessment and Management.* San Diego, CA: Plural Publishing Inc.; pp 253–280.

Chee NW, Tong HM. (2002) Acoustic neuroma presenting as exercise-induced vertigo. *J Laryngol Otol.* 116 (8), 630–632.

Cheng PW, Huang TW, Young YH. (2003) The influence of clicks versus short tone bursts on the vestibular evoked myogenic potentials. *Ear Hear.* 24, 195–197.

Choi KD, Kim JS, Kim HJ, Koo JW, Kim JH, Kim CY, et al. (2007) Hyperventilation-induced nystagmus in peripheral vestibulopathy and cerebellopontine angle tumor. *Neurology.* 69, 1050–1059.

Colebatch JG, Halmagyi GM. (1992) Vestibular evoked potentials in human neck muscles before and after unilateral vestibular deafferentation. *Neurology.* 42, 1635–1636.

Davies R. (2004) Bedside neuro-otological examination and interpretation of commonly used investigations. *J Neurol Neurosurg Psychiatry.* 75 (suppl 4), 32–44.

Fife TD, Tusa RJ, Furman JM, Zee DS, Frohman E, Baloh RW, et al. (2000) Assessment: vestibular testing techniques in adults and children, report of the Therapeutics and Technology Assessment Subcommittee of the American Academy of Neurology. *Neurology.* 55, 1431–1441.

Friedmann G. (1970) The judgment of the visual vertical and horizontal with peripheral and central vestibular lesions. *Brain.* 93, 313–328.

Fukuda T. (1959) The stepping test: two phases of the labyrinthine reflex. *Acta Otolaryngol.* 50 (2), 95–108.

Furman JM, Cass SP. (1996) *Balance Disorders: A Case-Study Approach.* Philadelphia, PA: FA Davis Co.

Furman JM, Cass SP. (2003) *Vestibular Disorders: A Case-Study Approach.* 2nd ed. New York: Oxford University Press.

Gillespie LD, Robertson MC, Gillespie WJ, Sherrington C, Gates S, Clemson LM, et al. (2013) Interventions for preventing falls in older people living in the community [Review]. *Cochrane Database Syst Rev.* (9), CD007146.

Grossman GE, Leigh RJ, Bruce EN, Huebner WP, Lanska, DJ. (1989) Performance of the human vestibuloocular reflex during locomotion. *J Neurophysiol.* 62 (1), 264–272.

Guidetti G, Monzani D, Rovatti V. (2006) Clinical examination of labyrinthine-defective patients out of the vertigo attack: sensitivity and specificity of three low-cost methods. *Acta Otorhinolaryngol Ital.* 26 (2), 96–101.

Hain TC, Fetter M, Zee DS. (1987) Head-shaking nystagmus in patients with unilateral peripheral vestibular lesions. *Am J Otolaryngol.* 8, 36–37.

Halmagyi GM, Curthoys IS. (1988) A clinical sign of canal paresis. *Arch Neurol.* 45, 737–739.

Honaker JA, Shepard NT. (2012) Performance of Fukuda stepping test as a function of the severity of caloric weakness in chronic dizzy patients. *J Am Acad Audiol.* 23 (8), 616–622.

Itoh A, Kim YS, Yoshioka K, Kanaya M, Enomoto H, Hiraiwa F, et al. (2001) Clinical study of vestibular evoked myogenic potentials and auditory brainstem responses in patients with brainstem lesions. *Acta Otolaryngol Suppl.* 545, 116–119.

Jacobson GP, Newman CW. (1990) The development of the dizziness handicap inventory. *Arch Otolaryngol Head Neck Surg.* 116, 424–427.

Li MW, Houlden D, Tomlinson RD. (1999) Click evoked EMG responses in sternocleidomastoid muscles: characteristics in normal subjects. *J Vestib Res.* 9, 327–334.

Lin HW, Bhattacharyya N. (2012) Balance disorders in the elderly: epidemiology and functional impact. *Laryngoscope.* 122, 1858–1861.

Longridge NS, Mallinson AI. (1984) A discussion of the dynamic illegible E test: a new method of screening for aminoglycoside vestibulotoxicity. *Otolaryngol Head Neck Surg.* 92, 671–676.

Minor LB. (2005) Clinical manifestations of superior semicircular canal dehiscence. *Laryngoscope.* 115, 1717–1727.

Minor LB, Haslwanter T, Straumann D, Zee DS. (1999) Hyperventilation-induced nystagmus in patients with vestibular schwannoma. *Neurology.* 53, 2158–2167.

Newman-Toker DE, Saber-Tehrani AS, Mantokoudis G, Pula JH, Guede, CI, Kerber KA, et al. (2013) Quantitative video-oculography to help diagnose stroke in acute vertigo and dizziness: toward an ECG for the eyes. *Stroke.* 44 (4), 1158–1161.

NIH News in Health. (2012) Dizziness can be a drag: coping with balance disorders. Available online at: http://newsinhealth.nih.gov/issue/aug2012/feature1. Accessed May 10, 2013.

Perez N, Rama-Lopez I. (2003) Head-impulse and caloric tests in patients with dizziness. *Otol Neurotol.* 24, 913–917.

Ruckenstein M, Shepard NT. (2000) Balance function testing: a rationale approach. In: Shepard NT, Solomon D, eds. *The Otolaryngologic Clinics of North America.* Philadelphia, PA: WB Saunders.

Schubert MC, Tusa RJ, Grine LE, Herdman SJ. (2004) Optimizing the sensitivity of the head thrust test for identifying vestibular hypofunction. *Phys Ther.* 84, 151–158.

Shepard NT, Schultz A, Alexander NB, Gu MJ, Boismier T. (1993) Postural control in young and elderly adults when stance is challenged: clinical versus laboratory measurements. *Ann Otol Rhinol Laryngol.* 102, 508–517.

Shepard NT, Telian SA. (1996) *Practical Management of the Balance Disorder Patient.* San Diego, CA: Singular Publishing Group.

Sheykoholeslami K, Schmerber S, Kermany MH, Kaga K. (2004) Vestibular-evoked myogenic potentials in three patients with large vestibular aqueduct. *Hear Res.* 190 (1–2), 161–168.

Sowerby LJ, Rotenberg B, Brine M, George CFP, Parnes LS. (2010) Sleep apnea, daytime somnolence, and idiopathic dizziness - a novel association. *Laryngoscope.* 120, 1274–1278.

Vibert D, Hausler R, Safran AB. (1999) Subjective visual vertical in peripheral unilateral vestibular diseases. *J Vestib Res.* 9, 145–152.

Waespe W, Cohen B, Raphan T. (1985) Dynamic modification of the vestibulo–ocular reflex by the nodulus and uvula. *Science.* 228, 199–202.

Walker MF, Zee DS. (2000) Bedside vestibular examination. In: Shepard NT, Solomon D, eds. *Otolaryngologic Clinics of North America.* Philadelphia, PA: WB Saunders.

Wetzig J, Wetzig J, Reiser M, Martin E, Bregenzer N, von Baumgarten RJ. (1990) Unilateral centrifugation of the otoliths as a new method to determine bilateral asymmetries of the otolith apparatus in man. *Acta Astronaut.* 21 (6–7), 519–525.

Yardley L, Masson E, Verschurr C, Haacke N, Luxon L. (1992) Symptoms, anxiety and handicap in dizzy patients: development of the vertigo symptom scale. *J Psychosom Res.* 36, 731–741.

Zee DS, Fletcher WA. (1996) Bedside examination. In: Baloh RW, Halmagyi GM, eds. *Disorders of the Vestibular System.* New York: Oxford University Press; pp 178–190.

CHAPTER 22

Vestibular Rehabilitative Therapy

Richard Gans

INTRODUCTION

The National Institutes of Health (NIH) (2009) estimates that 40% of the US population will experience an episode of dizziness severe enough to motivate patients to seek the attention of a physician. Vestibular disorders account for approximately 85% of these symptoms. This chapter will address current practices in treating these disorders, based on a thorough understanding of the materials found in Chapter 20 (Neurophysiology of the Vestibular System) and Chapter 21 (Evaluation of Dizziness and Balance Disorders).

The vestibular system is subject to insult, trauma, and disease (Table 22.1). The disorder or disease process may cause the vestibular system to suffer a reduction or loss of function in one or both labyrinths. Most otologic disorders, however, will typically affect only one labyrinth at a time. The damage or change may occur either as a loss of sensory receptors within the end organ or within the nerve itself. The sudden loss or reduction of function will typically produce debilitating vertigo with associated autonomic and parasympathetic nervous system responses of nausea, emesis (vomiting), and diaphoresis (profuse sweating), symptoms similar to those occurring with intense motion sickness. Fortunately, the acute phase of most conditions will pass within hours; however, other symptoms may linger for days and weeks or, as in the case of Meniere's, be problematic for years. During the acute phase of the condition, patients will require medical and pharmacologic management, usually preferring to stay quiet and immobilized until they recover their homeostasis and manage to move about without distress.

TABLE 22.1

Common Causes of Unilateral Vestibular Dysfunction

Autoimmune disorders
Labyrinthitis
Labyrinthine concussion
Labyrinthine ischemia
Meniere's disease
Ramsey Hunt syndrome
Vestibular migraine
Vestibular neuritis

STATUS OF VESTIBULAR DYSFUNCTION

As described in Chapter 20, patients with vestibular disorders may have had insult or damage to either the peripheral (labyrinth part of the inner ear) or central (brainstem or cerebellum) portions of the vestibular mechanism. Common inner ear disorders that cause vestibular dysfunction include labyrinthitis, vestibular neuritis, herpes zoster oticus (shingles), vestibular migraine, labyrinthine ischemia, and Meniere's disease. Many of these patients experience either a relatively short phase of acute vertigo or are post-surgery for treatment of intractable inner ear disease such as Meniere's disease. Once out of the acute phase, they may be left with chronic symptoms affecting their sense of spatial orientation, gaze stabilization, or balance.

Vestibular disorders affect three output modalities: (1) The vestibuloocular reflex (VOR), which controls eye movement and gaze stabilization during active head movement; (2) the vestibulospinal reflex (VSR), which influences postural stability, translated through the musculoskeletal system and antigravity muscles; and (3) the vestibulocollic reflex (VCR), responsible for signals from the otolithic gravity sensor and the neck muscle proprioceptors. Patients with vestibular disorders may present with defects of gaze stabilization problems during active head movement, exaggeration or hallucination of motion, or prolonged unsteadiness, usually when challenged by uneven surfaces, by quick turns, or with reduced vision.

For those with slow, insidious vestibular changes that gradually increase over years, patients will not experience vertigo, but rather a loss of equilibrium and increased unsteadiness while walking, a condition which often occurs in older adults or those with a variety of nonvestibular or nonotologic-related disease processes (Table 22.2). Individuals who have had bilateral vestibular losses secondary to aminoglyside toxicity usually present with an associated complaint of oscillopsia during head movement.

Vestibular rehabilitation therapy (VRT) has been shown to be a highly effective management strategy for

TABLE 22.2

Common Causes of Bilateral Vestibular Dysfunction

Aminoglysides
Arteriosclerosis
Diabetes
Microvascular disease

patients with chronic symptoms related to a labyrinthine event (Herdman, 2001). Numerous investigators have demonstrated the efficacy of VRT for various populations (Gans, 2013). To provide the most efficacious treatment possible, the clinician must understand the status of the disorder, beginning with a comprehensive interview and thorough case history. Information is needed about the onset of the initial event, any subsequent attacks as well as their duration (minutes vs. hours), and any associated symptoms during the attack or episode, that is, hearing loss or tinnitus.

Prior to the referral for and undertaking of VRT, it will be necessary to define and categorize the status of the vestibular involvement. The ideal candidate for VRT is a patient with stabilized, but noncompensated unilateral vestibular dysfunction (UVD), conditions which are described in the following section.

Stabilized versus Nonstabilized

A stabilized condition can best be described as one that is no longer producing attacks or episodes of debilitating vertigo and other otologic or parasympathetic responses, such as acute nausea and emesis. This group of symptoms has been described as the labyrinthine storm. Once the acute phase has passed, most conditions such as vestibular neuritis or labyrinthitis become stabilized. The anatomical and physiological damage to the system is typically caused by either a viral or bacterial infection of the labyrinthine fluids or a viral/bacterial inflammation of the vestibular portion of CN VIII. The patient may have symptoms related to the chronic VOR dysfunction, but no longer is subject to attacks.

Meniere's disease is perhaps the best example of a chronic nonstabilized vestibular disease that may persist in an active or agitated state for years. Another example of a nonstabilized disease is a nonoperable acoustic neuroma, or vestibular schwannoma. Since benign paroxysmal positional vertigo (BPPV) (see Chapters 20 and 21) is a biomechanical phenomenon, rather than a neurophysiological state, it does not fit into the categorization of a stabilized- versus nonstabilized-type lesion. BPPV is treated with a high level of success, 97% or greater in most patients using canalith repositioning maneuvers (CRM) once the affected ear and semicircular canal involvement are properly identified. A comprehensive presentation of the CRM protocols with step-by-step instructions may be found in Roberts and Gans (2008).

Compensated versus Noncompensated

The central nervous system (CNS) will, within days, weeks, or months, accommodate to the asymmetrical labyrinthine function and, without any external help, will reset or retune the VOR function. Central compensation is believed to occur through the plasticity of the CNS within the brainstem and cerebellum. It has been described as a neurophysiological motor relearning phenomenon and has been documented in the literature with animal and human models (Kramer et al., 1998; Lisberger, 1998).

Patients who have a spontaneous recovery of the VOR function subsequent to an acute vestibulopathy are considered to have a compensated lesion. Although these patients will continue to present with abnormal findings (i.e., reduced labyrinthine reactivity or caloric weakness on a VNG caloric; described in Chapter 21), they will have no subjective report of exaggerated sense of motion, oscillopsia, or visually provoked symptoms. On tests of VOR function such as vestibular autorotation testing, they will present with normal gain and phase, just as if they were otherwise normal. Tests of dynamic visual acuity (DVA, discussed later in this chapter) will appear normal, despite the caloric weakness.

The caveat to clinicians is that a reduced labyrinthine reactivity does not mean that it is the origin of the patient's complaints. Patients may have normal caloric responses because either the horizontal canal was not involved or the problem is in the higher frequency sensitivity of the system. The caloric test evaluates only the ultra low frequency (0.003 Hz) sensitivity of the horizontal semicircular canal, making it less than a comprehensive assessment of total end organ function, as noted in Chapter 21.

A noncompensated condition describes a patient who continues to have symptoms, regardless of nonfunctional test results. As the peripheral labyrinthine end organ system operates as a gravity and velocity detector, symptoms are typically produced with change in head or body position or active head movement, usually in a particular plane of movement or speed. Since compensation does not behave as an all-or-nothing phenomenon, patients may progress over time so that the nature of their present gravity- or movement-triggered complaints is different and has changed. They may be getting better, but are not completely better or normal in their ability to perform even simple everyday activities. It is not uncommon for them to report that "I am better than I was, but I still don't feel right."

Several factors may affect one's ability to compensate naturally over time. Most individuals who experience a vestibular event (e.g., vestibular neuritis) recover fully over a period of weeks without the need for VRT. The factors that may compromise or inhibit compensation include physical or psychologic dependence on antimotion drugs or sedatives such as valium, lack of movement and activity, and a predisposition to motion intolerance commonly seen within

families or among individuals with a history of migraine, which is also familial.

IDENTIFYING VRT CANDIDATES

Those patients who find their everyday function is adversely affected or limited by this stabilized, but noncompensated asymmetry in vestibular function are the ideal candidates for VRT. The vestibulopathy may affect the full range of acceleration, or frequency, or just regions of acceleration, similar to a frequency-specific hearing loss within the cochlea. Likewise, the direction of the acceleration may also be involved. Typically, the patient's symptoms will be more provoked with acceleration toward the involved or impaired labyrinth. The manifestation of a vestibulopathy will often result in a VOR deficiency. The VOR is responsible for stabilizing eye/head position at frequencies starting at about 0.6 Hz. The errors that occur in the VOR function may affect the gain or accuracy and the phase or timing of the reflex. Correct VOR function is dependent on the brain's ability to correctly signal the extraocular eye muscles to correspond their response with the initiating head movement. The hydromechanical movement of the fluid within the semicircular canals initiates this signal.

The inability of the eyes to be correctly positioned with active head movement causes a retinal slippage. This results in an oscillopsia, in which the image to be viewed appears to jump or jiggle. These patients often state, "My eyes feel as if they need new shock absorbers." It may be restricted to the plane of the involvement. The extraocular eye muscles are correlated with the specific plane of the balance canals.

VRT consists of systematic repetitive exercises and protocols that extinguish or ameliorate patients' motion-provoked symptoms, as well as enhancing postural stability and equilibrium. VRT is not new; it has reached past its half-century mark. Cawthorne (1944) and Cooksey (1946) both discuss the benefit of active eye and head movement exercise for patients who experienced labyrinthine problems. Since then, research and clinical experience have greatly advanced the scientific application of this treatment methodology. See Gans (2013) for a comprehensive summary of this research.

CLINICAL ASSESSMENT OF VESTIBULAR FUNCTION

Chapter 21 provides an in-depth review of the clinical assessment of vestibular function. For this chapter, please note that patient evaluation may include a variety of assessment tools that are specific to revealing VOR and VSR abnormalities, without the use of technology. These evaluation protocols may be used by audiologists, physicians, and physical and occupational therapists alike. Many are considered classic "bedside" protocols, or in some communities or emerging economies may be the only evaluation techniques available. Table 22.3 presents a review of standardized and easily performed tests shown to have high sensitivity in demonstrating noncompensated vestibulopathy as well as corresponding VRT diagnosis–based strategies. These may include tests of gaze stabilization and visual acuity with active head movement. Tests of VSR function, such as the modified Clinical Test of Sensory Integration of Balance (CTSIB) (Shumway-Cook and Horak, 1986), have shown excellent sensitivity for UVD patterns. Traditionally performed tests, such as VNG calorics, or less commonly performed rotary chair, may not reveal a high-frequency UVD. Patients who experience VOR problems at a higher frequency can often be identified with tests that disrupt visual acuity during active head movement (Roberts and Gans, 2007; Roberts et al., 2006; Schubert et al., 2002) or tests that quantify the VOR gain and phase (O'Leary and Davis-O'Leary, 1990).

DVA or gaze stabilization tests hold significant promise in simple and straightforward analysis of VOR function. All too often, patients with undetected UVD whose history and symptoms strongly correlate with a stabilized, but noncompensated UVD are dismissed as being a nonvestibular patient, as a result of an unremarkable caloric or VNG study. Tests of DVA have a wide range of application for both civilian and military populations. The military and NASA, specifically during the space shuttle years, were in the vanguard of research because of the concerns of astronauts flying with undiagnosed or untreated VOR problems (Hillman et al., 1999). DVA is also used in the identification and outcome measures with posttraumatic head trauma and concussion (Alsalaheen et al., 2010; Heitger et al., 2009).

Subjective Handicap Scales and Patient Reports

In addition to the invaluable data collected from a case history, patient rating scales are used to identify and quantify the functional disability created by physical ailment or illness. Disease or activity-specific and global health status patient handicap scales provide a valuable resource for establishing baseline, as well as serial or outcome measures. Table 22.4 provides several scales used and clinically documented in the vestibular literature. The results of these self-reporting measures often provide excellent insight for clinicians in determining candidates for VRT or for those who may require further testing, as indicated by inclusion in a thorough clinical assessment. These clinimetrics (i.e., subjective patient rating scales) are standardized to measure a disease-specific disability or an overall quality of life.

PHYSIOLOGICAL BASIS OF VRT

The underlying physiological basis for VRT is the plasticity of the CNS. VRT does not actually involve a regeneration or treatment of the damaged vestibular end organ itself. Instead, it works by allowing the CNS and the brain to acclimate or adapt to asymmetrical/conflicting input from the

TABLE 22.3

Clinical Assessment of Vestibular Function

Evaluation	Sensitivity	Findings	Cause	VR Protocols
Clinical Test of Sensory Integration of Balance (CTSIB) (Shumway-Cook and Horak, 1986)	High	Fall on foam w/o vision	Inability of the weakened vestibular system to maintain postural stability with degraded somatosensory input and no visual input	Substitution protocols will reduce visual and/or surface dependence forcing vestibular input to be maximized
Head Thrust (Halmagyi et al., 1990/1991)	High	Corrective saccade in the direction of head acceleration	Asymmetrical labyrinthine input to the VOR	Adaptation including gaze stabilization. The abnormal corrective saccade will likely also degrade performance on Dynamic Visual Acuity Test
Bidirectional Full Visual Field (80%) Optokinetic (OKN) Test	Moderate	Reduced OKN when stimuli move toward involved ear	Asymmetrical vestibular-optokinetic reflex indicating a weakened labyrinthine input	Adaptation. May suggest visual-vestibular integration protocols be included–VRT protocols should create visual conflict
Dynamic Visual Acuity Test (Herdman, 2001; Roberts and Gans, 2008; Schubert et al., 2002)	High	Degraded visual acuity with active head movement horizontal and/or vertical in VOR frequency range	Abnormal VOR gain at test frequencies	Adaptation with emphasis on gaze stabilization protocols in the plane and frequency of the deficiency
Provoked Vertigo Test includes positioning (modified Hallpikes for PC-BPPV), lateral positions for HC-BPPV, and static symptoms w and w/o vision and lateral positions with a 20-s head shake (vision denied)	High	Positive for PC-BPPV and/or HC-BPPV Static positions provoked (w and w/o vision) nystagmus/symptoms–labyrinthine or CNS patterns	Patterns of BPPV with transient nystagmus/vertigo consistent with and correlated to head–ear–canal position	Canalith repositioning maneuver (CRM) as appropriate for ear, canal, and patient's biomechanical/physical needs
Dizziness Handicap Inventory (DHI) (Jacobson and Newman, 1990)	High	Used as the "gold standard" self-report clinimetric for the patient's subjective level of disability relative to noncompensated vestibulopathy	Scoring comprises three areas: Function, physical, and emotional as well as an overall score	Provides pre-therapy baseline and validation of outcomes at time of discharge. Also identifies aspects of psychologic overlay which may need to be addressed either during or following central compensation

TABLE 22.4

Subjective Handicap Instruments

Dizziness Handicap Inventory [DHI] [Jacobson and Newman, 1990]
Health Survey Questionnaire–SF-36 [Ware, 1988]
Meniere's Disease–Patient-Oriented Subjective Improvement [Gates, 2000]
Vestibular Disorder Activities of Daily Living Scale [Cohen, 2000]
Activities-Specific Balance Confidence Scale [Powell and Myers, 1995]

two vestibular systems. Possible mechanisms include the spontaneous rebalancing of the tonic activity within the vestibular nuclei, recovery of the VOR through adaptation, and the habituation effect (a lessening of response to the same stimuli over time). Theoretically, central compensation should occur within 90 days following dysfunction or loss of one of the vestibular systems. Many lesions, particularly those that occur with rapid onset, do not benefit from this compensation phenomenon.

Understandably, patients are often reluctant to perform therapeutic activities involving active head motions that produce symptoms of dizziness. This reluctance likely delays the development of central compensation. Motion intolerance or heightened motion sensitivity has been determined to be a genetic trait and is estimated to be a common aura seen in at least two-thirds of individuals with migraines (Selby and Lance, 1960). In essence, their central mechanisms are less likely to benefit from natural compensations because their "hard-wiring" is already less than desirable as a motion sensor. Simply put, they are not more likely to have a vestibular event, but if they do, they are less likely to compensate without assistance.

Another complicating factor includes the use of commonly prescribed drugs such as meclizine, antivert, valium, and other pharmaceuticals that suppress either peripheral vestibular or CNS function. These drugs will delay or prevent the CNS from relearning or adapting to asymmetrical sensory input. Unfortunately, the dizzy patient, in his/her heightened state of anxiety about becoming dizzy (especially while at work or driving), becomes reliant on pharmaceuticals that assist in suppressing distressing symptoms.

VRT is most effective when it is used with individuals who are no longer in the acute phase of a condition. The patient who is in the midst of a labyrinthine storm secondary to labyrinthitis, vestibular neuritis, or active Meniere's disease will receive little or no benefit from VRT. Ideally, patients will be in a stabilized condition when beginning VRT.

These patients present symptoms that are provoked by active head movement, often at a particular frequency of motion and in a particular direction. For instance, it may be intolerable to view numerous telephone poles while riding in an automobile. Commonly, patients express a sensation of motion sickness when looking at certain patterns of floor tiles or wall coverings. A common complaint is the difficulty in turning one's head from side-to-side while walking down an aisle at a grocery store.

DIAGNOSIS-BASED STRATEGIES

Several researchers (e.g., Black et al., 2000; Cohen, 1992; Shepard and Telian, 1995) have supported and promoted the application of specific rehabilitation strategies to specific functional disabilities. Many well-meaning practitioners continue to use the 60-year-old Cawthorne (1944) and 30-year-old Brandt-Daroff (1980) exercises for dizzy patients, regardless of the patient's diagnosis or condition. Clinical experience and contemporary research strongly indicate that the success of vestibular rehabilitation is related to applying the correct treatment methodology to the appropriate corresponding dysfunction. Table 22.5 identifies those appropriate treatment methodologies with their corresponding functional components.

Diagnosis-based strategies as an individualized or customized therapeutic approach have been shown to produce successful outcomes (Gans, 1996). These strategies link the underlying physiological changes that occurred because of the disease or insult with the patient's functional symptoms. There are three approaches to therapy: (1) Adaptation with subsets of gaze stabilization and habituation; (2) substitution; and (3) canalith repositioning maneuvers (CRM). These approaches may be used independently or in conjunction with one another, depending on the patient's needs.

Adaptation

Adaptation will reset or retune the VOR by repetitive activities. These activities will include those situations or movements that provoke the very symptoms the patient has been trying to avoid. Examples of the exercises are shown in

TABLE 22.5

Diagnosis-Based Strategies

Functional Symptom	Treatment Protocol
Oscillopsia (blurred or distorted vision with active head movement)	**Adaptation**–gaze stabilization: Resets VOR gain
Vestibular recruitment (exaggerated or hypersensitivity to movement or sense of after-motion)	**Adaptation**–habituation: Extinguishes noxious signal
Visual and surface dependence (vision and touch substitute for vestibular)	**Substitution:** Forces increased vestibular function

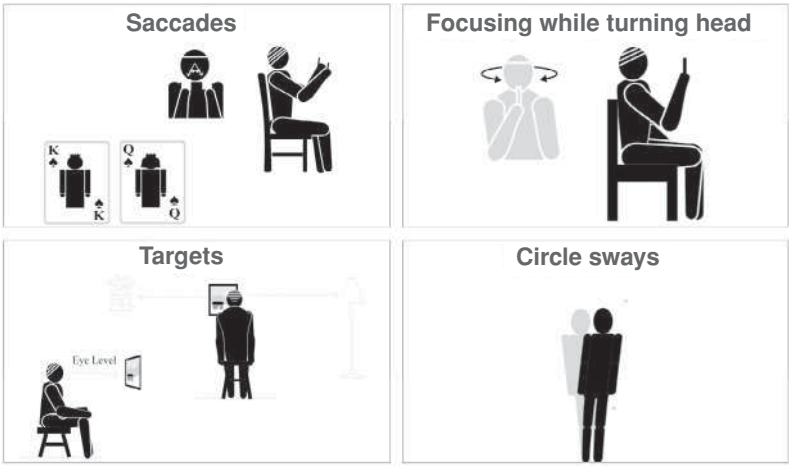


FIGURE 22.1 Adaptation. [Courtesy of Gans RE. [2010] *Vestibular Rehabilitation: Protocols and Programs.* Tampa Bay, FL: AIB Education Press.]

Figure 22.1. A complex activity will incorporate gaze stabilization exercises, mostly coordinated head and eye movement preferably while walking at different rates and on a variety of planes.

A good example of this exercise would be to have the patient sit on a balance ball with a slight bounce, while turning the head from side to side and while also reading two separate word lists (e.g., grocery lists). Activities that disrupt the predictability of gaze stabilization or somatosensory input will be useful. Gaze stabilization exercises may progress from easy to more difficult as a progression, beginning with the patient performing side-to-side head turns while seated on a stationary chair and moving on to those while seated on a ball.

Baseline, serial, or final performance can be evaluated with any technique that evaluates the VOR function. This may be as simple as testing DVA with a Snellen eye chart or the American Institute of Balance (AIB) Dynamic Visual Acuity Test or as complicated as using the technologically advanced vestibular autorotation testing that provides a computerized analysis of the eye and head velocity. An important subset of adaptation is that of habituation as shown in Figure 22.2. These exercises reduce the hallucination of motion or movement as well as extinguishing the sensation of after-motion. This response is based on neural plasticity within the brain and works only through the systematic repetition of the movements and acceleration with speed or direction that provoke the symptoms. The brain is exposed to the noxious stimuli repeatedly in a short time span. The patient's subjective reports of the intensity of the motion and duration of after-motion are utilized to determine treatment efficacy.

Substitution

Substitution protocols as shown in Figure 22.3 will strengthen the weakened systems by reducing the dependence on the remaining ones. In the case where a sensory modality is deficient or absent, these protocols will work to strengthen or make the remaining systems more accurate in their response to a dynamically changing environment. A patient with a weakened vestibular system is forced to make that system more dominant by reducing or challenging the somatosensory input, for instance by standing on a trampoline. The visual sense could be further disrupted or diminished by

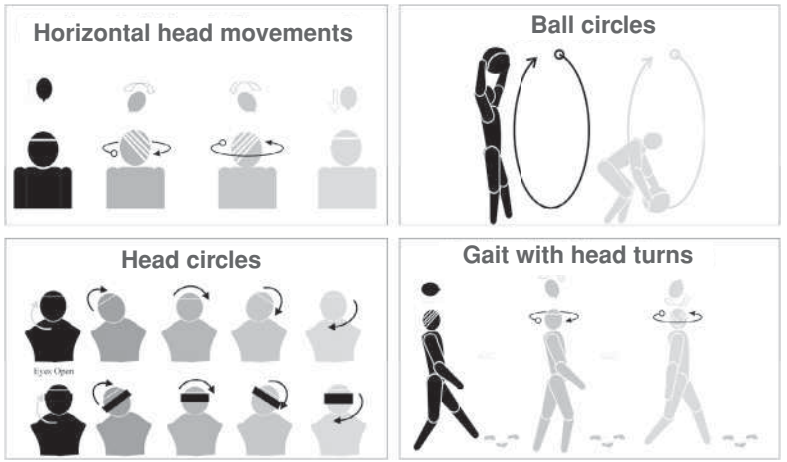


FIGURE 22.2 Habituation. [Courtesy of Gans RE. [2010] *Vestibular Rehabilitation: Protocols and Programs.* Tampa Bay, FL: AIB Education Press.]

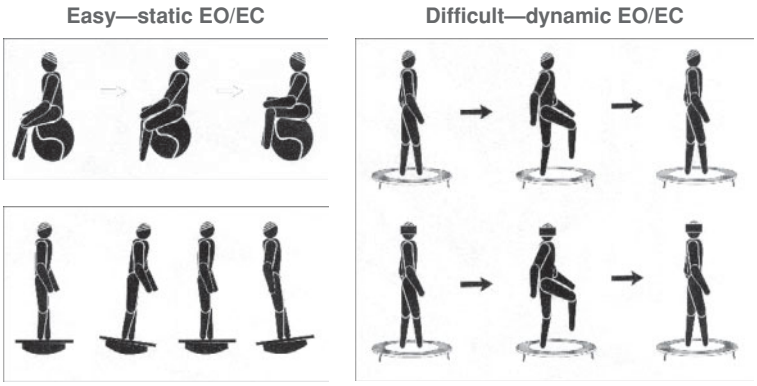


FIGURE 22.3 Substitution protocols. [Courtesy of Gans RE. [2010] *Vestibular Rehabilitation: Protocols and Programs.* Tampa Bay, FL: AIB Education Press.]

having the patient close his/her eyes or watch a moving visual stimulus while maintaining his/her balance.

Evaluation of a patient's performance may include tests of postural stability on dynamic (moving or changeable) surfaces with absent vision. A simple version of this test is Schumway-Cook and Horak's (1986) classic CTSIB, which utilizes a standardized foam base of support (BOS) on which patients stand, first with eyes open and then closed. The hallmark of a noncompensated vestibulopathy will be the patients' inability to maintain their balance when they close their eyes while standing on the foam base. The premise is that individuals with intact vestibular function should be able to maintain their balance while standing on a dynamic surface even when they close their eyes. Through substitution, the vestibular system is being tested and forced to overcome the challenge of the dynamic BOS and absence of visual input. A more complex test is the computerized dynamic posturography, originally used by NASA in the early 1980s to evaluate balance function of returning shuttle astronauts (Paloski et al., 1992). This test trains patients on a Balance Master unit, providing them with visual feedback about their limits of stability (LOS) and balance function during therapy.

CASE STUDY

The following case study and clinical pathway are provided as an example of a typical vestibular patient's symptoms and treatment.

History and Symptoms

A 52-year-old male was referred to the clinic by his primary care physician with a chief complaint of acute positional vertigo. His physician obtained a magnetic resonance imaging (MRI) and blood work profile, all of which were reported as normal. His history included episodes of vertigo, the first of which began approximately 4 months prior. The initial episode lasted for approximately 7 to 8 days, with severe nausea and emesis throughout the episodic period, followed by a spontaneous recovery of the vertigo and symptoms, until approximately 2 months later.

The second attack also lasted about 7 to 8 days. The only other condition or symptom the patient related occurring during these acute episodes was a significant outbreak of cold sores. Following the last attack (within 4 to 5 days), the patient reported an acute episode of vertigo when he would lie flat or turn his head while lying down. This vertigo would last only seconds. His complaints also included a sensation that the world was "jiggling" when he looked from side to side or when he walked. He felt as though his eyes "don't have any shock absorbers." Although the sensation of his "bouncing world" was not as frightening as the acute positional vertigo, it was annoying and limited his activity level. No hearing loss was associated with the attacks or subsequent to them, nor was there a history of migraine or migraine equivalent.

Clinical Findings

Video-oculography revealed a left posterior canalithiasis during modified Hallpike positioning. A 43% left unilateral weakness was revealed on the caloric portion of VNG testing. Vestibular autorotation testing indicated abnormal (hypofunction) gain in the horizontal and vertical plane. Computerized dynamic visual acuity test (CDVAT) produced a 25% decrease in visual acuity with active head movement in the horizontal plane. All other audiologic studies were unremarkable. The patient had normal hearing acuity for all test frequencies. Normal immittance studies and distortion product otoacoustic emissions were also obtained.

Recommendations

The treatment strategy with this patient was twofold. First, the recurring attacks and nature of symptoms (including outbreak of cold sores) were suggestive of a viral vestibular neuritis, and an otologist was consulted. Following positive lab results for the herpes simplex type 1, the patient was placed on an antiviral medication prescription and a daily regimen of a lysine supplement by the otologist to control or inhibit further outbreaks. The patient was referred back to the clinic for treatment of the left posterior canal BPPV and noncompensated left UVD.

Treatment and Outcomes

The patient's VRT continuum of care included treatment of the left PC-BPPV and a noncompensated left vestibulopathy. During the first visit, the left posterior canal BPPV was treated with a Gans repositioning maneuver (GRM) (Roberts and Gans, 2008; Roberts et al., 2006). The patient was rechecked and re-treated on the same visit, which has been shown to increase the success rate to 97% in the first treatment visit. At the second visit, the video-oculographic recording was used to ensure that the BPPV had been cleared. The patient was then able to proceed with all aspects of a rigorous VRT program, which included adaptation with both gaze stabilization and habituation protocols as well as substitution.

The patient participated in an outpatient program provided by the trained physical therapist twice a week for 4 weeks, along with a supplement of home-based activities. At week 3 (20 days post initiation of the program), he reported a 100% reduction in symptoms. Retesting of vestibular autorotation and computerized dynamic visual acuity testing indicated a recovery and return to normal function on both tests. Additionally, scores on the Dizziness Handicap Inventory (Table 22.4) confirmed absence of subjective disability in him with a total score of 0%. The patient was pleased with these results and was discharged from clinical care. He was encouraged to follow up both with his primary care physician and the otologist, should he have a recurrence of the acute-phase vestibular neuritis. He was encouraged to resume his home-based vestibular rehabilitation protocols if at any time he felt a return of any of the oscillopsic symptoms during active head movement.

SUMMARY

With 60 years of research, clinical experience, and a growing patient acceptance, VRT has been demonstrated to be an important and effective nonmedical treatment for the symptoms of noncompensated vestibular disorders. Our understanding of the importance of intact VOR function in human equilibrium and new and simplified tests of this system will allow us to continue to quickly identify and treat this population. New research in cochleovestibular hair cell regeneration may someday make VRT unnecessary. In the meantime, it presents as one of the simplest and most successful treatment options available.

FOOD FOR THOUGHT

1. What are the two most common medical–otologic conditions that cause vertigo?
2. Which type of patient is the best candidate for VRT?
3. Describe how substitution-based therapy protocols can help strengthen a weakened vestibular system.

REFERENCES

Alsalaheen BA, Mucha A, Morris LO, Whitney SL, Furman JM, Camiolo-Reddy CE, et al. (2010) Vestibular rehabilitation for dizziness and balance disorders after concussion. *J Neurol Phys Ther.* 34, 87–93.

Black FO, Angel CR, Pesznecker SC, Gianna C. (2000) Outcome analysis of individualized vestibular rehabilitation protocols. *Am J Otol.* 21, 543–551.

Brandt T, Daroff RB. (1980) Physical therapy for benign paroxysmal positioning vertigo. *Arch Otolaryngol.* 106, 484–485.

Cawthorne T. (1944) The physiological basis for head exercises. *J Chart Soc Physiother.* 29, 30–106.

Cohen HS. (1992) Vestibular rehabilitation reduces functional disability. *Otolaryngol Head Neck Surg.* 107 (5), 638–643.

Cohen HS, Kimball KT, Adams AS. (2000) Application of vestibular disorders activities of daily living scale. *Laryngoscope*. 110, 1204–1209.

Cooksey FS. (1946) Rehabilitation and vestibular injuries. *Proc R Soc Med.* 39, 273–277.

Gans R. (2013) Vestibular rehabilitation therapy. In: Dispenza F, De Stefano A, eds. *Textbook of Vertigo Diagnosis and Management.* London: Jaypee Brothers Medical Publishers.

Gans RE. (1996) *Vestibular Rehabilitation: Protocols and Programs.* San Diego, CA: Singular Publishing.

Gans RE. (2010) *Vestibular Rehabilitation: Protocols and Programs.* Tampa Bay, FL: AIB Education Press.

Gates GA. (2000) Clinimetrics of Meniere's disease. *The Laryngoscope*. 110 (3), 8–11.

Halmagyi GM, Curthoys IS, Cremer PD, et al. (1990/1991) Head impulses after unilateral vestibular deafferentiation validate Ewald's Second Law. *J Vestib Res.* 1, 187–197.

Heitger MH, Jones RD, Macleod AD, Snell DL, Frampton CM, Anderson TJ. (2009) Impaired eye movements in post-concussion syndrome indicate suboptimal brain function beyond the influence of depression, malingering or intellectual ability. *Brain.* 132, 2850–2870.

Herdman SJ. (2001) Therapy: rehabilitation. In: Goebel JA, ed. *Practical Management of the Dizzy Patient.* Philadelphia, PA: Lippincott, William & Wilkins; pp 327–344.

Hillman E, Bloomberg J, McDonald P, Cohen H. (1999) Dynamic visual acuity while walking in normals and labyrinthine-deficient patients. *J Vestib Res.* 9, 49–57.

Jacobson GP, Newman CW. (1990) The development of the dizziness handicap inventory. *Arch Otolaryngol Head Neck Surg.* 116 (4), 424–427.

Kramer P, Shelhamer M, Zee DS. (1998) Short-term vestibulo-ocular adaptation: influence of context. *Otolaryngol Head Neck Surg.* 119, 60–64.

Lisberger S. (1998) Physiologic basis for motor learning in the vestibulo-ocular reflex. *Otolaryngol Head Neck Surg.* 119, 43–48.

National Institutes of Health. (2009) NIDCD fact sheet: balance disorders. Available online at: http://www.nidcd.nih.gov/staticresources/health/balance/BalanceDisordersFactSheet.pdf.

O'Leary DP, Davis-O'Leary LL. (1990) High frequency autorotation testing of the vestibulo-ocular reflex. *Neurol Clin.* 8, 297–312.

Paloski WH, Reschke MF, Black FO, Doxey DD, Harm DL. (1992) Recovery of postural equilibrium control following spaceflight. In: Cohen BD, Guedry F, eds. *Sensing and Controlling Motion: Vestibular and Sensorimotor Function.* New York: New York Academy of Sciences; pp 747–754.

Powell LE, Myers AM. (1995) The activities-specific balance confidence scale. *J Gerontol A Biol Sci Med Sci*. 50, M28–M34.

Roberts RA, Gans RE. (2007) Comparison of horizontal and vertical dynamic visual acuity in patients with vestibular dysfunction and nonvestibular dizziness. *J Am Acad Audiol.* 18, 236–244.

Roberts RA, Gans RE. (2008) Nonmedical management of positional vertigo. In: Jacobson GP, Shephard NT, eds. *Balance Function Assessment and Management.* San Diego, CA: Plural Publishing; pp 445–468.

Roberts RA, Gans RE, Johnson EL. (2006) Computerized dynamic visual acuity with volitional head movements in patients with vestibular dysfunction. *Ann Otol Rhinol Laryngol.* 115(9), 658–666.

Roberts RA, Gans RE Montaudo R. (2006) Efficacy of a new treatment maneuver for posterior canal benign paroxysmal positional vertigo. *J Am Acad Audiol.* 17, 598–604.

Schubert MC, Herdman SJ, Tusa RJ. (2002) Vertical dynamic visual acuity in normal subjects and patients with vestibular hypofunction. *Otol Neurotol.* 23, 372–377.

Selby G, Lance JW. (1960) Observations on 500 cases of migraine and allied vestibular headache. *J Neurol Neurosurg Psychiatry.* 23(1), 23–32.

Shepard NT, Telian SA. (1995) Programmatic vestibular rehabilitation. *Otolaryngol Head Neck Surg.* 112, 173–182.

Shumway-Cook A, Horak FB. (1986) Assessing the influence of sensory interaction on balance: suggestions from the field. *Phys Ther.* 66, 1548–1550.

Ware JE. (1988) How to score the revised MOS short form health scales (SF-36). Boston, MA: The Health Institute, New England Medical Center Hospitals.

SECTION III

Special Populations

CHAPTER 23

Newborn Hearing Screening

Karl R. White

INTRODUCTION

The importance of identifying children with permanent hearing loss as early as possible was emphasized more than 70 years ago when Ewing and Ewing (1944) noted

> . . . an urgent need to study further and more critically methods of testing hearing in young children . . . during this first year the existence of deafness needs to be ascertained . . . training needs to be begun at the earliest age that the diagnosis of deafness can be established. (pp 309–310)

Since then much time and effort has been devoted to finding the most efficient and accurate procedures, protocols, and equipment for screening, diagnosing, and treating children who are deaf or hard of hearing (DHH).*

In 1960, with support from the Children's Bureau in the US Department of Health, Education, and Welfare, the American Speech and Hearing Association convened an expert working group to develop guidelines for "Identification Audiometry." With respect to infants, the report of this group concluded that

> In the testing of a child from birth until approximately two months of age use can be made of the startle response . . . In a baby with good hearing and an intact central nervous system any sudden moderately loud sound will bring about a widespread response: the ongoing muscular activity is inhibited, the hands are pronated, the eyelids blink, etc. These startle responses are so uncomplicated, relatively speaking, that they may be easily observed. (Darley, 1961, p 21)

Efforts of many people over the next 30 years would prove that hearing screening for infants and young children was not as easy as it appeared to the participants of that conference in 1960.

In fact, 5 years later at the Toronto Conference on "The Young Deaf Child: Identification and Management" (Ireland and Davis, 1965), Hardy reported the results of one of the first prospective screening studies of over 1,000 newborns at Johns Hopkins Medical Hospital in Baltimore. Hardy was hardly optimistic. In his opinion, the testing of newborns, with the procedures they were using, was a useless effort and they planned to discontinue it.

Many others were having similar experiences. Indeed, progress in finding accurate and feasible methods for identifying infants and young children who were DHH was painfully slow during the next 25 years. In response to a conclusion by the National Institutes of Health Consensus Development Panel (1993) that recommended "screening of all newborns . . . for hearing impairment prior to discharge," Bess and Paradise (1994), in a widely cited *Pediatrics* article, argued that ". . . universal newborn hearing screening in our present state of knowledge is not necessarily the only, or the best, or the most cost-effective way to achieve [early identification of hearing loss] and more importantly . . . the benefits of universal newborn hearing screening may be outweighed by its risks." By 1996, the US Preventive Services Task Force, while acknowledging that "congenital hearing loss is a serious health problem associated with developmental delay and speech and language function," concluded that "there is little evidence to support the use of routine universal screening for all neonates."

By the late 1990s, however, there was a combination of advances in screening and diagnostic equipment, action by various professional organizations, legislative initiatives, and government-funded demonstration programs in various countries. This resulted in a dramatic improvement in our ability to identify and provide services to infants and young children who were DHH and their families.

This chapter summarizes the principles that should guide any health-related screening program, briefly reviews the global situation related to infant hearing screening, and describes the current status of early hearing detection and intervention (EHDI)† programs in the United States with particular attention to the evidence-based practices for

*Many different terms are used to refer to children with permanent hearing loss (e.g., deafness, hearing impairment, hearing loss, auditory disorders). Recognizing that there are limitations to any single term, this chapter will use the term "children who are deaf or hard of hearing (DHH)" except in those cases where a source is quoted.

†Recognizing the importance of linking hearing screening programs to diagnostic and treatment programs, most people have replaced the term "universal newborn hearing screening program" by the more inclusive term "early hearing detection and intervention" (EHDI) program. This change recognizes that screening is just the first step in the process needed to help children who are DHH reach their full potential.

establishing and operating efficient and effective hearing screening programs for infants.

PRINCIPLES OF EFFECTIVE PUBLIC HEALTH SCREENING PROGRAMS

Almost 50 years ago Wilson and Jungner (1968) proposed principles that have become the accepted criteria for deciding if and how to implement public health screening programs. The report, commissioned by the World Health Organization (WHO), came at a time that technologic advances in medicine had made screening a topic of growing importance and controversy.

Their suggestions are worth considering whenever the design and operation of screening programs are being considered. According to Wilson and Jungner,

> In theory, therefore, screening is an admirable method of combating disease . . . In practice, there are snags . . . The central idea of early disease detection and treatment is essentially simple. However, the path to its successful achievement (on the one hand, bringing to treatment those with previously undetected disease, and, on the other, avoiding harm to those persons not in need of treatment) is far from simple though sometimes it may appear deceptively easy. (p 7 and 26)

In what has deservedly become a classic in the public health literature, Wilson and Jungner outlined the following 10 criteria for deciding whether a condition was appropriate for screening. They noted that these criteria were "especially important when case-finding is carried out by a public health agency, where the pitfalls may be more numerous than when screening is performed by a personal physician" (p 26). As discussed in this chapter, even though there is still room for improvement, screening to identify infants who are DHH meets all of those criteria. Thus, it is not surprising that hearing screening programs for infants continue to expand around the world.

1. The condition to be detected by screening should be an important health problem. Congenial hearing loss is the most frequent birth defect in the United States, affecting about 3 newborns per 1,000 (White et al., 2010). Worldwide, permanent hearing loss affects approximately 1% of young children in industrialized nations and the incidence is probably higher, but not well documented, in developing countries. When not detected early in life, children who are DHH lag behind their peers in language, social, and cognitive development; fail more frequently in school; and often do not acquire the skills to be successfully employed.
2. There should be an accepted treatment for cases identified. As documented in the other chapters of this book, educational, audiologic, and medical treatments for children who are DHH are straightforward and relatively inexpensive. Even for children needing more expensive treatments, there is clear evidence that the benefits significantly outweigh the costs.
3. Facilities for diagnosis and treatment should be available. Although there are still shortages of clinicians and intervention programs, most children who are DHH in the United States have access to high-quality treatment programs and well-trained professionals. In many ways, demand is driving supply. As more and more children are identified earlier and earlier, the number of well-trained professionals and access to effective treatment programs are improving.
4. There should be a recognizable latent or early symptomatic stage. In other words, it should be possible to identify the condition while there is still time to do something to improve the outcome. Hearing screening enables us to identify infants who are DHH before there are outward signs of the hearing loss, such as delayed language development. The earlier children who are DHH are identified and receive treatment, the better the outcomes. This is true for all types and degrees of hearing loss.
5. There should be a suitable screening test. Physiological screening tests for hearing are sensitive, specific, and relatively inexpensive.
6. The test should be acceptable to the population. Hearing screening tests can be completed in less than 15 minutes per child, are painless, and have no negative side effects. Parents express a high degree of satisfaction with hearing screening tests.
7. The natural history of the condition should be understood. Because permanent hearing loss occurs relatively frequently and the consequences are so obvious, the condition has been studied for hundreds of years and the natural history is well understood.
8. There should be an agreed policy on whom to treat as patients. Because hearing loss of any degree and type affects language, social, and cognitive development, there is widespread agreement about the importance of identifying and treating all children who are DHH.
9. The cost of case-finding (including diagnosis and treatment of those diagnosed) should be economically balanced in relation to possible expenditure on medical care as a whole. Hearing screening and diagnosis are relatively inexpensive and there is now good evidence that the cost of identification and treatment is small compared to the benefits.
10. Case-finding should be a continuing process and not a "once and for all" project. Although most major initiatives during the last 20 years have focused on newborn hearing screening, there is increasing emphasis on hearing screening for preschool and school-aged children.

The criteria suggested by Wilson and Jungner in 1968 are still relevant today. They provide a useful framework

by which hearing screening programs can continue to be refined and outcomes can be improved.

GLOBAL STATUS OF NEWBORN HEARING SCREENING

Policy makers and healthcare providers in many different countries have recognized the benefits and feasibility of newborn hearing screening programs. At least seven countries (Austria, the Netherlands, Oman, Poland, Slovakia, the United Kingdom, and the United States) provide hearing screenings for more than 90% of their births and nine other countries screen 30% to 89% of their births (Australia, Belgium, Canada, Germany, Ireland, Philippines, Russia, Singapore, and Taiwan). At least 60 other studies have published reports of smaller scale universal newborn hearing screening (UNHS) programs in their countries and are working toward establishing national systems (NCHAM, 2013a; White, 2011).

The WHO has long been a strong advocate of hearing screening for infants and young children. The 48th World Health Assembly urged member states "to prepare national plans for early detection in babies, toddlers and children" (Resolution 48.9) and the WHO recommended "that a policy of universal neonatal screening be adopted in all countries and communities with available rehabilitation services and that the policy be extended to other countries and communities as rehabilitation services are established" (WHO, 2010).

In 2009 an international group of experts convened by the WHO proposed guiding principles for action related to infant hearing screening. The report noted that in spite of the global progress that has been made toward UNHS, there are still many countries where the implementation of such a program is considered too costly and/or its value is questioned. Even in countries where a significant number of newborns are screened for hearing, there are often no consistent approach or quality control procedures, oversight is frequently not implemented, and resources for follow-up are often limited. However, the WHO noted that the operation of effective hearing screening programs for infants and young children is not always related to resources—some wealthy countries have fragmented and ineffective programs, whereas other less-wealthy countries have very successful EHDI programs. The report noted that, "Quality assurance issues in particular are vital to successful newborn and infant hearing screening and related interventions—in some settings, it is estimated that the poor training and performance of screeners renders up to 80% of screening useless" (WHO, 2010).

Although the WHO report concluded that all newborns should be screened for hearing loss using a physiological measure such as otoacoustic emissions (OAE) or automated auditory brainstem response (A-ABR), it acknowledged that some countries cannot implement such programs because of limited financial resources or because appropriate equipment and personnel are not available. In such situations, the WHO recommended that some combination of targeting particular subgroups of the population or the use of questionnaires completed by family members or behavioral testing be considered (see Figure 23.1).

		Screening methods		
		Questionnaire completed by family	**Behavioral**	**Physiological**
Infants to be screened	**Targeted by:** Geographical subset			
	NICU babies			
	Babies with risk factors			
	Population based			

FIGURE 23.1 Hearing screening options recommended by World Health Organization (2010).

Questionnaires can be used to ask parents or other caregivers about the response of the infant to sounds and the infant's use of language, including early indicators of language such as babbling and other vocalizations. Infants and young children who perform poorly on such measures can then be referred for more comprehensive audiologic assessment. Whereas some researchers have reported encouraging results for such questionnaires in screening children for hearing loss (e.g., Newton et al., 2001), others have recommended against using questionnaires because of relatively high false-positive and false-negative rates (e.g., Li et al., 2009; Watkin et al., 1990). The usefulness of questionnaires may depend, in part, on the age of children being screened, the degree of hearing loss targeted for detection, and the knowledge of parents or caregivers about normal language development. Even though questionnaires are relatively inexpensive, more evidence about their specificity and sensitivity is needed before wide-scale use can be recommended. In those situations, where physiological screening is impossible, questionnaires will likely result in some children who are DHH being identified, but the negative effects associated with potential false negatives and false positives are of great concern.

Behavioral measures, such as noisemakers or other more sophisticated audiologic procedures and equipment, can also be used to identify infants and young children who are DHH. However, such methods also have relatively large numbers of false negatives and false positives when used with babies less than 12 months of age. For example,

Watkin et al. (1990) did a retrospective analysis of over 55,000 2- to 15-year-old children in England who had completed a behavioral evaluation for hearing when they were 7 to 12 months of age. Of the 39 children later identified with severe to profound bilateral hearing losses, only 44% were identified when they were 7 to 12 months old based on the behavioral evaluation. The remaining children were identified later based on school-age screening programs, parental concern, or by healthcare providers. For children with mild to moderate bilateral hearing losses and children with unilateral hearing losses, the behavioral evaluation at 7 to 12 months of age identified only 25% and less than 10%, respectively. Even when home visitors are specifically trained to do behavioral evaluations of hearing in a home setting, most young children who are DHH will be missed using such procedures.

The WHO report also recommended that when it is not feasible to implement universal hearing screening programs for all newborns, countries should consider starting with a hearing screening program that focuses on a subset of infants and young children. For example, when newborn hearing screening programs are being established, it is not unusual to focus on babies in a particular geographical region because they are more accessible or equipment and personnel are more available. Because the incidence of permanent hearing loss is much higher among neonates who require intensive medical care during the first few days of life, hearing screening programs can focus on those admitted to a neonatal intensive care unit if they are unable to screen all babies.

There is a great deal of evidence that babies with certain "risk indicators" have much higher rates of permanent hearing loss than those who do not. The Joint Committee on Infant Hearing (JCIH, 2007) has identified 11 risk indicators (e.g., family history of permanent childhood hearing loss, being in a neonatal intensive care unit for more than 5 days, presence of craniofacial anomalies) that are associated with permanent congenital or delayed-onset hearing loss. Even though only about 10% of all newborns exhibit one or more of these risk indicators, about 50% of the infants who are DHH will be in this group. Unfortunately, hearing screening programs that target only infants with risk indicators have not been successful in identifying many of the babies with hearing loss in this high-risk group. For example, Mahoney and Eichwald (1987) reported the results of a newborn hearing screening program that targeted all babies with a high-risk indicator born in their state over an 8-year period. Information about the presence of risk indicators was incorporated into the state's legally required birth certificate so information about risk indicators was collected on virtually all babies. A computerized mailing system and follow-up phone calls were used to offer all parents of children with risk indicators a free diagnostic audiologic assessment at local health department offices. Also a mobile van traveled around the state to provide free diagnostic testing for families in the rural parts of the state. Mahoney and Eichwald (1987) reported that only about 50% of the families who had a baby with a risk indicator made appointments for an audiologic assessment and only about 50% of those actually came to the appointment. The program was discontinued after 8 years because of the small number of babies identified (the prevalence of babies identified as being DHH was less than 0.30 per 1,000 or about 10% of the babies who were likely DHH in that cohort).

Before implementing a hearing screening program that targets only those babies with one of the JCIH-recommended risk indicators, it is important to remember that 95% of the babies who have one of the risk factors do not have hearing loss and that approximately half the babies who do have congenital hearing loss will not exhibit any risk factors (Mauk et al., 1991). Thus, even if a risk-based newborn hearing screening program worked perfectly, it would only identify half of the babies with permanent hearing loss. However, the yield from operational high-risk hearing screening programs has been much lower. Furthermore, the risk factors that are most predictive of hearing loss in babies will vary from country to country, so it is important to have local data about the sensitivity and specificity of risk factors before using this as a method of identifying children who are DHH.

Alternatives to UNHS based on physiological measures such as OAEs or A-ABRs do need to be considered in some situations. However, unless and until better data are available to demonstrate acceptable sensitivity and specificity of alternative approaches (e.g., parent questionnaires, behavioral measures, and programs targeting high-risk babies) program planners should recognize that most previous programs using these methods have had significant limitations. Such alternatives should be viewed as an interim step toward establishing a UNHS program. Recognizing that different approaches will need to be taken in different circumstances, the WHO report (2010) emphasized that all newborn hearing screening programs should have

- Clearly stated goals with well-specified roles and responsibilities for the people involved
- A clearly designated person who is responsible for the program
- Hands-on training for people who will be doing the screening
- Regular monitoring to ensure that the protocol is being correctly implemented
- Specific procedures about how to inform parents about the screening results
- Recording and reporting of information about the screening for each child in a health record
- A documented protocol based on local circumstances

It is also important to remember that successful newborn hearing screening programs have been implemented in many countries in many different ways. Despite the variety

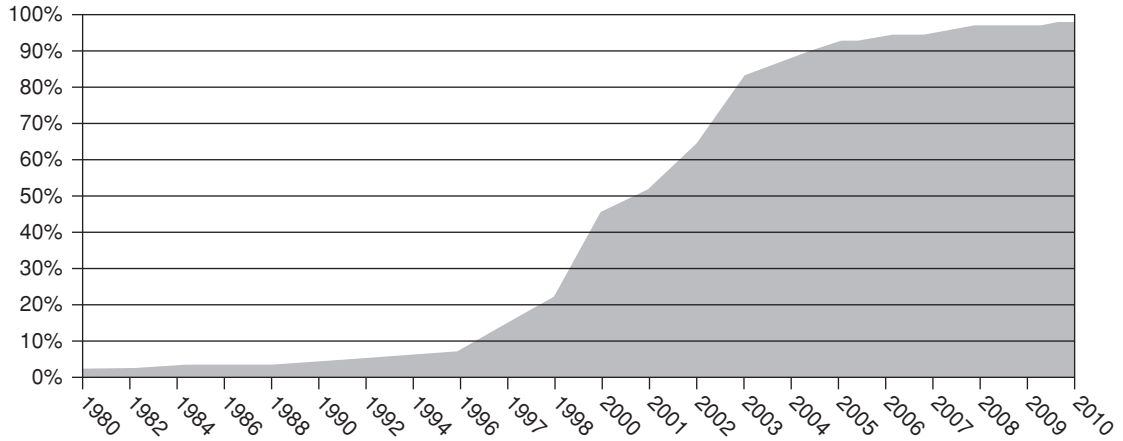


FIGURE 23.2 Percentage of newborns screened for hearing loss in the United States during the last 30 years.

of circumstances in which they operated, WHO (2010, p 34) noted that

> [T]he aims of [newborn hearing screening] programmes are widely accepted as both highly worthwhile and attainable and . . . should be expanded to include all neonates and infants. Although universal newborn hearing screening using OAE or A-ABR should be the goal for all countries, interim approaches using targeted screening based on questionnaires, behavioural methods and/or physiological methods guided by evidence from well-conducted pilot studies will also be beneficial. Whatever approach is used, it is important that the EHDI programme is linked to existing health care, social and educational systems, and that the procedures and outcomes of the programme be documented so that ongoing quality assurance activities can be implemented and experiences shared.

THE CURRENT STATUS OF EHDI PROGRAMS IN THE UNITED STATES

EHDI programs have expanded dramatically in the United States during the last 20 years. In 1999 the US Department of Health and Human Services established the following goal related to EHDI programs as a part of its objectives for *Healthy People 2010:*

> Increase the proportion of newborns who are screened for hearing loss by age 1 month, have audiologic evaluation by age 3 months, and are enrolled in appropriate intervention services by age 6 months.

This goal represented a major shift in the belief that children who are DHH could be identified earlier and provided with services that would enable them to be as successful as their normally hearing peers. The value of identifying congenital hearing loss during the first few months of life had been recognized for decades, but the belief that this goal could be achieved was relatively new.

When Dr. C. Everett Koop, Surgeon General of the United States, in 1989 called for increased efforts to identify congenital hearing loss during the first few months of life (Northern and Downs, 1991) he stated:

> . . . hearing impaired children who receive early help require less costly special education services later . . . I am optimistic. I foresee a time in this country . . . when no child reaches his or her first birthday with an undetected hearing impairment.

Many people were surprised by Dr. Koop's enthusiasm and his optimism that UNHS programs could be successfully established given the fact that fewer than 3% of all newborns in the United States were being screened for hearing loss at that time. Over the next 25 years, Dr. Koop's enthusiasm proved to be well founded as shown by the fact that more than 98% of all newborns in the United States are now screened for hearing loss (Centers for Disease Control and Prevention (CDC), 2013, see Figure 23.2). Understanding the factors that led to such a significant change can be useful as work continues to make hearing screening programs more effective and efficient.

Factors Contributing to the Expansion of Newborn Hearing Screening Programs

The establishment, expansion, and improvement of newborn hearing screening programs in the United States have been facilitated by (1) policy initiatives by government, professional associations, and advocacy groups, (2) financial assistance from the federal government, (3) improvements in technology, (4) legislative initiatives, and (5) the demonstrated success of early implementations.

Policy Initiatives

The value of identifying children who are DHH as early as possible is not a new concept for healthcare providers and administrators in the United States. For example, the Babbidge Report issued by the US Department of Health, Education, and Welfare in 1965 recommended the development and nationwide implementation of ". . . universally applied procedures for early identification and evaluation of hearing impairment." Four years later in 1969, based on the pioneering work of Marion Downs (Downs and Hemenway, 1969), the Joint Committee on Infant Hearing (JCIH, 2007) was established by a group of professional associations (e.g., American Speech and Hearing Association, American Academy of Pediatrics, American Academy of Otolaryngology—Head and Neck Surgery, among others). Even though the JCIH had no formal authority and few resources, they became, and have remained, a powerful force in advocating for earlier identification and better treatment of children who are DHH.

When it was first established, the JCIH focused on screening high-risk babies because inexpensive and effective hearing screening technology was not yet available. As new hearing screening technologies became available in the late 1980s, more resources were devoted to early identification of children who were DHH. These efforts were stimulated in part by a recommendation from the congressionally mandated Commission on Education of the Deaf (Toward Equality, 1988) that "the Department of Education, in collaboration with the Department of Health and Human Services, should . . . assist states in implementing improved screening procedures for each live birth."

A few years later, *Healthy People 2000* established a goal to "reduce the average age at which children with significant hearing impairment are identified to no more than 12 months":

> . . . it is difficult, if not impossible, for many [children with congenital hearing loss] to acquire the fundamental language, social, and cognitive skills that provide the foundation for later schooling and success in society. When early identification and intervention occur, hearing-impaired children make dramatic progress, are more successful in school, and become more productive members of society. The earlier intervention and habilitation begin, the more dramatic the benefits. (HHS, 1990, p 460)

Although similar goals had been discussed for 30 years, this one was different because it was linked to a federal mandate that progress toward each objective had to be tracked and reported at regular intervals.

Another major step forward happened in 1993 when a Consensus Development Panel convened by the National Institutes of Health recommended that "all infants [who are DHH] should be identified and treatment initiated by six months of age" and concluded that UNHS was the best way to accomplish this goal. To the surprise of many, progress was slow. It would be another 12 years before more than 90% of the newborns in the United States were screened prior to discharge (see Figure 23.2).

That so much time elapsed between the recommendation by NIH and the achievement of UNHS was in part because of the lack of research evidence about the value of and experience for such broad-scale implementation of newborn hearing screening. In the words of one skeptic in a commentary entitled, *Universal Newborn Hearing Screening: Should We Leap Before We Look?* (Paradise, 1999, pp 670–671):

> Across the nation pediatricians are being importuned, and indeed propelled, to implement universal newborn hearing screening, despite a total lack of information concerning ultimate costs and, particularly, risks . . . I feel compelled to try here once again to be heard, quixotic though it may seem in the face of such apparently formidable odds. My main objections to a universal screening program for presumably normal, low-risk newborns remain essentially unchanged . . . recent reports from screening programs offer no basis for greater optimism about reducing the numbers of false-positive identifications.

Federal Support for EHDI Initiatives

Partly because there was so little research about and experience with newborn hearing screening programs, significantly more federal funding was devoted to research, demonstration, and technical assistance projects related to newborn hearing screening during the late 1980s and early 1990s. Some of the best known were the Rhode Island Hearing Assessment Project (White and Behrens, 1993), the Marion Downs Hearing Center (MDHCF, 2013), and the National Center for Hearing Assessment and Management at Utah State University (NCHAM, 2013b) but there were many others.

Successful Implementation of Screening Programs

Although the concerns about newborn hearing screening expressed by Bess and Paradise (1994) and Paradise (1999) were widely criticized (e.g., White and Maxon, 1995), Bess and Paradise were correct in pointing out that there was very little research in 1993 from large, systematically implemented UNHS programs to support the recommendations of the NIH Consensus Development Panel. Besides the Rhode Island Hearing Assessment Project (White and Behrens, 1993), the available evidence about newborn hearing screening was based on small samples of infants (primarily from NICUs) over short periods of time. The controversy about the NIH recommendations generated by Bess and Paradise stimulated a great deal of activity between 1994 and 1999 as

the percentage of babies being screened for hearing loss prior to hospital discharge increased steadily (see Figure 23.2). By 1998 there was a growing body of research supporting the feasibility, cost-efficiency, and benefits of newborn hearing screening (e.g., Finitzo et al., 1998; Mehl and Thomson, 1998; White, 1997) and dozens of large-scale UNHS programs had become operational in various states. Since that time, more and more research has been published showing the benefits of newborn hearing screening (e.g., McCann et al., 2009), and the United States Preventive Services Task Force now "recommends screening of hearing loss in all newborn infants" (USPSTF, 2008, p 143).

Technologic Advances

Technologic breakthroughs in hearing screening equipment in the late 1980s were a major contributor to the growth of newborn hearing screening programs. Without the improvements in OAEs and A-ABRs, the many policy initiatives, federally funded projects, and clinical screening programs that combined to demonstrate the practicality and efficacy of UNHS programs, this success would never have happened. The advances in technology are likely to continue.

Endorsements by Professional and Advocacy Groups

Published research studies combined with statewide UNHS programs that were identifying hundreds of babies at ever younger ages led to more endorsements and policy statements by government, professional, and advocacy organizations—including the American Academy of Pediatrics, the American Speech-Language-Hearing Association, the American Academy of Audiology, the National Association of the Deaf, March of Dimes, and the American College of Medical Genetics (see NCHAM, 2013c for a summary of endorsements by various organizations).

By the end of 2001, EHDI programs were clearly established as a part of the public health system in the United States, with all 50 states having established an EHDI program (White, 2003). Also in 1998, the federal Maternal and Child Health Bureau (MCHB) began requiring states to report the percent of newborns they had screened for hearing loss before hospital discharge as one of 18 core performance measures states must report annually to receive federal MCHB block grant funding (MCHB, 2002).

Legislation Related to Newborn Hearing Screening

The preceding activities were important in creating an atmosphere where many newborn hearing screening programs could be implemented, but legislative and administrative actions in the late 1990s and early 2000s contributed to expanding the reach and sustainability of these programs. There are now 43 states with statutes or rules related to newborn hearing screening. A recent analysis by Green et al. (2007) concluded that states with legislation were much more likely to be screening 95% or more of their babies than those without legislation. Copies of each statute and/or rule as well as an analysis of the provisions of each statute is available at NCHAM (2013d). Several points about existing legislation are worth noting:

1. Most legislation (34 of 43 states) was approved after 1998. The increase in legislative activity was probably influenced by the publication of the Position Statement by the American Academy of Pediatrics (1999) and the increased amount of research evidence about the efficacy, accuracy, and feasibility of newborn hearing screening programs.
2. The existence of legislation is neither necessary nor sufficient to guarantee an effective EHDI program as demonstrated by the fact that some states that have not passed legislation have EHDI programs that are functioning as well or better than some states with statutes.
3. Only 28 of 43 states (65%) require all babies to be screened. Some statutes set the standard as low as 85% of all newborns which raises questions about equal access to hearing screening—at least in those states.
4. The fact that only seven states (16%) require parents to provide written informed consent suggests that most states view hearing screening as a routine part of newborn health care.
5. Twenty-nine of 43 states (67%) require hospitals to report data from newborn hearing screening to the State Department of Health—suggesting that these states are treating EHDI as a public health program.
6. Twenty-one statutes (49%) indicate that newborn hearing screening must be a covered benefit of health insurance policies issued in the state. However, because of how insurance reimbursement is done, many hospitals do not receive money for screening because payments are made as a lump sum for all services associated with the birth. The federal Affordable Care Act stipulates that newborn hearing screening is a covered preventive service. More information about the implications of the Affordable Care Act for how EHDI programs actually function and what services are available to children and families is available at NCHAM (2013e).

It is also important to note that legislation specifies the minimum requirements of state policy, but often does not describe what is actually happening in the state's EHDI program. For example, the Rhode Island EHDI program has one of the nation's best tracking and reporting systems, reports data to the Department of Health, and has an advisory committee, even though none of these are required by the Rhode Island hearing screening legislation (NCHAM, 2013d).

National Goals for EHDI Programs

As a result of work done by the MCHB, the CDC, and the JCIH, most people have stopped using the phrase "universal newborn hearing screening" (UNHS) in favor of "early hearing detection and intervention" (EHDI). The change is important because it underscores that successfully identifying and serving infants and young children who are DHH requires more than an effective newborn hearing screening program. To be effective, the screening program must be connected to a system that includes audiologic diagnosis and appropriate medical, audiologic, and educational intervention. Newborn hearing screening programs should also be coordinated with the child's primary healthcare provider (PHCP) (often referred to as the child's Medical Home); a tracking and surveillance system; and a process for monitoring/evaluating how the system is functioning.

Newborn hearing screening programs in the United States are almost always hospital based because that is where the vast majority of babies are born. The basic process is similar, even though the specifics, as discussed later in this chapter, vary to a considerable degree. For example, screening may be done by nurses, technicians, audiologists, or someone else. Some programs use OAEs, some use A-ABRs, and some use both. Screening is almost always done before the baby is discharged from the birth admission, but it can be completed at different times of the day depending on the hospital's routine and in different locations (e.g., the nursery, the mother's room, a room designated specifically for screening). Some hospitals do diagnostic evaluations for babies who do not pass the screening test, and others refer those babies elsewhere. Because newborn hearing screening has become a part of routine medical care for newborns, the screening procedures must conform with the hospital's practices related to such matters as safety, privacy, and infection control.

As newborn hearing screening programs expanded during the mid-1990s, it became clear that screening was only the first step in an intertwined process of identifying infants with hearing loss and providing them and their families with timely and appropriate services. Understanding how to best implement and maintain this first step (screening) requires a brief discussion of the other steps (many of which are discussed in more detail in other chapters of this book). A brief You Tube video shows these procedures (www.infanthearing.org/videos/ncham.html#sb).

In collaboration with state EHDI program coordinators and representatives from other federal, professional, and advocacy agencies, CDC has developed National EHDI Goals, Program Objectives and Performance Indicators that are based on EHDI guidelines from various states and the position statements of the Joint Committee on Infant Hearing (JCIH, 2007) and the American Academy of Pediatrics (AAP, 1999). These National Goals (CDC, 2004) are summarized in Table 23.1 and each is discussed in the remainder of this section.

Goal No. 1: All Newborns Will Be Screened for Hearing Loss

CDC (2013) reported that 98.4% of newborns were screened in 2011 (excluding infant deaths and parent refusals). Interestingly, no particular protocol or type of screening equipment is preferred by most people. As shown in Table 23.2, a survey conducted by NCHAM (2013f) showed that 50.3% of all screening programs were using OAE testing, and 62.4% were using A-ABRs (percentages sum to more than 100% because some programs use both OAE and A-ABR). Approximately 40% percent of programs did all of their screening prior to hospital discharge, whereas about 60% of programs used a two-stage protocol in which screening was not completed until an outpatient screening was done following discharge. The variety of screening protocols being used suggests that no single protocol is "best" for all situations. Because, the JCIH (2007, p 904) now recommends "ABR technology as the only appropriate screening technique for use in the NICU [neonatal intensive care unit]" the percentage of programs using A-ABR is expected to increase.

Deciding what type of equipment and which protocol to use in a newborn hearing screening program depends on the circumstances and preferences of the program administrators. In situations where an outpatient screening is a part of the protocol and it is difficult to get babies to come back, A-ABR has an advantage because refer rates at time of discharge are typically lower (but, the cost of equipment and consumables is somewhat higher). It is also important to consider what degree of hearing loss is targeted by the screening program. Most of the currently available A-ABR screening equipment uses a 35-dBnHL click for the stimulus which means that many babies with mild hearing loss will likely pass the screening test (Johnson et al., 2005). In most states, the decision about what type of hearing screening equipment and protocol to use is left to the discretion of the hospital screening program administrator. In fact, NCHAM (2013f) found that only 67% of state EHDI coordinators even keep track of what equipment and/or protocol was used by hospital-based screening programs.

A small, but important subgroup that is not being well served by current EHDI programs are babies who are born at home. With 1% to 2% of all births in the United States occurring outside the hospital, this represents 40,000 to 80,000 babies per year. Only 21 states reported that they had a systematic program in place to screen these babies, and those states only screened an estimated 41% of out-of-hospital births (NCHAM, 2013f). Midwives are well-positioned to screen and follow-up with babies born outside of the hospital, but Goedert et al. (2011) reported that most respondents to a national survey of the American

TABLE 23.1

National Goals for EHDI Programs (CDC, 2004)

Goal 1. All newborns will be screened for hearing loss before 1 mo of age, preferably before hospital discharge	Hospitals will have a written protocol to ensure all births are screened, results are reported to the infant's parents and PCHP, and referred infants (≤4%) are referred for diagnostic evaluation. Demographic data will be collected for each infant and appropriate educational material provided to parents. States will reduce/eliminate financial barriers to screening and ensure screening of out-of-hospital births
Goal 2. All infants who screen positive will have a diagnostic audiologic evaluation before 3 mos of age	States will develop audiologic diagnostic guidelines and maintain a list of qualified providers to ensure infants referred from screening receive a comprehensive audiologic evaluation before 3 mos of age and are referred to appropriate services. States will provide appropriate education and/or training about diagnostic audiologic evaluation to parents, PCHPs, and audiologists
Goal 3. All infants identified with hearing loss will receive appropriate early intervention services before 6 mos of age (medical, audiologic, and early intervention)	States will develop policies and resource guides to ensure all parents of children with hearing loss receive appropriate medical (including vision screening and genetic services), audiologic, and early intervention services (based on the communication mode chosen by the family). States will ensure that early intervention service providers are educated about issues related to infants and young children with hearing loss
Goal 4. All infants and children with late-onset or progressive hearing loss will be identified at the earliest possible time	Hospitals and others will report information about risk factors for hearing loss to the state, who will monitor the status of children with risk factors and provide appropriate follow-up services
Goal 5. All infants with hearing loss will have a medical home as defined by the American Academy of Pediatrics	A primary care provider who assists the family in obtaining appropriate services will be identified for all infants with confirmed hearing loss before 3 mos of age. The state will provide unbiased education about issues related to hearing loss for parents and medical home providers
Goal 6. Every state will have an EHDI Tracking and Surveillance System that minimizes loss to follow-up	A computerized statewide tracking and reporting system will record information about screening results, risk factors, and follow-up for all births. The system will have appropriate safeguards, be linked to other relevant state data systems, and be accessible to authorized healthcare providers
Goal 7. Every state will have a system that monitors and evaluates the progress toward the EHDI goals and objectives	A systematic plan for monitoring and evaluation will be developed and implemented by an advisory committee to regularly collect data and provide feedback to families and ensure that infants and children with hearing loss receive appropriate services

TABLE 23.2

Protocols Used in EHDI Programs

Before Hospital Discharge	After Hospital Discharge	Percent of Newborns Screened
OAE	–	11.6
ABR	–	23.3
OAE/ABR	–	6.7
OAE	OAE	21.4
OAE	ABR	4.2
ABR	OAE	2.8
ABR	ABR	23.2
OAE/ABR	OAE/ABR	6.4
Other protocol	–	0.3

College of Nurses-Midwives members were not well informed about the importance of newborn hearing screening and had significant gaps in their knowledge about screening procedures, steps for referral, and the availability of resources when newborns did not pass the test.

Goal No. 2: Referred Infants Will Be Diagnosed Before 3 Months of Age

For babies who do not pass the newborn screening test, audiologic diagnosis should be completed as soon as possible, but no later than 3 months of age. Figure 23.3 shows that in states with well-developed EHDI programs the average age of diagnosis for children who are identified as DHH has dropped dramatically over the last 25 years.

Unfortunately, CDC (2013) reported that in 2011 for the country as a whole, state EHDI programs were not able

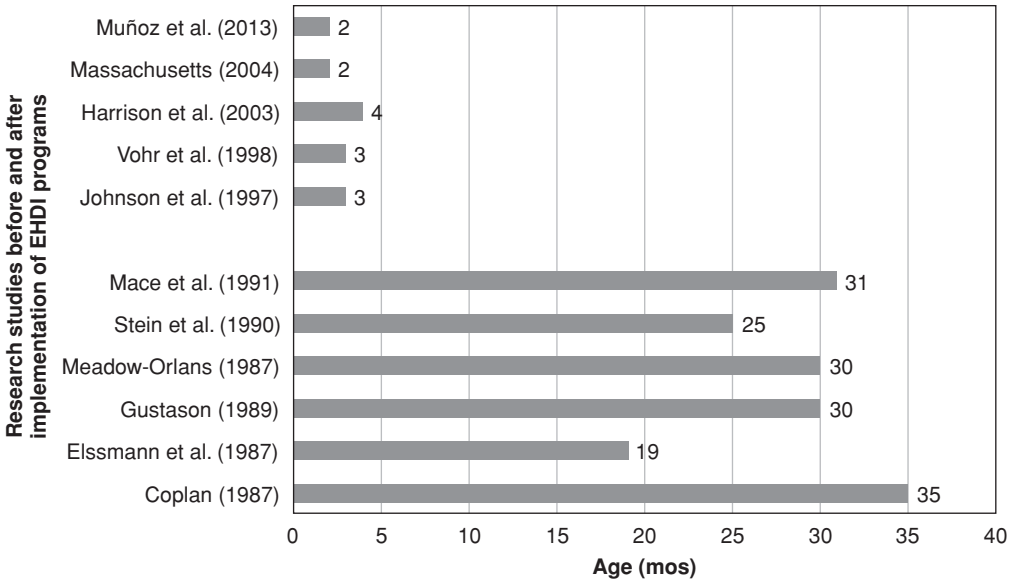


FIGURE 23.3 Age in months at which permanent hearing loss was diagnosed. References to the studies cited may be found at http://thePoint.lww.com.

to document whether diagnostic evaluations were actually completed for 35.3% of the infants who needed them (see Figure 23.4). Most states (90%) have developed written guidelines for conducting diagnostic audiologic evaluations, and most (78%) had compiled a list of centers or individuals who were qualified and had appropriate equipment and experience to do diagnostic audiologic evaluations for infants under 3 months of age (NCHAM, 2013f).

Unfortunately, there is no general agreement on what constitutes a qualified pediatric audiologist, and these lists

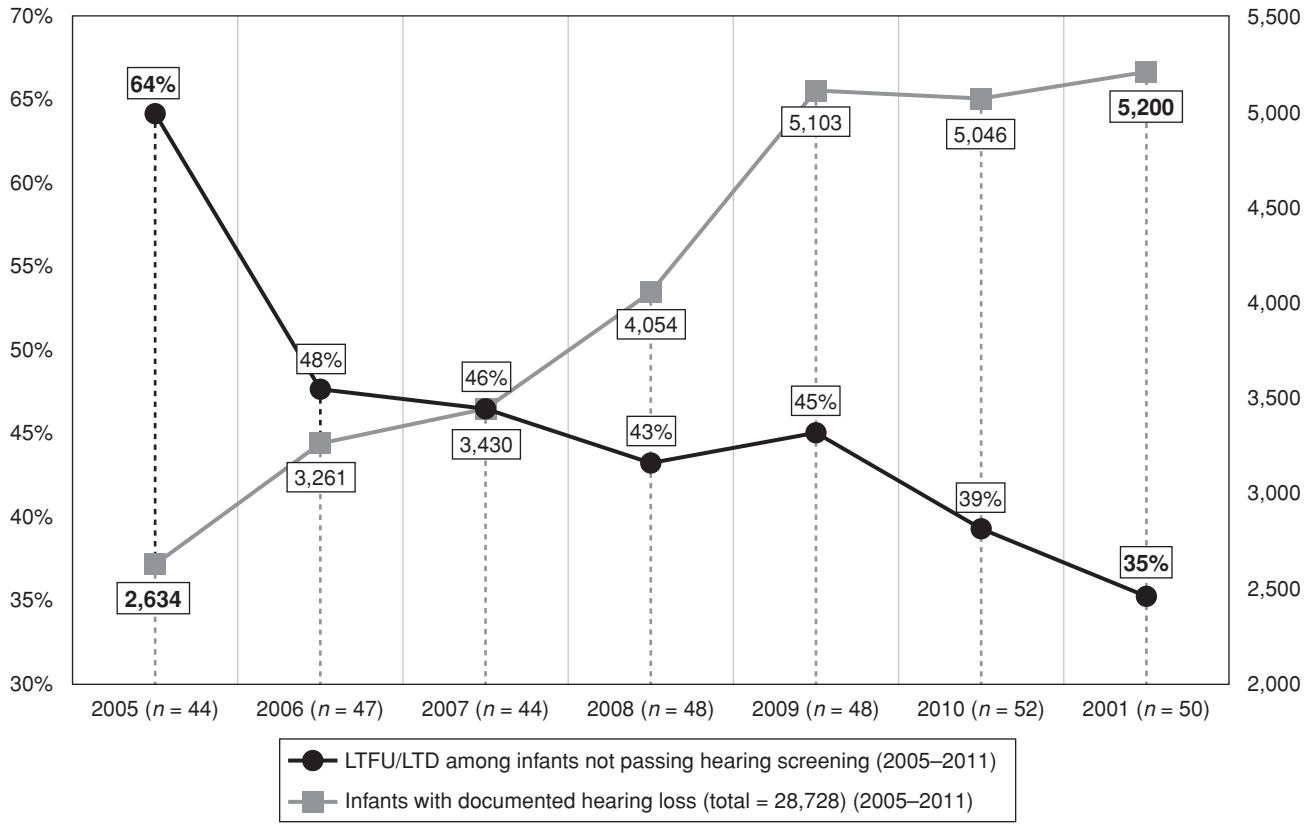


FIGURE 23.4 Number of children who are DHH identified and LTFU/LTD rates from 2005 to 2011.

are mostly composed of self-defined pediatric audiologists. Most state EHDI coordinators (79%) said it would be "beneficial if there were a license or certification for audiologists who specialize in diagnostic assessments and/or hearing aid fitting for infants and toddlers." In 2011, the American Board of Audiology launched the Pediatric Audiology Specialty Certification (PASC) that is supposed to address this need. The PASC was "developed to elevate professional standards in pediatric audiology, enhance individual performance, and recognize those professionals who have acquired specialized knowledge in the field of pediatric audiology" (American Board of Audiology, 2013). The program is still new (only 43 audiologists were certified as of November 15, 2013), so time will tell whether the availability of the PASC improves pediatric audiology services.

Recently, CDC made a web-based service available to help parents and others find qualified pediatric audiologist throughout the nation. Developed in conjunction with collaborators from ASHA, AAA, JCIH, Hands & Voices, NCHAM, and others, the EHDI-PALS (EHDI-Pediatric Audiology Links to Services) provides up-to-date information about facilities that offer pediatric audiology services. All of the facilities listed must report that they have appropriate equipment and expertise to serve children and have licensed audiologists. Similar to other web-based search tools, EHDI-PALS users are asked to answer few simple questions that help pinpoint their location and need. Then the program generates a list of the nearest audiology facilities that match the request. Each listing comes with information about that facility including types of services offered, availability of language interpretation services, payment options, and appointment availability. The service is free and is not linked to any commercial products or services. The system can be accessed at http://www.ehdipals.org/.

In a national evaluation of newborn hearing screening and intervention programs reported by Shulman et al. (2010) the following factors were identified as contributing to poor follow-up rates for audiologic diagnosis:

1. Lack of qualified audiologists to do diagnostic evaluations
2. Lack of appropriate equipment
3. Lack of knowledge among health providers about the importance and urgency of follow-up testing
4. Difficulties with transportation, ability to pay, and motivation on the part of families
5. Poor communication among PHCP, audiologists, and the state EHDI program

Goal No. 3: Provision of Appropriate Medical, Audiologic, and Educational Intervention Before 6 Months of Age

Providing appropriate medical, audiologic, and educational services to infants and young children who are DHH is a complex, multifaceted undertaking. The shortage of experienced and qualified pediatric audiologists often interferes with fitting appropriate hearing technology as early as desired. Another problem is that many PHCPs are not up-to-date regarding early identification of hearing loss. For example, JCIH (2007) recommends that all infants with confirmed hearing loss be referred to a geneticist and an ophthalmologist who has "knowledge of pediatric hearing loss." However, when almost 2,000 PHCPs who care for children in 22 different states and territories responded to a question on a 2005 survey about to whom they would refer a newborn patient who had been "diagnosed with a moderate to profound bilateral hearing loss . . . [when] no other indications are present," only 0.6% said they would refer to an ophthalmologist and 8.9% to a geneticist. When asked at what age an infant could be fit with hearing aids, only 47.3% knew that hearing aids could be fit on children under 4 months of age (Moeller et al., 2006). In a similar survey completed in 2013 (NCHAM, 2013g), a national sample of over 2,000 PHCPs from 26 states responded similarly—only 2.2% and 9.3% would refer to an ophthalmologist or a geneticist, respectively, and only 39.1% knew that children under 4 months of age could be fit with a hearing aid. Clearly, more work needs to be done educating PHCPs so that they can be better partners in providing and supporting families who have children who are DHH.

According to state EHDI coordinators, appropriate educational intervention programs for infants and toddlers with hearing loss are also not as widely available as needed. Part C of the federal Individuals with Disabilities Education Act (IDEA) requires all states to provide appropriate early intervention programs for all infants and toddlers with disabilities. Most children in Part C-funded early intervention programs are enrolled based on the fact that they exhibit significant delays from normal development. Infants and toddlers who are DHH often do not exhibit measurable delays in language, cognitive, or social skills until they are 18 to 24 months of age. Even though federal regulations provide for serving children who have "established conditions that are likely to lead to developmental delays," only 5 of the 51 state plans for Part C provide an operational definition of how children who are DHH would qualify for such services (White, 2006). Of greater concern, CDC (2013) reported that in 2011 state EHDI coordinators were only able to document that 63% of infants and toddlers who the EHDI program had identified as being DHH were enrolled in Part C programs and only 68% of those could be documented as having been enrolled before 6 months of age.

Goal No. 4: Infants and Children with Late-Onset or Progressive Hearing Loss Will Be Identified at the Earliest Possible Time

In describing hearing loss, the terms "late onset" and "progressive" are frequently used together which may lead to

some people assuming that they are synonyms for the same condition. They are not. A progressive hearing loss is one that gets worse over time, whether the hearing loss is congenital or late onset. The term "late-onset hearing loss" should only be used when normal hearing was present at birth and a permanent hearing loss occurred later.

The Joint Committee on Infant Hearing (JCIH, 2007, p 899) recommends that

> Infants who pass the neonatal screening but have a risk factor should have at least 1 diagnostic audiology assessment by 24 to 30 months of age. . . . All infants should have an objective standardized screening of global development with a validated assessment tool at 9, 18, and 24 to 30 months of age . . . Infants who do not pass the speech-language portion of a medical home global screening or for whom there is a concern regarding hearing or language should be referred for speech-language evaluation and audiology assessment.

In 2004 (the latest data available), only 14 states were collecting risk indicator information from "all hospitals" and 17 states were collecting it for "some hospitals." Eight states reported that they received risk indicator data for ≥85% of all births. In many cases the state EHDI program reports the presence of the risk indicator to the child's PHCP and/or parent and takes no further action. States that were collecting risk factor data reported that they tried to do audiologic monitoring for 57% of the children that had risk indicators. Unfortunately, they were only able to complete "at least one audiologic monitoring during the first year of life" for 40% of those children where an attempt was made (NCHAM, 2013f). In a recent review of the literature, Beswick et al. (2012) found surprisingly little good evidence about costs or benefits of monitoring children who pass a newborn hearing screening test, but have one or more of the risk factors for hearing loss. They called for more large-scale, population-based research to assist with the development of evidence-based guidelines for monitoring the hearing status of children who have passed newborn hearing screening.

Clearly, detection of late-onset hearing losses should be a part of a comprehensive EHDI program. Although more work is needed to determine how this can be done most efficiently, recent research suggests that screening with OAEs is a viable alternative. Eiserman et al. (2008) reported results by lay screeners for more than 4,000 children in Early Head Start programs in four states using portable OAE equipment. One hundred and seven children (23.6 per 1,000 screened) were determined to have fluctuating conductive hearing losses requiring medical and/or audiologic treatment, and seven children (1.54 per 1,000 screened) were diagnosed with permanent hearing loss, including four who had passed their newborn hearing screening test. Foust et al. (2013) and Bhatia et al. (2013) reported on separate screening programs in which portable OAE equipment was used in federally funded clinics serving low-income and uninsured children in metropolitan areas. Foust and colleagues reported 3.55 children per 1,000 identified with permanent hearing loss based on 846 children screened, and Bhatia and colleagues reported 2.45 children per 1,000 identified with permanent hearing loss based on almost 2,000 children screened. These studies provide good evidence that OAEs are a viable tool for doing hearing screening of infants and young children. In its latest national survey of physicians, NCHAM (2013g) found that 29% report that they are doing hearing screening of infants and young children in their offices, and 66% of these report using OAE equipment as a part of their screening protocol.

Goal No. 5: All Infants with Hearing Loss Will Have a Medical Home

The American Academy of Pediatrics advocates that all children should have access to health care that is accessible, family centered, comprehensive, continuous, coordinated, compassionate, and culturally effective—often referred to as the Medical Home (Jackson et al., 2013). It is clear that services for infants and toddlers with hearing loss would be much better if families of children who are DHH were connected soon after birth to a PHCP who is familiar with their circumstances, is knowledgeable about the consequences and treatment of children who are DHH, and is known and trusted by the family.

Unfortunately, according to state EHDI coordinators, this is not the case for many infants and toddlers with hearing loss. Shulman et al. (2010) reported that only 73% of coordinators said that hospitals in their state contacted the PHCP when a child did not pass the newborn hearing screening test. NCHAM (2013f) reported that the name of the PHCP who will care for the baby during the first 3 months of life was known only for about 75% of newborns discharged from the hospital. Furthermore, many PHCPs are not well informed about issues related to early identification of hearing loss (NCHAM, 2013g). This is not surprising given the rapid changes that have occurred in our knowledge about identification and treatment of children who are DHH during the last 15 years. It is unrealistic to expect all PHCPs to remain up-to-date about a condition that affects only about 3 babies per 1,000. Thus, states must find ways of providing this information to PHCPs on an "as needed" basis. The American Academy of Pediatrics is actively working with state EHDI coordinators to develop such informational materials, but much remains to be done. According to MCHB (2010), State Title V Directors estimated that only 43% of children with special healthcare needs receive healthcare services in a setting that meets the minimal requirements for a medical home. State EHDI coordinators estimated that results about hearing screening tests were sent to medical home for 73% of the births, but it is unclear how frequently these results reached the correct PHCP (Shulman et al., 2010). In fact, NCHAM (2013g) found that 46% of

physicians said they never received information from the state EHDI program, and 68% reported that they never sent information to their state EHDI program.

Goal No. 6: Every State Will Have a Tracking and Surveillance System to Minimize Loss to Follow-Up

CDC currently awards funding to 52 states and territories to assist with the development and enhancement of improved tracking and data management systems that can be linked with other state public health information systems. A recent survey of public health agencies concluded that information from EHDI programs was the child health information most likely to be integrated with other health systems, but continued effort and improved coordination among agencies is still needed (Bara et al., 2009).

As noted in Figure 23.4, Loss to Follow-up/Loss to Documentation remains a serious problem with state EHDI programs being unable to document the hearing status of 35% of the newborns who do not pass the hearing screening test. Shulman et al. (2010) reported that hospitals report the results of hearing screenings to the state EHDI program using various methods, including paper forms, software developed specifically for this purpose, adaptations to the bloodspot screening cards, or electronic birth certificates. Some state EHDI programs mandate how reporting is to be done, but most allow each hospital to choose which system they will use. This means that only half the EHDI programs received screening results from all hospitals through a single method, the most common being a faxed or mailed paper form.

More systematic approaches such as those used in other countries would likely have better results. For example, well-established UNHS programs in the United Kingdom (UK National Screening Committee, 2013), Poland (Radziszdsska-Konopka et al., 2008), and the Netherlands (Nederlandse Stichting voor het Dove en Slechthorende Kind [NSDSK], 2007) report national screening rates of more than 95% with loss to follow-up/loss to documentation rates of less than 10%. It is interesting to note that low loss to follow-up/loss to documentation is achieved even though in England about 20% of the babies are not screened in the birth hospital and in the Netherlands 70% of the babies are screened at home, which would seem to be even more challenging for follow-up. Only four states in the United States (CA, IN, MA, and MI) reported loss to follow-up/loss to documentation rates of less than 10% in 2011 (CDC, 2013).

Eighty-five percent of EHDI programs received data about the screening outcomes of individual babies which means that most state EHDI programs are able to assist in follow-up with individual families. Linkages with other public health data systems are also expanding with 15 states reporting in a 2004 NCHAM survey that they had some type of linkage with newborn dried bloodspot screening programs, 13 with vital statistics and 4 each with immunization registries and early intervention programs (NCHAM, 2013f; Shulman et al., 2010). As these linkages are refined and stabilized, it will eliminate duplication and will mean that services to families can be better coordinated.

Goal No. 7: All States Will Have a System to Monitor and Evaluate Progress Toward the EHDI Goals and Objectives

Closely related to the development of tracking and data management systems is the implementation of systematic evaluation and quality assurance programs. As visualized in the CDC National Goals, an EHDI advisory committee in each state should assist with developing and maintaining the EHDI system. Almost all states have an EHDI Advisory committee that meets at least quarterly and has representation from diverse stakeholders including audiologists, parents of children with hearing loss, PHCPs, and early intervention providers. These committees have made good progress in overseeing the development of educational materials for PHCPs and parents. Coordinators in 78% of the states reported that they had good to excellent materials for educating parents about the states' EHDI programs. More work is needed in developing materials to educate PHCPs about EHDI and to educate parents of children who are DHH about communication options where only 53% and 56%, respectively, of EHDI coordinators said that they had good to excellent materials. Shulman et al. (2010) reported that 83% of the states had developed materials in languages other than English.

Systematic evaluation and monitoring of state EHDI programs is an area where more work is needed. NCHAM (2013f) found that states were using a variety of methods to gather information about the EHDI program, but only 18 states reported that a systematic evaluation of their state's EHDI program had been completed during the last 5 years. Interestingly, 10 of these 18 evaluations were internal evaluations conducted by state EHDI program staff and only 8 resulted in a written report.

Making progress toward achieving EHDI goals presumes that there is adequate funding to sustain the program. Unfortunately, most EHDI programs are on somewhat tenuous financial footing. NCHAM (2013f) found that almost two-thirds of the resources for operating EHDI programs came from the MCHB grants and CDC cooperative agreements that are viewed by Congress as temporary sources of support. Only 17% of the financial resources for state EHDI programs came from state appropriations and only six states provided more than half of the resources for their EHDI program from nonfederal sources. Shulman et al. (2010) reported that 42% of EHDI coordinators were unsure whether the program could be continued if federal funding were to be discontinued.

OPERATING EFFECTIVE NEWBORN HEARING SCREENING PROGRAMS

The preceding discussion about the goals of the EHDI system helps define where EHDI programs are headed and how well they are doing. Much has also been written about how to implement and operate an effective newborn hearing screening program (e.g., AAA, 2011; JCIH, 2007; White and Muñoz, 2013). Instead, of repeating similar information here, the following section discusses several issues about the operation of newborn hearing screening programs that are often overlooked.

Recognizing Newborn Hearing Screening as the Standard of Care

A newborn hearing screening program will only be successful if all the stakeholders are supportive and recognize its value and importance. One of the strongest rationales for providing a medical service is if it is recognized as the medical/legal "standard of care." Arguably, UNHS programs have now achieved that status, and hospitals and State Departments of Public Health are exposing themselves to significant liability risks if they are not operating effective hearing screening programs for all newborns.

Marlowe (1996) was one of the first to suggest that newborn hearing screening was becoming the actual medical/legal standard of care in the United States:

> Every medical and allied health practitioner and every hospital administrator should be keenly aware that they are held to a hypothetical standard of care whenever their professional conduct is being evaluated legally. . . . Definition of a standard of care is complicated by the fact that it is not usually articulated in a specific, identifiable form and it may be subject to clarification on a case-by-case basis should legal actions arise.

Even though there have not yet been court cases that definitively establish newborn hearing screening as the legal standard of care, healthcare providers and hospital administrators should be aware that newborn hearing screening seems to meet each of the following guidelines that have been used in the past for establishing a practice as the standard of care.

Expectations for a Reasonable Practitioner Under Similar Circumstances

An often cited case in determining what constitutes a standard of care in a particular situation was the 1898 *Pike v. Honsinger* case, in which the Court of Appeals decision stated that

> A physician . . . impliedly represents that he possesses . . . that reasonable degree of learning and skill . . . ordinarily possessed by physicians in his locality . . . [It is the physician's] duty to use reasonable care and diligence in the exercise of his skill and learning. . . . [he must] keep abreast of the times . . . departure from approved methods and general use, if it injures the patient, will render him liable.

The fact that newborn hearing screening is now being provided for over 98% of all newborns and have been successfully functioning in many parts of the United States for 15 years means that it would be difficult for any healthcare provider to successfully argue that UNHS programs should be viewed as experimental or unproven.

Support from Governmental, Professional, and Advocacy Groups

It is difficult to think of healthcare procedures that are not yet routinely implemented which have been endorsed by so many different authoritative groups ranging from the American Academy of Pediatrics to the National Institutes of Health to the March of Dimes—all of whom have concluded that UNHS is feasible to implement, results in earlier identification of hearing loss, and can be done with equipment which is accurate, practical to use, and economical.

Availability of Appropriate Technology to Implement the Practice

Ginsburg (1993) suggested that one of the criteria for establishing a standard of care

> . . . is when an inexpensive reliable device comes onto the market, the technology and concept of which have already been adopted by a group who specializes in the concept . . . a guideline becomes a standard of care when the device behind the guideline is available and readily usable. (p. 125)

Newborn hearing screening equipment is widely available, relatively inexpensive, and continually improving which means that it easily meets Ginsburg's standard of being "available and readily usable."

Selecting Screening Equipment and Protocols

Deciding what equipment to use and what protocol to follow is one of the first steps in setting up a newborn hearing screening program. During the past 20 years, many different pieces of equipment have been successfully used in newborn

hearing screening programs—transient-evoked OAEs, distortion product OAEs, and A-ABR. Each type of equipment has its proponents and detractors, but it is clear that the particular brand and type of equipment is not the primary determinate of whether a program will be successful.

In fact, the type and degree of hearing loss that is targeted by the screening program is much more important than the type and/or brand of screening equipment that will be used. This was demonstrated by Johnson et al. (2005) who evaluated how many infants are diagnosed with permanent hearing loss after passing a two-stage hearing screening protocol in which all infants are screened first with OAE and some are screened with A-ABR. In this protocol, no additional testing is done with infants who pass the OAE, but infants who fail the OAE are next screened with A-ABR. Those infants who fail the A-ABR screening are referred for diagnostic testing to determine if they have permanent hearing loss. Those who pass the A-ABR are assumed to have normal hearing and are not tested further. The objective of this multicenter study was to determine whether a substantial number of infants who fail the initial OAE and pass the A-ABR have permanent hearing loss at approximately 9 months of age.

Seven geographically dispersed birthing centers that had been successfully using a two-stage OAE/A-ABR screening protocol were included in the study. Almost 87,000 babies were screened at these centers during the period of the study. Infants who failed the OAE but passed the A-ABR in at least one ear (1.8%) were enrolled in the study and invited back for a diagnostic audiologic evaluation when they were on average 9.3 months of age. Diagnostic audiologic evaluations were completed for 64% of the enrolled infants (1,432 ears from 973 infants). Twenty-one infants (30 ears) who had failed the OAE but passed the A-ABR were identified with permanent bilateral or unilateral hearing loss, with most of them (77%) having mild hearing loss.

The results of this study suggest that if all infants were screened for hearing loss using the two-stage OAE/A-ABR hearing screening protocol currently used in many hospitals, approximately 23% of those with permanent hearing loss at approximately 9 months of age would have passed the A-ABR with the presumption that they had normal hearing. This happens in part because most currently used A-ABR screening equipment uses a 35-dBnHL click, which is best for identifying infants with moderate or greater hearing loss. Thus, program administrators should be certain that they are using equipment and protocols that are appropriate for identifying the type of hearing loss they wish to target.

Another example of why it is important to pay attention to selecting the equipment and protocol used in a newborn hearing screening program is the need to identify babies who are DHH because of auditory neuropathy spectrum disorder (ANSD). Such babies are a challenge to identify in some newborn hearing screening programs because they have normal or near-normal OAEs but an absent/abnormal auditory brainstem response (ABR). Thus, a program that uses only OAE for screening would miss such babies. Although it does occur in well-baby nurseries, most babies with ANSD have spent time in the NICU. For this reason, the JCIH (2007) recommends ABR technology as the only appropriate screening technique for use in the NICU. Berlin et al. (2010) provide additional information about the diagnosis and management of children with ANSD.

Regardless of the screening technology used, program administrators also need to be thoughtful about the number of screening tests that are done for each infant. To keep refer rates low at the time of hospital discharge, many programs repeat screening tests a number of times if the baby does not pass on the first test. JCIH (2007, p 903) cautions that "the likelihood of obtaining a pass outcome by chance alone is increased when screening is performed repeatedly." Because of this caution, many state EHDI programs have guidelines that babies should not be screened more than two or three times before leaving the hospital. Although screening a baby too many times is often not an efficient use of the screener's time, it does little to increase the probability of obtaining "a pass outcome by chance alone." Nelson and White (2014) had testers who were DHH repeat OAE tests in their own ear 1,000 times to determine how often a pass result would be obtained for an ear that has moderate to severe permanent hearing loss. They found an average of 1 false-negative result per 1,000 tests. Statistical probability calculations were then used to show that if the screening test was repeated three times for EVERY baby in a state with 100,000 annual births, only 1 baby who is DHH would be missed and 300 babies who are DHH would be correctly identified. If every baby were screened 10 times, only three babies who are DHH would be missed. In short, the negative consequences of repeat testing with respect to babies passing the screening test by chance have been greatly exaggerated.

Operating an Effective Newborn Hearing Screening Program

Regardless of the technology and protocol used, several procedural issues are important for an efficient, successful program.

Responsibility and Leadership

If everyone is responsible for a task, no one feels responsible for failure. Successful newborn hearing screening programs are being conducted in hundreds of different ways, but any successful program requires attention to detail and someone who makes sure all elements of the program are systematically addressed. The person responsible for day-to-day operation of the program does not need specific professional certification, but he or she needs to have good connections with the nursery, understand how screening

happens, and, most of all, be committed to the success of the program.

Ensuring Competent Screeners

Thousands of successful programs have demonstrated that screening can be done by a wide variety of people, including nurses, audiologists, technicians, healthcare assistants, volunteers, and students. Some states have laws about who can do hearing screening and how they must be supervised—others do not. Whoever does the screening should have received hands-on training under the supervision of someone who has already demonstrated his or her ability to be a successful screener. Although it is often said that practice makes perfect, it is more accurate to say that practice makes permanent. Consequently, it is important to provide timely feedback to people who are just learning to screen so errors can be corrected before they become ingrained. After initial training is completed, there should be regular one-on-one observation and feedback. It is useful to have a regular report of each screener's performance with regard to the number of babies screened, babies passed, invalid tests, and screens completed per hour of work. Such data are useful in identifying screeners who are having difficulty and need assistance. Feedback should be given in a way that is viewed as assistance instead of punishment.

When Should Screening Be Done?

Regardless of how screening is done, it will be faster and more effective if babies are quiet and the environment is not too noisy. Most programs do screening in the early morning or during the night when fewer people need to have access to the baby. However, depending on how the hospital nursery is organized, screening can be done at almost anytime that fits with the routine of that hospital. Whatever decision is made, dozens of other hospitals are doing it at approximately the same time, so there really is no wrong time to do newborn hearing screening.

Written Permission for Screening Is Seldom Necessary

In most hospitals, hearing screening is done routinely as a part of the standard medical care provided to all newborns. As with other procedures, parents are not expected to explicitly consent to each procedure. However, it is best if parents understand what happens during newborn hearing screening so they can make an informed decision about whether they want their baby to be screened. Such parent education can be accomplished with information in the preadmission materials, prenatal classes, media materials, or placed in the baby's crib. If, based on this information, parents do not want their baby to be screened for hearing, they have the right to refuse.

Communicating with Parents and Healthcare Providers

Many people have a stake in the results of a newborn hearing screening program. Parents are among the most important stakeholders because they have the long-term responsibility to ensure that the child receives appropriate care and services. They are also the ones who have the strongest feelings (but often limited experience) about what it means to have a child who is DHH. It is essential that each parent be told the results of their baby's hearing screening test and this should involve more than just being informed that the baby passed or failed. Based on data collected from a national sample of hundreds of parents using individual interviews and focus groups Arnold et al. (2006) concluded that "the most opportune time to begin discussion of newborn hearing screening is before the birth." Arnold et al. (2006) also provided suggestions and sample materials about how to communicate with parents at all stages of the EHDI process. A web-based instructional module based on these suggestions is available at NCHAM (2013b). Most successful hearing screening programs use a variety of materials (often available in multiple languages) to educate, inform, and follow-up with parents, including pamphlets about the screening program, parent education materials, letters sent to parents about the results of the test, and cards used to make return appointments for rescreens or diagnostic evaluations.

The discussion with parents about the result of the newborn hearing screening test is an ideal time to help parents understand that passing the newborn hearing screening test does not mean that the child will never have future problems with hearing or language development. Parents understand that the newborn hearing screening test only provides information about the status of the infant's hearing at the time of discharge. Late-onset loss can occur at any time for a number of reasons (JCIH, 2007). Parents should request another hearing evaluation at any time they have concerns about their child's hearing or language development.

It is also very important for the child's PHCP to understand that medical evaluation is an essential part of the diagnostic process and that healthcare providers are a critical part of that multidisciplinary team. It is also important that everyone involved in the baby's medical management understand how detrimental it is when the diagnostic process requires several months, instead of being completed within a few weeks. For babies without other medical complications, the goal should be to have a definitive diagnosis, be fit with hearing aids (if parents choose to do so), and begin early intervention within a few weeks of birth. There should be as system for notifying every baby's PHCP about the screening results for his or her patients with a clear recommendation of what should happen next. Few things will undermine the success of a newborn hearing screening program as much as the baby's PHCP telling the parent during a well-baby check that it is really not that important to

follow up with the outpatient screen or diagnostic evaluation procedures.

Does Hearing Screening Create Excessive Anxiety for Some Parents?

Many people (e.g., Nelson et al., 2008; Paradise, 1999) have suggested that UNHS creates unduly high levels anxiety, worry, and concern for parents and might even interfere with parent–child bonding—particularly for parents of babies who fail the initial screen and are found on subsequent testing to have normal hearing (the false positives from screening). Tueller (2006) found dozens of studies that had examined this issue, with most reporting that 4% to 15% of parents in the general population and 14% to 25% in the false-positive group experienced increased levels of anxiety. The problem with most of these studies is that there was no explicit basis for comparison (i.e., were parents any more worried about their child's hearing than they were about other aspects of the child's development?).

To more accurately assess whether the worry expressed by parents was unduly high, Tueller collected data from 191 mothers (split between those whose babies had passed the initial screening test and those who failed the initial test in the hospital and passed a rescreen when they were 1 to 4 weeks of age). Data were collected when the baby was 1 week of age, and again at 6 weeks of age (which was after the time that babies received a rescreen if they failed the initial screen). Mothers were asked to rate whether they were "not at all, somewhat, moderately, or very worried" about the baby's hearing as well as 20 other aspects of infant development (e.g., irritability, sleeping habits, eyesight). When babies were 1 week old, 14.6% of the mothers reported that they were moderately worried or very worried about their child's hearing (similar to what has been reported in other studies). But, hearing was ranked sixth on the list of 21 items about which they might be concerned and it was not statistically significantly different from 14 of the other items.

At 1 week of age, mothers whose babies had failed the initial hearing screening test ranked hearing as the item about which they were most worried, but it was not statistically significantly different from 15 of the other items. But, at 6 weeks of age (after the baby passed the hearing rescreen test) mothers ranked hearing as eighth on their list of possible concerns and none of the mothers indicated that they were either moderately or very worried.

Tueller's results suggest that mothers worry somewhat about lots of issues related to their new baby. If asked whether they are worried about only hearing, about 15% will say yes. But this is no different than the percentage who worry about other aspects of their child's development (e.g., eating, sleeping, irritability). Of course, newborn hearing screening programs should educate parents about the screening process and why hearing and language development are important. However, there is no convincing evidence that the newborn hearing screening process causes parents to be unduly concerned about their baby's hearing.

Complying with Federal Privacy Protection Laws

Successful EHDI programs share personally identifiable information about infants and young children among people who are responsible for screening, diagnosis, early intervention, family support, and medical home services. Many people involved with EHDI programs complain that federal privacy laws (e.g., HIPAA, FERPA, Part C Privacy Regulations) make it impossible for EHDI programs to be successful (Houston et al., 2010). Most of these concerns are based on misperceptions or false information about the requirements of those laws. For example, HIPAA expressly allows for sharing of information among healthcare providers to facilitate healthcare services and for reporting information to public health programs. There is nothing in HIPAA that prevents screening program personnel from reporting screening results to other hospitals, state EHDI programs, pediatricians, or Part C Early Intervention programs. All of these can be done even if informed consent is not obtained from parents (NCHAM, 2013h). To help parents be full partners in the EHDI process, it makes sense to inform them before sharing information about their family with anyone in the EHDI system. Even though it is not legally required under HIPAA, one of the best ways to ensure that parents are well informed is to have a signed consent.

FERPA and Part C Privacy Regulations are more restrictive than HIPAA, but these regulations are not in force until an agency that is receiving federal funds provides services to the child. Thus, in most cases, screening and diagnosis of hearing loss and referral to an early intervention program will be completed before the provisions of Part C Privacy Regulations or FERPA take effect. Once a child has been referred to Part C, information about that child cannot be given by the Part C program staff to the EHDI program, the audiologist who did the diagnostic evaluation, or a pediatrician—unless the parent provides informed consent. Effective strategies are listed below and examples of the forms and documents being used by state EHDI programs to support many of these strategies are available from NCHAM (2013h):

1. Coordinated consent forms that comply with the requirements of HIPAA and Part C Privacy Regulations can be used to streamline the referral process and to relieve parents of the burden of completing similar forms for the same purpose.
2. Memoranda of Agreement that designate EHDI programs as participating agencies of the Part C system are useful in those cases where EHDI is more than a primary referral source for child-find.
3. Parents should always be given copies of diagnostic evaluation reports, treatment plans, Individualized Family

Service Plans (IFSPs), and signed consent forms. This enables the parent to provide information at will and provide back-up documentation for services the child is receiving.
4. Although not required by HIPAA, FERPA, or Part C Privacy Regulations, state laws that mandate reporting of screening, diagnostic, and early intervention information to EHDI programs and to the child's pediatrician are often helpful.
5. The IFSP should include an option for parents to give permission for the document to be shared with EHDI staff, the child's pediatrician, and other healthcare providers.

Implementing the preceding strategies requires strong interagency and personal relations among key stakeholders, including EHDI programs, Part C Early Intervention programs, the child's pediatrician, and family support groups. Consistent training is usually needed at the community level to ensure that all stakeholders understand the importance of sharing information and helping families to be full participants in the process.

Data Management and Tracking

Arranging for a data and patient information management system is a task that is easy to procrastinate. The amount of information that needs to be managed continues to multiply as more and more babies are born. If a system is not in place when the screening programs starts, program staff will soon be overwhelmed in piles of paper and yellow sticky notes. The importance of including an effective information management system in newborn screening programs has been emphasized by the Joint Committee on Infant Hearing (JCIH, 2007, p 913):

> Information management is used to improve services to infants and their families; to assess the quantity and timeliness of screening, evaluation, and enrollment into intervention; and to facilitate collection of demographic data…[it is also] used in measuring quality indicators associated with program services.

An appropriate data management system depends on how the screening program is designed. In its simplest form, screening, diagnosis, and early intervention are each provided by a single source or homogenous group of sources. Infants flow seamlessly from the initial screening process to a diagnostic center and receive appropriate treatment or intervention (including family support). In this type of program, a data management system is relatively simple and straightforward. More commonly, however, the screening system has multiple screening sites, several diagnostic facilities, and many different providers who must be involved in the delivery of treatment, intervention, and family support services. Tracking infants through such a system, although challenging, is the only way to ensure that program goals are met.

Creating an effective data management system is one of the most challenging aspects of operating an effective newborn screening program. If all that was required was to count and report the total number of births, the number of infants screened, and the number who passed and failed, data management would be easy. When all the other information necessary to follow-up and track babies is added, designing a data management system becomes much more complex. Even the simplest of programs generates an astounding amount of data that can quickly overwhelm the capacity of a poorly conceived data management system.

Implementing an effective and efficient newborn hearing screening program is more difficult than it sounds and well-designed and managed data management systems play the following important roles:

1. A "safety net" to ensure that all babies are screened and to identify those babies who need, but have not received, follow-up screening or testing
2. A communication tool that automatically generates emails or letters to parents, healthcare providers, and/or education programs about the results of screening tests, follow-up procedures needed, and/or reminders of upcoming appointments
3. A protocol management assistant that reminds screening program personnel about who should be tested and what procedures should be followed
4. A quality assurance/quality improvement tool that identifies facilities or screeners who are performing above or below acceptable standards so that training and support can be efficiently targeted or superior performance recognized and rewarded
5. A system for documenting system performance so that reports can be made to funding agencies, public officials, consumers, and law makers about what the program is accomplishing and areas where additional resources are needed
6. A basis for integrating data from various health-related programs so that children and families can be provided with better and more efficient services.
7. A tool for collecting data to be used for research about such things as the prevalence, incidence, etiology, comorbidity, predictability, and treatment of various conditions.

The successful accomplishment of all of these purposes requires that the right data be collected in a timely manner and that the data are reliable and valid.

Not only does a good data management system help ensure that babies and their families are receiving timely and appropriate services, but it also helps to document what has been accomplished, identifies areas that need improvement, and provides information necessary for continued improvement and expansion. It is important that the creation and

operation of such systems be done thoughtfully and carefully, because computer-based systems are capable of generating incredibly large amounts of data. If not done carefully, administrators of newborn screening programs may find themselves drowning in information, but starving for knowledge. The key to success is to make sure that the purposes of a newborn hearing screening management system are thoughtfully considered by all stakeholders before a system is purchased or developed. Then the features and capabilities of the selected system must be carefully matched to those goals and purposes. The advice attributed to Mark Twain should be kept in mind, "Data is like garbage. You'd better know what you are going to do with it before you collect it."

CONCLUSIONS

The current status of EHDI programs in the United States is like the proverbial glass that can be viewed as being either half full or half empty. Certainly, the likelihood of an infant or toddler who is DHH receiving timely and appropriate services is better than ever. The substantial accomplishments of the last 25 years provide an excellent foundation for future progress.

- Ninety-eight percent of all newborns are now being screened for hearing loss prior to discharge and all states and territories have formally established EHDI programs.
- The fact that legislation or regulations related to UNHS have been approved in 43 states bodes well for the sustainability of these programs.
- Although not guaranteed for the long term, federal funding continues to be available for all states to refine, expand, and improve statewide EHDI programs and the Affordable Care Act covers hearing screening as a preventive service.
- There is substantial involvement and support from prestigious federal and professional organizations such as MCHB, CDC, NIH, the American Academy of Pediatrics, the American Academy of Audiology, the American Speech Language Hearing Association, and March of Dimes.
- Screening equipment and protocols continue to improve, and progress is being made on improving connections to diagnostic and early interventions programs and reducing the loss to follow-up/loss to documentation rates that have been so troubling for so long.

According to the National Goals established by CDC, all children who are DHH should be diagnosed before 3 months of age. But we are still a long way from achieving the more modest goal set by Dr. Koop in 1990 that "no child [would reach] his or her first birthday with an undetected hearing loss." To effectively identify children who are DHH and provide them and their families with the services they need, significant improvement must be made in the availability of pediatric audiologists, tracking and data management, program evaluation and quality assurance, availability of appropriate early intervention programs, and linkages with medical home providers.

In contrast to the early 1990s, there is now a solid research and an experiential basis for addressing all of these issues, but it will continue to require the commitment and resources of state health officials, hospital administrators, healthcare providers, and parents. As pointed out by Wilson and Jungner (1968, p 7 and 26)

> . . . in theory, screening is an admirable method of combating disease . . . [but] in practice, there are snags . . . The central idea of early disease detection and treatment is essentially simple. However, the path to its successful achievement . . . is far from simple though sometimes it may appear deceptively easy.

The issues that need to be resolved are complex and will require stakeholders to continue working together over a sustained period of time. As a result of continuing such work, infants and young children who are DHH will be able to acquire the "fundamental language, social, and cognitive skills that provide the foundation for later schooling and success in society" as foreseen almost 30 years ago in establishing the goals for *Healthy People 2000.*

CREDITS

Supported in part by the Health Resources and Services Administration (HRSA), Maternal and Child Health Bureau under Grant No. U52MC043916. The opinions and conclusions in this article are those of the author and do not necessarily represent the official position of the Health Resources and Services Administration.

FOOD FOR THOUGHT

1. Virtually all newborns in the United States are now screened for hearing loss before leaving the hospital. CDC's National Goals for EHDI Programs still recommend that "hospitals and others [report] information about risk factors for hearing loss to the state, who will monitor the status of children with risk factors and provide appropriate follow-up services." What are the pros and cons of continuing to monitor the status of children with risk factors with respect to issues such as identifying childhood hearing loss, costs, demands on the health care system, and burden for families?
2. Although the percentage of children failing a newborn hearing screening test who are lost to follow-up and or documentation is slowly declining, it remains a very significant issue. What approaches, programs, or initiatives are likely to significantly reduce the percentage of children being lost to follow-up?
3. There continues to be a critical shortage of audiologists who have the expertise, experience, and desire to provide comprehensive audiological services to infants and young children. What can be done to increase the number of fully qualified pediatric audiologists?

KEY REFERENCES

thePoint A full list of references for this chapter can be found at http://thePoint.lww.com. Below are the key references for this chapter.

American Academy of Audiology. (2011) Clinical practice guidelines: childhood hearing screening. Available online at: http://www.audiology.org/resources/documentlibrary/Documents/ChildhoodScreeningGuidelines.pdf (accessed on November 22, 2013).

American Academy of Pediatrics (AAP). (1999) Newborn and infant hearing loss: detection and intervention. *Pediatrics*. 103 (2), 527–530.

American Board of Audiology. (2013) Pediatric audiology specialty certification. Available online at: http://www.americanboardofaudiology.org/specialty/pediatric.html (accessed on November 22, 2013).

Arnold CL, Davis TC, Humiston SG, Bocchini JA, Bass PF, Bocchini A, et al. (2006) Infant hearing screening: stakeholder recommendations for parent-centered communication. *Pediatrics.* 117 (5), S341–S349.

Babbidge H. (1965) *Education of the Deaf in the United States: Report of the Advisory Committee on Education of the Deaf.* Washington, DC: US Government Printing Office.

Bara D, Mann YM, McPhillips-Tangum C, Wild EL. (2009) Integrating child health information systems in public health agencies. *J Public Health Manag Pract.* 15(6), 451–458.

Berlin CI, Hood LJ, Morlet T, Wilensky D, Li L, Mattingly KR, et al. (2010) Multi-site diagnosis and management of 260 patients with auditory neuropathy/dys synchrony (auditory neuropathy spectrum disorder). *Int J Audiol.* 49 (1), 30–43.

Bess FH, Paradise JL. (1994) Universal screening for infant hearing impairment: not simple, not risk-free, not necessarily beneficial, and not presently justified. *Pediatrics.* 93, 330–334.

Beswick R, Driscoll C, Kei J. (2012) Monitoring for postnatal hearing loss using risk factors: a systematic literature review. *Ear Hear.* 33 (6), 745–756. doi: 10.1097/AUD.0b013e31825b1cd9.

Bhatia P, Mintz S, Hecht BF, Deavenport A, Kuo AA. (2013) Early identification of young children with hearing loss in federally qualified health centers. *J Dev Behav Pediatr.* 34 (1), 15–21. doi: 10.1097/DBP.0b013e318279899c.

Centers for Disease Control and Prevention (CDC). (2004) National EHDI goals. Available online at: http://www.cdc.gov/ncbddd/ehdi/nationalgoals.htm (accessed November 22, 2013).

Centers for Disease Control and Prevention (CDC). (2013) 2011 Annual Data Early Hearing Detection and Intervention (EHDI) Program. Available online at: http://www.cdc.gov/ncbddd/hearingloss/ehdi-data2011.html (accessed November 22, 2013).

Darley FL. (1961) Identification audiometry: a report prepared by the Committee on Identification Audiometry of the American Speech and Hearing Association. Monograph supplement number 9. *J Speech Hear Disord.* 1–68.

Downs MP, Hemenway WG. (1969) Report on the hearing screening of 17,000 neonates. *Int Audiol.* 8, 72–76.

Eiserman WD, Hartel DM, Shisler L, Buhrmann J, White KR, Foust T. (2008) Using otoacoustic emissions to screen for hearing loss in early childhood care settings. *Int J Pediatr Otorhinolaryngol.* 72 (4), 475–82. doi: 10.1016/j.ijporl.2007.12.006.

Ewing IR, Ewing AWG. (1944) The ascertainment of deafness in infancy and early childhood. *J Laryngol Otol.* 59, 309–333.

Finitzo T, Albright K, O'Neal J. (1998) The newborn with hearing loss: detection in the nursery. *Pediatrics.* 102, 1452–1460.

Foust T, Eiserman W, Shisler L, Geroso A. (2013) Using otoacoustic emissions to screen young children for hearing loss in primary care settings. *Pediatrics.* 132 (1), 118–123. doi: 10.1542/peds.2012-3868. Epub June 3, 2013.

Ginsburg WH Jr. (1993) When does a guideline become a standard? The new American Society of Anesthesiologists guidelines give us a clue. *Ann Emerg Med.* 22, 1891–1896.

Goedert MH, Moeller MP, White KR. (2011) Midwives' knowledge, attitudes, and practices related to newborn hearing screening. *J Midwifery Womens Health.* 56 (2), 147–153.

Green DR, Gaffney M, Devine O, Grosse SD. (2007) Determining the effect of newborn hearing screening legislation: an analysis of state hearing screening rates. *Public Health Rep.* 122 (2), 198–205.

Houston KT, Behl DD, White KR, Forsman I. (2010) Federal privacy regulations and the provision of early hearing detection intervention programs. *Pediatrics.* 126, S28–S33. doi: 10.1542/peds.2010-0354G

Ireland PE, Davis H. (1965) The young deaf child: identification and management. Proceedings of a conference held in Toronto, Canada on 8–9 October, 1964. *Acta Otolaryngol Suppl.* 206, s1–s258.

Jackson GL, Powers BJ, Chatterjee R, Bettger JP, Kemper AR, Hasselblad V, et al. (2013) The patient-centered medical home: a systematic review. *Ann Intern Med.* 158 (3), 169–178. doi: 10.7326/0003-4819-158-3-201302050-00579

Johnson J, White KR, Widen JE, Gravel JS, James M, Kennalley T, et al. (2005) A multi-center evaluation of how many infants with permanent hearing loss pass a two-stage OAE/A-ABR newborn hearing screening protocol. *Pediatrics.* 116 (3), 663–672.

Joint Committee on Infant Hearing. (2007) Year 2007 Position Statement: principles and guidelines for early hearing detection and intervention programs. *Pediatrics.* 120 (4), 898–921.

Li X, Driscoll C, Culbert N. (2009) Investigating the performance of a questionnaire for hearing screening of school children in China. *Aust NZ J Audiol.* 31 (1), 45–52.

Mahoney TM, Eichwald JE. (1987) The ups and "downs" of high-risk screening: the Utah statewide program. *Semin Hear.* 8 (2), 155–165.

Marion Downs Hearing Center Foundation. (2013) Marion Downs Hearing Center, Colorado. Available online at: http://www.mariondowns.com (accessed November 22, 2013).

Marlowe JA. (1996) Legal and risk management issues in newborn hearing screening. *Semin Hear.* 17 (2), 153–164.

Mauk GW, White KR, Mortensen LB, Behrens TR. (1991) The effectiveness of screening programs based on high-risk characteristics in early identification of hearing impairment. *Ear Hear.* 12 (5), 312–319.

McCann DC, Worsfold S, Law CM, Kennedy CR, Mullee M, Petrou S, et al. (2009) Reading and communication skills after universal newborn screening for permanent childhood hearing impairment. *Arch Dis Child.* 94 (4), 293–297.

Meadow-Orlans K. (1987) An analysis of the effectiveness of early intervention programs for hearing impaired children. In Guralnick M, Bennett FC, eds. *The effectiveness of early*

intervention for at-risk and handicapped children. New York, NY: Academic Press; pp.325–357.

Mehl AL, Thomson V. (1998) Newborn hearing screening: the great omission. *Pediatrics.* 101, E4.

Moeller MP, White KR, Shisler L. (2006) Primary care physicians' knowledge, attitudes and practices related to newborn hearing screening. *Pediatrics.* 118, 1357–1370.

National Center for Hearing Assessment and Management (NCHAM). (2013a) *Global status of hearing screening.* Available online at: http://infanthearing.org/iEHDI/index.html (accessed November 22, 2013).

National Center for Hearing Assessment and Management (NCHAM). (2013b) *Home page.* Available online at: http://www.infanthearing.org (accessed November 22, 2013).

National Center for Hearing Assessment and Management (NCHAM). (2013c) *Policy statements regarding newborn hearing screening.* Available online at: http://www.infanthearing.org/resources_home/positionstatements (accessed November 22, 2013).

National Center for Hearing Assessment and Management (NCHAM). (2013d) *Legislative activities.* Available online at: http://www.infanthearing.org/legislative/summary/index.html (accessed November 22, 2013).

National Center for Hearing Assessment and Management (NCHAM). (2013e) *Financing and reimbursements.* Available online at: http://www.infanthearing.org/financing/index.html (accessed November 22, 2013).

National Center for Hearing Assessment and Management (NCHAM). (2013f) *State EHDI Program Survey.* Available online at: http://www.infanthearing.org/survey/2004statesurvey/index.html (accessed November 22, 2013).

National Center for Hearing Assessment and Management (NCHAM). (2013g) National survey of physicians' attitudes, knowledge, and practices regarding EHDI programs. Available online at: http://ehdimeeting.org/System/Uploads/pdfs/1314KarlWhite.pdf (accessed on November 22, 2013).

National Center for Hearing Assessment and Management (NCHAM). (2013h) The impact of privacy regulations: how EHDI, Part C, and health providers can ensure that children and families get needed services. Available online at: http://www.infanthearing.org/privacy/index.html (accessed November 22, 2013).

National Institutes of Health. (1993) *Early Identification of Hearing Impairment in Infants and Younger Children.* Rockville, MD: National Institutes of Health.

Nederlandse Stichting voor het Dove en Slechthorende Kind [NSDSK]. (2007) *Jaarverslag 2006.* Available online at: http://nsdk.nl/bijlagen/rapporten/2/ (accessed November 22, 2013).

Nelson HD, Bougatsos C, Nygren P. (2008) Universal newborn hearing screening: systematic review to update the 2001 American US Preventive Services Task Force Recommendation. *Pediatrics.* 122, e266. doi: 10.1542/peds.2007-1422.

Nelson L, White KR. (2014) *Incidence of false negative OAE newborn hearing screening results as a result of repeat screenings.* Paper presented at the 2014 National EHDI Meeting, Jacksonville, FL, April 14, 2014.

Newton VE, Macharia I, Mugwe P, Ototo B, Kan SW. (2001) Evaluation of the use of a questionnaire to detect hearing loss in Kenyan pre-school children. *Int J Pediatr Otorhinolaryngol.* 57 (3), 229–234.

Northern JL, Downs MP. (1991) *Hearing in Children.* 4th ed. Baltimore, MD, Williams and Wilkins, pp 2–3.

Paradise JL. (1999) Universal newborn hearing screening: should we leap before we look? *Pediatrics.* 103 (3), 670–672.

Radziszdsska-Konopka M, Niemczyk K, Grzanka A, Owsiak J. (2008) The universal newborn hearing screening program in Poland – five years of experience. Proceedings of NHS 2008 Conference – Beyond Newborn Hearing Screening: Infant and Childhood Hearing in Science and Clinical Practice, Cernobbio, Italy, June 19–21 2008, p 161.

Shulman S, Besculides M, Saltzman A, Ireys H, White KR, Forsman I. (2010) Evaluation of the universal newborn hearing screening and intervention program. *Pediatrics.* 126 (Suppl 1), S19–S27. doi: 10.1542/peds.2010-0354F.

Toward Equality. (1988) *A Report to the Congress of the United States: Toward Equality—Commission on Education of the Deaf.* Washington, DC: US Government Printing Office.

Tueller SJ. (2006) Maternal worry about infant health, maternal anxiety, and maternal perceptions of child vulnerability associated with newborn hearing screen results. Master's Thesis, Utah State University, Logan, UT.

UK National Screening Committee. (2013) NHS Newborn Hearing Screening Programme 2010-11–Annual Report and 2009–10 Data Report. Available online at: http://hearing.screening.nhs.uk/publications (Accessed November 22, 2013).

US Department of Health and Human Services (HHS). (1990) *Healthy People 2000: National Health Promotion and Disease Prevention Objectives.* Washington, DC: Public Health Service.

US Department of Health and Human Services (HHS). (1999) *Health Resources and Services Administration: Measuring Success for Healthy People 2010: National Agenda for Children with Special Health Care Needs.* Washington, DC: US Department of Health and Human Services.

US Preventive Services Task Force. (1996) Screening for hearing impairment. In: *US Preventive Services Task Force Guide to Clinical Preventive Services.* 2nd ed. Baltimore, MD: Williams & Wilkins; pp 393–405.

US Preventive Services Task Force. (2008) Universal screening for hearing loss in newborns: US Preventive Services Task Force recommendation statement. *Pediatrics.* 122 (1), 143–148.

Watkin PM, Baldwin M, Laoide S. (1990) Parental suspicion and identification of hearing impairment. *Arch Dis Child Educ Pract Ed.* 65, 846–850.

White KR. (1997) Universal newborn hearing screening: issues and evidence. Available online at: http://www.infanthearing.org/summary/prevalence.html (accessed November 22, 2013).

White KR. (2003) The current status of EHDI programs in the United States. *Ment Retard Dev Disabil Res Rev.* 9, 79–88.

White KR. (2006) Early intervention for children with permanent hearing loss: finishing the EHDI revolution. *Volta Rev.* 106 (3), 237–258.

White KR. (2011) Universal infant hearing screening: successes and continuing challenges. Keynote Address. Proceedings of Phonak's 5th International Pediatric Conference: A Sound Foundation through Early Amplification, Chicago, IL, November 8–10, 2010.

White KR, Behrens TR. (eds). (1993) The Rhode Island Hearing Assessment Project: implications for universal newborn hearing screening. *Semin Hear.* 14, 1–119.

White KR, Forsman I, Eichwald J, Muñoz K. (2010) The evolution of early hearing detection and intervention programs in the United States. *Semin Perinatol.* 34 (2), 170–179.

White KR, Maxon AB. (1995) Universal screening for infant hearing impairment: simple, beneficial, and presently justified. *Int J Pediatr Otorhinolaryngol.* 32, 201–211.

White KR, Muñoz K. (2013) Newborn hearing screening. In: Maddell JR, Flexer C, eds. *Pediatric Audiology: Birth through Adolescence.* 2nd ed. New York: Thieme Medical Publishers, Inc.; pp 31–44.

Wilson JMG, Jungner G. (1968) *Principles and Practice of Screening for Disease.* Geneva: WHO. Available online at: http://www.who.int/bulletin/volumes/86/4/07-050112BP.pdf (accessed November 22, 2013).

World Health Organization (WHO). (2010) *Newborn and infant hearing screening: current issues and guiding principles for action.* Outcome of a WHO informal consultation held at WHO Headquarters, Geneva, Switzerland, November 09–10, 2009.

CHAPTER 24

Assessment of Hearing Loss in Children

Allan O. Diefendorf

INTRODUCTION

Current policy in public health advocacy and primary healthcare delivery has focused on detecting hearing loss in infants and young children within the first 3 months of life through universal newborn hearing screening and timely diagnostic follow-up. This emphasis in hearing health care represents a standard of service delivery that has evolved over the past 45 years of advocacy by the Joint Committee on Infant Hearing (JCIH), and subsequently endorsed by the Centers for Disease Control and Prevention (CDC-P), the National Institutes of Health (NIH), and numerous coalitions of professional organizations.

It should be noted that all professional groups involved in the early detection of hearing loss have endorsed the terminology "early hearing detection and intervention (EHDI) programs." Experience has shown that, to successfully identify and serve infants and young children (including their families) with hearing loss, professionals had to expand their emphasis from screening to include comprehensive, coordinated, and continuous audiologic care, hence the adoption of the terminology EHDI. To be successful, all components of follow-up from newborn hearing screening need to be included to meet and serve the child's and family's complex needs, through coordinated care and collaboration with other professionals, as well as frequently communicated care plans that include intervention goals and strategies for hearing loss. Early referral for suspected hearing loss has evolved into best practice and is supported by undisputable evidence that the earlier confirmation of hearing loss occurs, the earlier intervention can begin, thereby increasing the likelihood of optimizing a child's full potential in all developmental areas.

The purpose of this chapter is to focus on the audiologic assessment and early diagnosis of infants and young children with hearing loss. Additionally, this chapter is developed with an emphasis on the following premise: Audiologic procedures must be age appropriate, outcome based, and cost-effective, and all procedures must have demonstrated validity and reliability. In addition, audiologic care must be comprehensive, collaborative, coordinated, culturally sensitive, and continuous.

Evidence Supports Early Detection of Hearing Loss

Undetected hearing loss in infants and young children compromises optimal language development and personal achievement. Without appropriate opportunities to learn language, children fall behind their hearing peers in language, cognition, social-emotional development, and academic achievement. However, research demonstrates that when hearing loss is identified early (prior to 6 months of age) and followed immediately (within 2 months) with appropriate intervention services, outcomes in language development, communication competency, and social-emotional development will be significantly better when compared with children with later identified congenital hearing loss (Moeller, 2000; Yoshinaga-Itano et al., 1998). It was also noted that when the same identification and intervention benchmarks are achieved (prior to 6 months of age), children perform as much as 20 to 40 percentile points higher on school-related measures (reading, arithmetic, vocabulary, articulation, intelligibility, social adjustment, and behavior) (Yoshinaga-Itano, 2003).

Early Detection Facilitates Favorable Outcomes

To achieve beneficial outcomes for children who are hard of hearing and deaf, families must be informed about how to find facilities and audiologists who are able to provide age-appropriate, comprehensive, and family-centered follow-up in a timely manner. The CDC-P provides resources to maximize and achieve this outcome (www.cdc.gov/ncbddd/hearingloss/ehdi-goals.html). To facilitate early intervention, audiologists must provide detailed diagnostic evaluation of hearing status within weeks of referral.

The diagnostic evaluation provides the first opportunity for developing a supportive relationship with the family and for initiating an audiologic care plan (diagnosis, counseling, referral if necessary, intervention, and ongoing care coordination as indicated). The interaction with the family during the diagnostic evaluation is critical because the support, counseling, guidance, and education a family receives

at this time helps to facilitate smooth transitions between referral source and early intervention programs. In turn, well-adjusted and well-informed families become empowered to make informed decisions, resulting in a unified approach to audiologic care plans and desired outcomes. Best practices indicate that each stage of the early detection process must be coordinated, collaborative, and communicated with all parties (families, pediatricians, relevant agencies). For a comprehensive review of these practices, see Chapter 44.

ESTABLISHING THE ETIOLOGY OF HEARING LOSS

As with all disorders, early diagnosis enables early intervention and improves prognosis. The importance of early audiologic diagnosis cannot be overstated; often the identification of hearing loss may be the first indication of a compromised sensory system or additional health problems. As the child's medical profile develops, a multidisciplinary team may become involved in the child's treatment plan.

After hearing loss is confirmed, consideration should be given to identify the etiology of the hearing loss. Current data indicate that 50% to 60% of congenital hearing loss is because of hereditary factors, with the balance due to environmental causes and, occasionally, interactions between genetics and environment (Dent et al., 2004). Table 24.1 provides a framework for delineating an etiology distribution of hearing loss with selected examples.

Historically, the past 30 years of demographic data have consistently reported that 30% to 40% of children with hearing loss have one or more *additional disabilities* (Gallaudet Research Institute, 2005). Table 24.2 lists physical and cognitive/intellectual conditions that are frequently reported to accompany hearing loss. (See Chapter 31 for more information regarding concomitant disorders.)

TABLE 24.1

Framework for Delineating the Etiology of Hearing Loss

Hereditary Hearing Loss: 50–60% of Hearing Loss	
Single gene (nonsyndromic: 60–70%)	Gap junction protein, beta 2: The GJB2 gene provides instructions for connexin proteins which form channels called gap junctions that permit transport of nutrients. Channels made with connexin 26 help to maintain correct levels of potassium ions
	Myosin VI: The MYO6 gene provides instructions for proteins that play a role in the development and maintenance of stereocilia
	TECTA: The TECTA gene provides instructions for a protein called alpha-tectorin. This protein interacts with other proteins to form the tectorial membrane
Chromosomal abnormality (syndromic: 30–40%)	Usher syndrome: Hearing loss accompanied with eye disease
	Jervell and Lange-Nielsen syndrome: Hearing loss accompanied with cardiac defect
	Pendred syndrome: Hearing loss accompanied with thyroid goiter (endocrine defect)
	Wardenburg syndrome: Hearing loss accompanied with pigment defect (eyes, skin, hair)
	Alport syndrome: Hearing loss accompanied with chronic nephritis (kidney disease)
Environmental Hearing Loss: 25–35% of Hearing Loss	
Maternal influences	Viral disease: Cytomegalovirus, rubella, mumps
	Bacterial disease: Bacterial meningitis, bacterial sepsis
	Environmental: Lead, mercury, radiation
	Drugs: Substance abuse
Complications after birth	Respiratory distress
	Hyperbilirubinemia
	Peri/intraventricular hemorrhage
	Perinatal asphyxia
Combination of Genes and Environment: <10% of Hearing Loss	
Medicines, but only in individuals who have certain mutations in genes	Various

TABLE 24.2

Physical and Cognitive/Intellectual Conditions Accompanying Hearing Loss in Children

Physical Conditions Accompanying Hearing Loss in Children	
Pulmonary	Pulmonic stenosis Cystic fibrosis Asthma
Cardiovascular	Cardiac conduction defect Cardiac rhythm disturbance Obstructive congenital heart defects
Ophthalmic	Legal blindness Progressive eye degeneration Cataracts
Neurologic	Cerebral palsy Muscular dystrophy Myoclonic epilepsy
Orthopedic	Syndactyly Hip dysplasia Rickets
Endocrine	Thyroid gland disorders Adrenal gland disorders Pituitary gland disorders
Immunologic	HIV/Aids Allergies Leukemia
Renal	Chronic nephritis Malformations of the kidney Infantile renal tubular acidosis
Cognitive/Intellectual Conditions Accompanying Hearing Loss in Children	
Intellectual disability	
Specific learning disability	Attention deficit disorder Auditory perceptual difficulties Visual perceptual difficulties
Emotional/ behavioral disorders	Hyperactivity Obsessive/compulsive behavior Aggressive/abusive behavior

Tables 24.1 and 24.2 should be viewed together. The manner in which multiple conditions coexist and the manner in which multiple conditions are expressed (by degree of involvement) contribute to each child's unique developmental profile and subsequent rehabilitation recommendations.

As the widespread use of newly developed vaccines decreases the prevalence of etiologies such as measles, mumps, rubella, and childhood meningitis, the percentage of early-onset hearing loss attributable to genetic etiologies should increase. The multidisciplinary team should include a geneticist to help establish the etiologic basis for hearing loss to improve outcomes in coordinated and comprehensive care and to provide families necessary information as they consider planning for subsequent children.

COMPREHENSIVE SERVICE DELIVERY IN AUDIOLOGY

The goal of the initial diagnostic assessment of infants and young children is to confirm or rule out hearing loss, to quantify the extent and configuration of hearing loss, and to determine the functional health of the auditory system. Additionally, comprehensive assessment should be provided for each ear even if only one ear was in question from the newborn hearing screening.

Thorough and valid diagnostic information is essential in a timely manner to permit early intervention services. For this reason, age-appropriate, cost-effective, and efficient diagnostic protocols are critical at the earliest opportunity for optimizing early intervention services. In addition, an emphasis on timely scheduling of diagnostic follow-up should occur for children and families whose initial audiometric profile is incomplete.

Additionally, follow-up visits are essential to monitor infant's overall auditory status and changes in hearing, as well as verifying the development of auditory skills and functional use of hearing. It should also be recognized that ongoing surveillance for fluctuating and/or progressive hearing loss must be closely coordinated between the child's primary care provider (medical home) and other service providers in the child's habilitation plan.

Age-appropriate Assessment

The use of age-appropriate techniques in diagnostic audiology is vital in the evaluation of infants and young children. It requires clinicians to select differential diagnostic techniques that are within the child's developmental capabilities. Because children undergo rapid sensory, motor, and cognitive development and because some children will present with multiple health concerns and subsequent developmental challenges, it is vital that assessment tools are appropriate for the neurodevelopmental status of the child. Factors (physical and cognitive) that can influence developmental status must be considered prior to the selection of an assessment strategy. For example, with the goal of audiologic diagnosis by 3 months of age (JCIH, 2007), it is likely that some infants with hearing loss and multiple health concerns will not have had these other disorders identified at the time of the audiologic assessment. Some problems may be relatively easy to identify (e.g., cerebral palsy). Others (e.g., learning disabilities or Asperger syndrome) are more difficult to identify. For this reason, audiologists face challenges when dealing with the variety of characteristics that may

be encountered. Of course, knowledge of the handicapping conditions will enable audiologists to plan effective and age-appropriate diagnostic strategies. Therefore, audiologists should investigate factors involved in each individual by use of interviews, case histories, assessments by other professionals, and close observation. As pointed out by Tharpe (2009), audiologists must be mindful of the possibility that unexpected and/or undiagnosed conditions may influence their testing and subsequent audiologic outcomes.

The Test Battery Approach

It is strongly recommended that the initial audiologic test battery to confirm hearing loss include auditory-evoked potentials (AEPs), physiological measures, and, when developmentally appropriate, behavioral methods. The use of any test alone for assessing children's hearing sensitivity is discouraged. The desirability of using multiple tests in clinical practice is based on the complex nature of the auditory mechanism and the fact that auditory dysfunction may result from pathology at one or more levels of the auditory system. In test battery selection, audiologists use test procedures that are outcome based and cost-effective, and greater weight should be given to the results of those tests for which validity and reliability are highest. If test results are not in agreement, the reason for the discrepancy must be explored before arriving at an audiologic diagnosis.

Jerger and Hayes (1976) promoted the concept of a test battery approach so that a single test is not interpreted in isolation but, instead, various tests act as *cross-checks* of the final outcome (see Chapter 8). Thus, audiologists benefit by having a battery of tests appropriate for the diagnosis of hearing loss in infants and young children. As pointed out by Turner (2003), the purpose of multiple tests is to increase the accuracy of audiologic diagnosis. This accuracy is accomplished when appropriate diagnostic tests are selected for the individual's test battery. Subsequently, tests must be carefully administered and data appropriately interpreted, followed by a clinical decision based on the entire test battery. After weighing the agreement/disagreement between tests, audiologists can reach a confident diagnosis. Clinical decisions involve not only test selection, but also determining the number of tests administered during a single session, interpreting individual test data, and drawing conclusions based on the performance of the entire test battery.

Multicultural Considerations

The US population is becoming increasingly multicultural. Over one-third of the population is represented by racial and ethnic minority groups and approximately 25% of the population over age 5 speaks a language other than English at home (Institute of Medicine, 2002). In addition, these population statistics are expected to increase over the next 30 years.

The Institute of Medicine (2002) reviewed over 100 studies that assessed the quality of health care for various ethnic and racial minority groups. The report highlighted existing disparities in the quality of health care provided to minorities and concluded that very few of these differences in healthcare quality for minorities can be attributed to patient attitudes and preferences. More significant contributors to these disparities include two primary areas: (1) The characteristics of healthcare systems and environmental factors which include cultural or linguistic barriers (e.g., lack of interpretative services), and fragmentation of healthcare systems; and (2) discrimination, including biases, stereotyping, and uncertainty. The report suggests that these disparities might originate from the provider, that is, prejudice against minorities, clinical uncertainty when interacting with minorities, and stereotypes held about minorities.

Linguistic barriers can minimize effective communication and compromise rapport with a family, both leading to poor compliance with audiologic recommendations. Clearly, linguistic barriers degrade the family's expectations of quality service delivery. Poor communication may also discourage familiarity with accessing resources and keeping future appointments.

A useful skill that audiologists must master is the effective use of interpreters when dealing with families who speak other languages. Some techniques to consider for ensuring successful exchange of information include using short concise sentences and pausing frequently between them to allow the interpreter to organize and effectively translate. Additionally, it is necessary to be mindful of nonverbal gestures that may be interpreted differently by different cultures and certain words that may not have the same meaning when translated. Appropriate eye contact and appropriate physical space between individuals are also important considerations in achieving effective communication.

As healthcare professionals, audiologists need to be respectful of and responsive to the needs of a diverse patient population by increasing their knowledge and skill in cultural competence. Perceptions regarding hearing loss differ across cultures. Some cultures may not attribute much significance or urgency to hearing loss and families may only pursue services relative to their specific attitudes and beliefs. Additionally, people from different cultures may not view hearing loss as a disability and may choose to embrace only those recommendations/interventions that are consistent with their cultural values, religious beliefs, or societal norms.

Continued Surveillance

Concern for hearing loss must not stop at birth. It must be recognized that limiting hearing screening to the neonatal period will result in a significant number of children with hearing loss excluded from the benefits of early detection. Some newborns and infants may pass initial hearing screening but require periodic monitoring of hearing to detect

delayed-onset hearing loss. The JCIH (2007) identified 11 risk indicators associated with either congenital or delayed-onset hearing loss, including family history and maternal illness (www.jcih.org/ExecSummFINAL.pdf). Therefore, heightened surveillance of all infants with risk indicators is recommended.

Unlike the newborn and school-age populations when nearly all of the children can be evaluated in hospitals or schools, preschoolers are generally not available in large, organized groups that lend themselves to universal detection of hearing loss. For this reason, an interdisciplinary, collaborative effort is particularly important for this age group. Physicians and other professionals who make up the child's medical home and other professionals who specialize in child development should be included in the planning and implementation of hearing screening programs to increase the likelihood of prompt referral of children suspected of hearing loss.

PEDIATRIC AUDIOLOGIC PRACTICES

Audiologic assessment of infants and young children includes a thorough patient history, otoscopic inspection, and both physiological and behavioral measures. As stated earlier, the need for a battery of tests in pediatric assessment is essential to optimally plan for and meet the diverse needs of the pediatric population.

During the diagnostic process, the integrity of the auditory system is evaluated for each ear, and the status of hearing sensitivity across the frequencies important for speech understanding is described, as well as the type and configuration of hearing loss. In turn, these data provide essential information for medical management when indicated and the data required to begin the amplification process. Finally, these data are further used as a baseline for continued audiologic monitoring.

Patient History

The case history is a component of the audiologic assessment that guides the audiologist in constructing an initial developmental profile based on the child's physical, developmental, and behavioral status. The outcome of the patient history is particularly important because it will often guide the strategy for the audiologic assessment and for making subsequent recommendations and referrals. It can also serve as the first cross-check on the audiologic test outcome.

The patient history process begins as early as calling the child's name in the waiting room. Time spent observing the child from the waiting room to the assessment area can provide valuable information about the child's physical and developmental status.

Some background health-related and developmental information (American Speech-Language-Hearing Association, 2004) can be obtained prior to the initial evaluation (by telephone and/or mail). The case history questionnaires are used to gather information regarding the family history, birth history, developmental history (including hearing and speech/language development), and medical history. This information guides the clinician to generate questions as each child's status may require a meaningful history intake process. See "Food for Thought" about the kinds of questions an audiologist should consider while taking a case history.

It is recommended that time for obtaining a patient history be viewed as a valuable opportunity to observe and interact with the child and family, build rapport, and instill confidence and comfort in the child and family, while acquiring patient-specific history information. The single most important thing that may determine the comfort level of the child is the communication between the family (parent/caregiver) and the audiologist.

Otoscopic Inspection

Otoscopy is intended as a general inspection of the external ear and tympanic membrane for obvious signs of disease, malformations, or blockage from atresia, stenosis, foreign bodies, cerumen, or other debris. To promote health and prevent disease it is appropriate to change specula before examining each ear to avoid cross-contamination. In addition, because several audiologic assessment procedures require the insertion of a probe into the external auditory canal, visual inspection serves to verify that there is no contraindication to placing a probe in the ear canal. For further information dealing with infection control, see Chapter 46.

AUDIOLOGIC TEST BATTERY: BIRTH TO 6 MONTHS OF AGE

The goal for both behavioral and physiological procedures is ear-specific assessment. Determining hearing sensitivity for each ear facilitates medical/surgical diagnosis and treatment, selecting and fitting amplification when appropriate, establishing baseline function, and monitoring auditory status when progressive, fluctuating, or late-onset hearing loss is suspected.

Electrophysical Assessments

The audiologic test battery for young infants, birth through 6 months, consists primarily of AEPs to estimate hearing thresholds at specific audiometric frequencies. These measures currently include the auditory brainstem response (ABR) and the auditory steady-state response (ASSR). Other physiological measures that make up the test battery include distortion product otoacoustic emissions (DPOAEs) or transient-evoked otoacoustic emissions (TEOAEs) and acoustic

immittance. (For a more complete discussion of these procedures, see Chapters 11, 13, 14, and 15.)

Measurement of AEPs provides accurate estimates of threshold sensitivity in young infants, with the clinical goal of achieving accurate estimations of frequency-specific, behavioral thresholds from the AEPs. In turn, these data provide the basis for appropriate medical intervention and facilitate early intervention, particularly the selection and fitting of amplification systems.

The differences between estimated and actual thresholds vary for ABR and ASSR. The four most important variations are associated with (1) frequency of the stimulus, (2) degree of hearing loss, (3) age of the patient, and (4) duration of the recording. Of utmost importance is controlling/maximizing the signal-to-noise ratio in all AEP recordings. In infants, noise levels are often higher than in young adults, and the responses are smaller. When the duration of the recording is longer, the residual noise of the electroencephalogram (EEG) is less, making small, near-threshold responses easier to recognize. Specifically, the amplitude of the residual noise decreases by the square root of the number of sweeps. A longer time spent in the recording will result in less background EEG noise, making it easier to recognize responses and be more precise in threshold estimation.

Additionally, because residual EEG noise levels vary from patient to patient, in part because of the level of muscle activity in the recording, it is optimal when patients are relaxed or sleeping during recording. Many children in this age group can be tested during natural sleep, without sedation, using sleep deprivation with nap and feeding times coordinated around the test session. However, active or older infants may require sedation to allow adequate time for acquisition of high-quality recordings and sufficient frequency-specific information.

Moderate Sedation

To gain the cooperation of some infants and young children during measurements of AEPs, sedation may be required. However, sedation of pediatric patients, with or without other health problems, may be contraindicated because of factors such as airway obstruction, apnea, cardiopulmonary impairment, and hypoventilation. Therefore, moderate sedation should only be administered by, or in the presence of, individuals skilled in airway management and cardiopulmonary resuscitation. Additionally, the oversight by a sedation team and the availability of age- and size-appropriate equipment, medications, and continuous monitoring are essential during procedures and for resuscitating a child should an adverse sedation event occur.

The ASSR is a synchronized brainstem response elicited by a continuous frequency-specific stimulus that is modulated (i.e., frequency and/or amplitude modulated) and presented at a given frequency. The recorded response is generated in the EEG response rather than specifically in the auditory brainstem pathway, as is the case with the ABR. Whereas the ABR response is determined through the identification of peaks and troughs in the time domain, the presence or absence of the ASSR is determined through statistical algorithms in the frequency domain.

One shortcoming of the ABR technique is that only one ear and one frequency can be tested at the same time (single-frequency sequential techniques). Another challenge of the ABR to brief tones is that detection of a response in the waveform depends on skilled, subjective assessment of replicated responses, allowing for error in judgment of the presence of responses depending on the experience of the clinician.

On the other hand, ASSRs are detected objectively using statistical tests; as such, their detection does not rely on the experience of the clinician. In addition, the ASSR technique may have other advantages over the ABR beyond automated response detection, including increased frequency specificity, the ability to test simultaneously at multiple frequencies, resulting in more time-efficient protocols, and higher stimulus presentation levels for comprehensive description of severity of hearing loss in the severe-to-profound categories (Picton et al., 2005; Stapells et al., 2004).

The advantage of a test battery in a comprehensive diagnostic assessment of frequency-specific thresholds is that multiple tests enhance our ability to "cross-check" results to arrive at the most responsible diagnosis. This is certainly the case in ASSR and ABR evaluations where the combination of each approach will facilitate the accuracy of low-frequency threshold measures, delineate the complexity of varying configurations of hearing loss, facilitate the accuracy of bone-conduction–measured outcomes, and delineate site-of-lesion assessment, particularly in the diagnosis of neural hearing loss.

If there are risk indicators for neural hearing loss (auditory neuropathy/auditory dyssynchrony [AN/AD]) such as hyperbilirubinemia or anoxia, then audiologic assessment should include click-evoked ABR. When recording a high-level (80 to 90 dB normal hearing level [nHL]) click ABR, responses should be measured separately for condensation and rarefaction single-polarity stimuli, and responses should be displayed in such a way as to identify the cochlear microphonic (CM) (i.e., superimposing averages to identify out-of-phase components). In these instances, precautions must be taken to distinguish the CM from a stimulus artifact.

The JCIH (2007) has included neural hearing loss (e.g., AN/AD) in infants admitted to the neonatal intensive care unit in their targeted definition of hearing loss. The audiologic community must be vigilant concerning this disorder because intervention and management is different from those with sensory hearing loss.

Otoacoustic emissions (OAEs) expand the pediatric audiology test battery by providing a physiological means of assessing preneural auditory function. OAEs are generated by the outer hair cells in the cochlea and serve as an indirect

measure of these cells. OAEs are not, in and of themselves, necessary for hearing, nor are they a mechanism of hearing, but rather, they reflect the status of structures that are necessary for hearing (see Chapter 19).

Transient-evoked OAEs (TEOAEs) are elicited following a transient (click) stimulus at approximately 80 dB peak sound pressure level (SPL). Although the transient click stimulus is a broadband stimulus that is not frequency specific, the response is analyzed in the frequency domain, thus providing information across frequencies from 500 to 5,000 Hz, although test performance is best for mid to high frequencies. DPOAEs are elicited following stimulation with two tones. DPOAEs are measured in response to two tones (primaries) that interact to produce nonlinear distortions in the cochlea. The two tones are typically selected so that the frequency ratio between the tones (f2/f1) is 1.22, which is known to produce the largest distortion product at most test frequencies in humans.

Evoked OAEs occur in response to an external auditory stimulus and are present in nearly all normal-hearing individuals. Thus, the presence of OAEs is consistent with normal or near-normal–hearing thresholds in a given frequency region. Response criteria typically include SNR and/or have a response reproducibility of greater than an established percentage at defined frequencies (see Chapter 19 for further details). Schemes for trying to determine the degree of hearing loss and/or predicting thresholds using OAEs have been investigated (Dorn et al., 2001; Gorga et al., 2003). Although some strategies have met with success, there is so much variability that threshold predictions should be viewed cautiously.

Because of their remarkable stability over time within the same ear, OAEs are also useful for monitoring the status of disease conditions that are progressive, including certain genetic disorders such as Usher syndrome (Meredith et al., 1992). In addition, over shorter time courses, OAEs are advantageous for monitoring the effects of treatments that are potentially damaging to the ear, like those involving such ototoxic antibiotics as tobramycin (Katbamna et al., 1999) or such antitumor agents as cisplatin (Ress et al., 1999).

Immittance

Acoustic immittance measures are an integral part of the pediatric assessment battery. Clinical decisions should be made on a quantitative assessment of the tympanogram, including consideration of equivalent ear canal volume, peak-compensated static acoustic admittance, tympanometric width, gradient, and tympanometric peak pressure (see Chapters 9 and 10 for a detailed description of the components of the acoustic immittance test battery).

Under the age of approximately 4 months, interpretation of tympanograms may be compromised when a conventional low-frequency (220- or 226-Hz) probe tone is used (Purdy and Williams, 2000). As such, a higher probe-tone frequency (e.g., 1,000 Hz) is recommended for identifying middle ear disorders in infants less than 4 months of age, and normative data for 1,000-Hz tympanometry are available for neonates and young infants (Margolis et al., 2003). Once a child reaches the age of 7 months, a low-frequency (226-Hz) probe tone is appropriate. Between 5 and 7 months of age, however, there is still a possibility of false-negative tympanograms in ears with middle ear effusion. Therefore, use of a 1,000-Hz probe tone for tympanometry in this subset of infants is recommended when attempting to identify middle ear effusion.

When a quantitative assessment of a tympanogram is used, care must be taken to ensure that there is correspondence between the graphic representation of the tympanogram and the absolute quantities indicated. With the pediatric population, sometimes there are irregularities in the tympanogram shape (because of movement artifact, crying, or vocalizing) that may be mistaken for a tympanogram peak by the instrument and may provide misleading absolute values.

In addition to providing confirmation of middle ear status, acoustic reflex measurement is useful in the interpretation of other components in the audiologic test battery. That is, the acoustic reflex may provide supplemental information dealing with the functional status of the middle ear, cochlea, and brainstem pathway (see Chapter 10). For example, acoustic reflexes are absent when AN/AD exists (Starr et al., 1996). Although there are insufficient data for routine use of acoustic reflex measurements in the initial diagnostic assessment under the age of 4 months, the acoustic reflex should be used to supplement the test battery at older ages. Together, these measures are fundamental components of the pediatric audiology test battery.

AUDIOLOGIC TEST BATTERY: INFANTS 6 MONTHS OF AGE AND OLDER

Assessing *auditory sensitivity* in older infants and children (>6 months) can be completed efficiently and effectively with behavioral, physiological, and AEPs as necessary. The audiologic test battery for infants 6 months of age and older includes conditioned behavioral audiometry (either visual reinforcement audiometry [VRA] or conditioned play audiometry [CPA]), speech detection and/or recognition measures (i.e., speech recognition threshold), acoustic immittance, and OAEs.

It is also important to establish *auditory responsivity* in selected children. For example, for children who have just received a cochlear implant, it is necessary to observe their initial responses to suprathreshold sounds. Additionally, children with multiple disabilities, including intellectual challenges, also represent a population for whom auditory responsivity is essential. Auditory responsiveness can be evaluated with behavioral observation audiometry (BOA), described in the next section.

AEPs should be performed, as necessary, when behavioral measures are not sufficiently reliable to provide ear-specific estimates of type, degree, and configuration of hearing loss or when these data are necessary to support other clinical questions (e.g., neurologic status). Importantly, the desire for behavioral hearing test results should not delay the selection and fitting of amplification when valid and reliable frequency-specific threshold information is available by AEPs.

As valuable as physiological procedures are in the early confirmation of hearing loss, the audiologist inevitably returns to behavioral testing to substantiate test results and monitor a child's hearing longitudinally. As advocated by Tharpe (2009), "We must bear in mind that behavioral tests provide an indication of how an individual uses his or her hearing, a very important factor when considering management needs" (p. 667).

Behavioral Observation Audiometry

This section will describe techniques in which the audiologist observes a child's responses to sounds to estimate hearing levels. However, before reviewing these techniques, a caution to the reader is essential: Unconditioned behavioral observation techniques with infants and young children are easily confounded by poor test–retest reliability and high inter- and intra-subject variability. This places cautious limitations on the use of BOA for determining *auditory sensitivity.* Therefore, BOA is no longer recommended for assessing frequency-specific threshold sensitivity in newborns, young infants (<5 months), or those children whose developmental disabilities preclude them from learning operant conditioning procedures. However, another goal in pediatric assessment is to examine auditory function by examining *auditory responsivity.* Although AEPs can quantify auditory sensitivity in infants with compromised cognitive function, BOA may provide useful insight into the quality of the child's auditory responsiveness. BOA may also provide an estimate of functional capabilities useful in planning intervention for selected children. That is, the audiologist can predict potential difficulties in auditory development and recommend aural habilitation strategies intended to improve the child's functional use of sound.

Procedural Guidelines

Infants whose motor, cognitive, and/or social development is under 6 months of age generally display a variety of reflexive and orienting responses to external stimuli at suprathreshold levels. Improving the accuracy of observing and judging the presence of these behaviors, however, often requires the use of multiple examiners/observers. That is, a number of examiners/observers are necessary to judge response behaviors to reduce two common errors of observation: (1) Judging that a response occurred when, in reality, there was no response and (2) judging that no response occurred when, in reality, a response did occur.

Observers monitor a range of behaviors, for example, head or limb reflex, increased motion, decreased motion, whole-body startle, eye widening, nonnutritive sucking, searching, eye blink or flutter, localization, smiling, laughing, and pointing. Because of behavior variability, it is important to minimize judgment errors by a single examiner. To add objectivity to BOA, observers should not be informed about presentation levels, should wear headphones with masking during the assessment process, and should be unaware of previous test results.

Response behaviors seen during BOA can be separated into those that are attentive-type, orienting behaviors (e.g., increased and decreased motion, eye widening, searching, localization, smiling, laughing, pointing) and those considered reflexive (e.g., head or limb reflex, whole-body startle [Moro reflex], sucking, eye blink or flutter). Analyzing response behaviors may provide useful information in children with developing auditory behaviors, as well as determining how youngsters attach functional meaning to sound.

Renshaw and Diefendorf (1998) listed three categories into which results of BOA testing may be placed: (1) No observable response to sound, (2) responses only to high-intensity stimuli (70 to 80 dB HL), and (3) responses to relatively soft and comfortable stimuli (30 to 50 dB HL). These categories provide some delineation about results obtained from BOA testing. In concert with BOA as a test of *auditory responsivity* (not *auditory sensitivity),* the categories promote the use of BOA as a behavioral measure, useful to support physiological findings and to verify the presence of a general level of functional hearing.

Instrumental (Operant) Conditioning: Basic Principles

The behavioral assessment of infants and young children can be approached through instrumental conditioning paradigms, specifically through an operant conditioning procedure. Operant behavior is frequently spoken of as wilful or purposeful behavior. In the operant procedure, a behavioral response is elicited by a target stimulus and is then *controlled* by the consequences of that behavior (introducing positive reinforcement). Skinner (1953) stated the term "operant" emphasizes the fact that the conditioned behavior *operates* to generate the desired consequences (receiving positive reinforcement).

In operant conditioning, a stimulus is used as a cue for the listener to respond with a specifically defined behavior (e.g., head-turning for infants). In turn, operant behavior (the head-turning response in infants) is increased by the application of positive reinforcement. Positive reinforcement is used to strengthen the response (operant) behavior and keep the child in an aroused and motivated state to continue to discriminate the target stimulus.

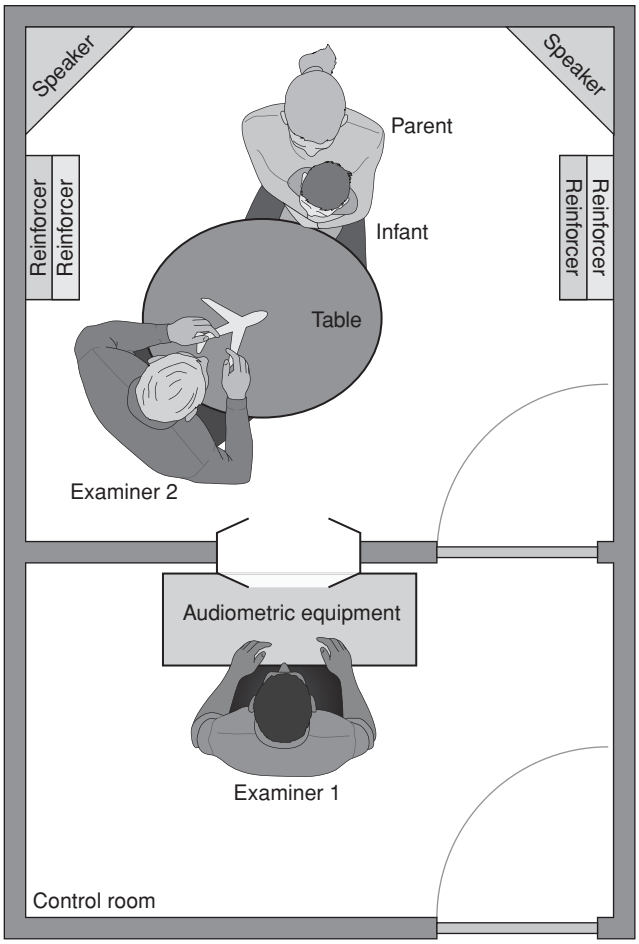


FIGURE 24.1 Test room arrangement commonly used in visual reinforcement audiometry (VRA). A second examiner maintains the interest of an infant in a midline position.

Test Room Arrangement

Figure 24.1 presents a room arrangement commonly used for VRA. The audiologist in the control room has full view of the testing situation. The ability to selectively darken only the audiologist's side of the test booth can be helpful. The infant, parent, and a second examiner are located within the test suite. The visual reinforcers, whether housed in smoked plexiglass enclosures (three-dimensional animated toys) or presented via monitors, are within 3 to 4 feet of the child and placed at 90-degree angles to the side of the child. Providing visual reinforcers on both the right and left of the child increases the complexity and novelty of the VRA procedure. From an instrumentation standpoint, it is a simple and relatively inexpensive matter to use several visual reinforcers on each side, in a stacked configuration. The application of multiple visual reinforcers serves to postpone habituation (Thompson et al., 1992), increasing the number of VRA responses that can be obtained in a given clinical visit.

The second examiner, seated in front of the infant, maintains the infant's head in a midline position by quietly encouraging the child to observe passively or to casually, but quietly, play with colorful, nonnoisy toys (Figure 24.1). An appealing toy is manipulated by the examiner in front of the infant as a distracter. The examiner's role is to maintain the infant's attention at midline and return the infant to this position once a response is made and reinforcement is completed. The audiologist must be creative in keeping the child alert and in a listening posture without the child becoming so focused on the activity. The toys used for this purpose should be appealing but not so attractive as to overly occupy the infant's attention. The closer the audiologist is to the child, the more easily the child is engaged in the activity. If the infant under test shows too much interest in the colorful, nonnoisy toys, then the potential exists for reduced responding during testing or for elevated response levels because of decreased attention. Conversely, if the examiner and toys are not sufficiently interesting, the likelihood of false responses (random head-turning toward the visual reinforcer) will be high.

Often, a touch of the hand on a child's shoe or leg will quietly redirect the child to a midline position. Actually sitting on the floor in front of the child allows for a totally unobstructed view of the child for the control room audiologist and being located slightly below the level of the child is a nonthreatening position. Clearly, the challenge for the second examiner is to balance between distracting the child without overly entertaining the child and consuming too much of the child's attention.

A single examiner approach from the control room can be accomplished by using a distracting "centering toy" positioned at midline in the test room for maintaining a child's midline gaze. Although the use of a "centering toy" is a frequent alternative to the more classical approach of using two examiners, this approach distances the examiner (in the control room) from the infant (in the exam room). Although a single examiner may be more cost-effective and practical in busy clinical settings, eliminating the second examiner reduces the ability to maintain the infant at midline with multiple distraction toys, that is, the "centering toy" is always the same toy, is somewhat noisy, and may be overly distracting for some infants. The fact that the "centering toy" is usually not housed in a dark plexiglass box raises the concern that the constant viewing of the colorful toy may eventually compromise the infant's interest in the visual reinforcement.

Depending on the child's acceptance, insert earphones are preferable over standard headphones. Inserts are useful for a number of reasons, including their comfort and light weight, their increased interaural attenuation compared to conventional earphones, and the reduced risk of ear canal collapse. And, although sound field data alone are better than no data, individual ear data are always preferable and the ultimate goal.

Visual Reinforcement Audiometry

In audiology, an operant discrimination (target) procedure conditions a child to discriminate changes in the listening environment. This approach is used as a threshold procedure where the target stimulus (e.g., puretones, warbled tones, filtered noise, speech) is *discriminated from a quiet background.* This specific procedure in audiology is called visual reinforcement audiometry (VRA), in which the child is reinforced with a visual "reward" (moving toys or video clips) for detecting the stimulus by responding with a head-turn. As demonstrated by Moore et al. (1977), audiometric signals (e.g., puretones or warble tones, and human speech) have limited reinforcing properties. Consequently, a positive reinforcement having high interest value (appealing to the infant) must be used to prolong response behavior in operant conditioning.

This approach also has application in assessing suprathreshold sound discriminations (i.e., visually reinforced infant speech discrimination—VRISD). In this application of the operant discrimination procedure, an infant is conditioned to *detect a discriminative stimulus from a repetitious background.* For example, when trying to determine developmental changes in speech sound discrimination, infants are presented with two of contrastive stimulus items (e.g., /va/ from /sa/) where the discriminative challenge is to detect a change (e.g., the syllable /va/) from a repetitious background stimulus (e.g., the syllable /sa/). Again, the infant is reinforced for discriminating the stimulus change by responding with a head-turn (Eilers et al., 1977).

Age Considerations and VRA

Normally developing infants initiate head-turns toward a sound source in the first few months of life. In fact, by the time a normally developing infant has reached a chronologic age of 5 to 6 months (Moore et al., 1977), this developmental behavior coupled with operant conditioning enables audiologists to implement VRA.

The success of VRA is related to the fact that the response, followed by visual reinforcement, is well suited to typically developing children between 6 months and 2½ years of age. However, delays in a child's developmental age can influence assessment outcomes in VRA.

Developmental age adjustments that must be considered in VRA include the influence of prematurity and the influence of mental age on VRA performance. Moore et al. (1992) concluded that VRA performance is related to corrected age for prematurity. They studied 60 premature infants (36 weeks of gestation or less) at corrected ages of 4 to 9 months. Their results imply that premature infants with a corrected age of 8 or 9 months are likely to perform acceptably in response to VRA (can be conditioned and respond with high success before habituation to task); that premature infants with a corrected age of 6 or 7 months may perform but with less success (can be conditioned but have limited responses before habituation to the task); and that premature infants with a corrected age of 4 or 5 months are not likely to respond to the VRA procedure. A comparison of these data to results of previous studies on full-term infants demonstrates that although full-term infants are likely to respond with high clinical success to VRA by a chronologic age of 6 months (Moore et al., 1977), premature infants are not likely to respond to VRA with good clinical success until approximately a corrected age of 8 months.

Widen (1990) also evaluated VRA as a function of developmental age in premature, high-risk babies. Clearly, the developmentally mature babies were more often tested successfully (the ability to be conditioned and provide threshold for at least one stimulus). The data from Moore et al. (1992) and Widen (1990) are highly consistent, that is, both reports indicate that VRA success with premature infants is related to corrected age and that VRA success with these infants is greater as they approach 8 to 9 months corrected age.

Why is it that premature infants, even after prematurity is corrected for, lag several months behind normally developing, full-term infants? Premature infants have been shown to display significantly poorer performance on standardized measures of mental ability when compared to full-term infants of the same postpartum age (Rubin et al., 1973). Kopp (1974) concluded that preterm infants engage in less cognitive exploration compared with full-term infants, which may also account for reduced motor development.

Several studies have reported on the use of VRA in children with Down syndrome and other developmental disabilities. Greenberg et al. (1978) reported on the use of VRA in 46 children with Down syndrome between the ages of 6 months and 6 years. As would be expected, the proportion of successful tests increased as age increased. Because it is expected that chronologic age would be a very poor predictor of success with the VRA procedure in these children, the Bayley Scales of Infant Development (Bayley, 1969) were used to provide an estimate of developmental level. If children with Down syndrome are considered on the basis of their developmental age, in contrast to chronologic age, it would be logical to assume that results might be similar to those found with normally developing infants. However Moore et al. (1977) found that normally developing infants 6 months of age and older accomplished the VRA procedure with a high rate of success, but Greenberg et al. (1978) found that individuals with Down syndrome did not achieve a high rate until 10 to 12 months BSID mental age equivalent. These investigators further pointed out that when one is predicting potential success with the VRA procedure for children with Down syndrome, the BSID mental age equivalent score provides the most distinct distribution between successful and unsuccessful tests, with the dividing point being a BSID mental age equivalent of at least 10 months. Similarly, Wilson et al. (1983) reported that 80%

of the children in their study with Down syndrome were testable by 12 months of age using VRA.

Although these data provide guidance, attempting behavioral audiologic evaluation on children presenting with any neurologic or development condition, including Down syndrome, under the age of 10 months is encouraged. In a clinical setting with a diverse and complex patient population, it is helpful to obtain any behavioral information that leads to more informed decision making and recommendations.

In summary, when VRA is implemented audiologists must consider (1) corrected age adjusted for prematurity rather than chronologic age or (2) mental age/developmental age when disparities exist between corrected age and the child's developmental status. Of the two predictors of VRA performance (corrected age and mental age), corrected age may be the more practical one to use in most cases because it can be obtained from parental report, case history information, and/or hospital records and does not require testing to determine mental age.

Conditioning in VRA

In clinical assessment, the first phase of VRA is the conditioning process. An examiner must be skilled in response training and sensitive to the various stages of response acquisition. Evidence suggests that different signals (e.g., tones, filtered noise, speech) are equally effective during the conditioning phase (Primus and Thompson, 1985).

Response shaping is critical to the success of the operant procedure. Two different, but commonly used approaches can be implemented to condition the head-turn response: (1) *Pairing* a suprathreshold auditory stimulus with the visual reinforcer or (2) presenting a suprathreshold auditory stimulus and *observing a spontaneous* response from the infant, *followed by* activation of the visual reinforcer. Successful completion of the training phase is the achievement of a pre-established criterion of consecutive head-turn responses (usually two, but no more than three consecutive responses following the response shaping trials). If the criterion is not reached, retraining is necessary until the criterion is met. The number of training trials needed before phase 2 trials begin varies, but the training phase is usually brief.

For the child with special needs, conditioning may take longer to establish the conditioned behavior. Successful completion of the conditioning phase occurs when the child is making contingent responses and random head-turning is at a minimum.

A key to response shaping is the presentation of a suprathreshold stimulus. For most infants, suprathreshold will be 30, 50, and, in some cases, 70 dB. Occasionally, some children, particularly those children with moderately severe to severe hearing loss, may require 90 dB or higher to qualify as a suprathreshold stimulus. Because hearing status is unknown, suprathreshold estimates are also unknown. Therefore, the possibility exists that the stimulus selected to shape the response behavior might be inaudible. Given that many infants referred for diagnostic work-up can be expected to have normal hearing, the most efficient test is one that uses a low starting level, approximately 30 dB. However, failure to condition rapidly should alert the audiologist to a potential equipment/calibration problem or a child who requires a greater starting intensity for conditioning. Attention to either issue must be immediate for a successful outcome.

When the infant's head-turn is not being shaped via air conduction, a bone oscillator can be placed on the infant's mastoid, in the hand, or rested against the arm to use as a vibrotactile stimulus. The traditional conditioning procedure is initiated, that is, pairing the stimulus with the reinforcement. The stimulus usually selected for bone-conducted conditioning is a 250-Hz narrowband noise presented at 60 dB HL. An infant with severe or profound hearing loss with no other developmental disabilities will show appropriate behavioral responses as long as the stimulus is salient (can be felt, even if not heard). Responses are obtained in this manner; subsequently, insert ear phones or headphone presentations follow with starting intensity levels dependent on the bone-conduction responses.

If the child fails to display conditioned responding, other issues such as compromised physical status, developmental delay, or immaturity are raised. For example, Condon (1991) noted several cognitive attainments necessary for a child to be assessed reliably using VRA. The child must be developing object permanence (i.e., knowing that objects exist in space and time, even when the child can no longer see them or act on them) and the ability to anticipate the reappearance of at least partially hidden objects, discover simple causality (i.e., an event or behavior is dependent on the other for its occurrence) and means–end relationships (i.e., behaviors that result in anticipated outcomes), and use simple schemes to explore toys.

Successful completion of training occurs when the child is making appropriate responses and random head-turning/false responses are at a minimum. Excessive false responses suggest that the infant is not under stimulus control. As such, audiologists should focus on two factors to improve clinical outcomes: (1) Reinstitute response shaping or (2) increase the distraction level of the activity to engage the child's interest at a midline position before presenting the auditory stimulus.

Following successful response shaping, the test phase of VRA begins (Figure 24.2). Signal intensity is attenuated 10 dB after every "yes" response or increased 5 dB after every "no" response (descending and ascending technique). Testing progresses until the stopping criterion (four reversals in the threshold search) has been achieved. Thresholds are defined as the mean of the four reversal points. Note: Thresholds are often called "minimum response levels (MRLs) in VRA

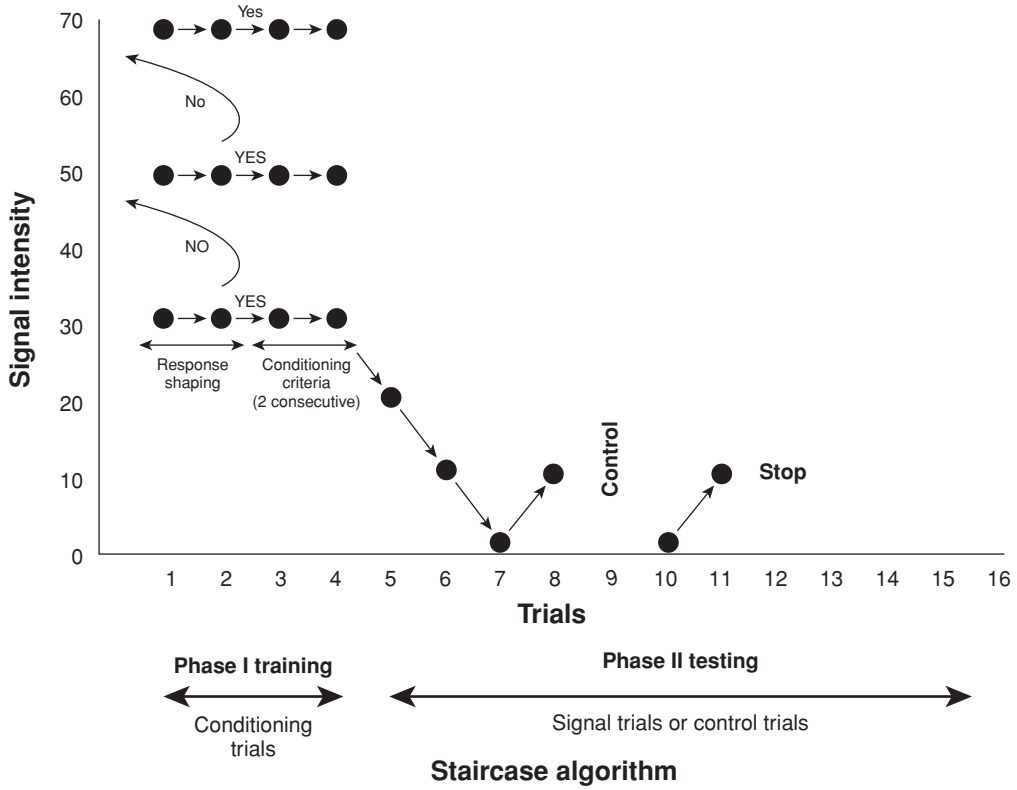


FIGURE 24.2 Algorithm commonly used in visual reinforcement audiometry (VRA).

because the term MRLs "serves as a reminder that improvement in response behavior at lower hearing levels should be anticipated with maturation" (Matkin, 1977, p. 130).

Procedural Guidelines

The recommended trial duration (incorporating the signal and response interval) is approximately 4 seconds (Primus, 1992). That is, although the signal duration is approximately 4 seconds, this 4-second duration also defines the interval of time during which a response should be judged to be present or not. Head-turn responses outside of the 4-second interval are typically not interpreted as valid responses, and therefore not reinforced. For some children with developmental and/or physical challenges, it may be necessary to increase the trial duration (beyond 4 seconds) during which a head-turn response is acceptable. However, by increasing trial duration beyond 4 seconds, audiologists also risk an increase in judging false responses as valid responses.

Conditioned head-turns are visually reinforced only for correct responses that occur during signal trials. Between the ages of 6 and 17 months, Lowery et al. (2009) demonstrated that infants are not more or less attentive to a dynamic video than to the three-dimensional animated toy as effective visual reinforcement. Therefore, when using colorful, three-dimensional toys as visual reinforcers it is sometimes judicious to use the light initially (showing the toy), eventually adding animation. By starting with the light only, the audiologist can gauge any potential for a fearful response to the three-dimensional toy. The novelty and strength of visual reinforcement may be preserved longer by introducing more complex reinforcement (light plus animation) as the testing progresses. The novelty of VRA is clearly strengthened with somewhat older children by using moving images generated by a digital video disc (DVD) player/monitor. Schmida et al. (2003) used digital video with 19- to 24-month-old children. Their results demonstrated a greater number of head-turn responses before habituation when viewing video reinforcement than when viewing conventional animated toy reinforcement. These results support the hypothesis that the complex and dynamic nature of the video reinforcement would be more effective in achieving a greater number of responses than the conventional toy reinforcer prior to habituation in the 2-year-old age group.

In general, a 100% reinforcement schedule (reinforcement for every correct response) results in more rapid conditioning, yet more rapid habituation. Conversely, an intermittent reinforcement schedule produces slower conditioning but also a slower rate of habituation. Consequently, most clinicians recommend a protocol that begins with a 100% reinforcement schedule and then gradually shifts to an intermittent reinforcement schedule.

Primus and Thompson (1985) compared a 100% reinforcement schedule to an intermittent reinforcement schedule with 2-year-old children. The two reinforcement

schedules resulted in no differences in the infants' rate of habituation or the number of infant responses to stimulus trials. These findings provide an excellent guideline for delivering reinforcement. Since Primus and Thompson's data suggest that withholding reinforcement should not affect the amount of response behavior, reinforcement should only be provided when the audiologist is certain about the validity of an infant's head-turn response. The risk of reinforcing a random head-turn is that it may lead to confusion for a child during the test session and increase the child's rate of false responding. Conversely, failure to reinforce a valid head-turn will not degrade subsequent responding. In this scenario, withholding reinforcement for a valid but ambiguous response is simply viewed as intermittent reinforcement.

Reinforcement duration is also a factor influencing response outcome from children around the age of 2 years (Culpepper and Thompson, 1994). Decreasing the duration of a child's exposure to the visual reinforcer (e.g., 4 to 0.5 seconds) results in an increase in response behavior and a decrease in habituation. Audiologists may increase the amount of audiometric information obtained from children by decreasing their exposure to the visual reinforcer. For the child with special needs who may have a slower response, the visual reinforcer should be activated for a sufficient length of time for the child to very briefly observe it, but prolonged visual reinforcement should be avoided.

Control Trials

Ensuring valid behavioral assessment outcomes depends on separating true responses from false responses during threshold acquisition. Infants are likely to produce a number of false responses during clinical assessment. To quantify false responding behavior in VRA, the use of control trials are necessary.

False responses are monitored by inserting control trials in the staircase algorithm (see Figure 24.2). A head-turn observed during a control trial is evidence of false responding. Thus, it is possible to systematically estimate errors or chance responding (false responses during signal trials) by calculating the number of false responses during control trials. Moore (1995) recommended that one out of four presentations should be a control trial and that test results are questionable if the false-positive rate exceeds 25%. Eilers et al. (1991) suggest that a false alarm rate of 30% to 40% is acceptable and adopting such a rate as acceptable does not compromise the accuracy of thresholds for clinical assessment. Clearly, high false alarm rates (>50%) require the audiologist to further consider that test results may be inaccurate.

Excessive false responses suggest that the infant is not under stimulus control. In this situation, audiologists should focus on two factors to rectify clinical outcomes: (1) Reinstituting phase 1 shaping and conditioning and (2) increasing the entertainment level of the distraction activity to engage the child's interest and attention at a midline position before starting a test trial. When in the test room, an examiner must be able to choose from a variety of toys available and judge when a toy change in either direction (enhanced distraction and more entertaining, or reduced distraction and less entertaining) is necessary to maintain the child's midline focus and optimum response readiness.

Occasionally, overactive parents can bias their children to respond, thereby resulting in excessive false responses. Therefore, parents may need to wear headphones through which music or noise is delivered.

Threshold determination in audiometry is based on the lowest intensity level where responses are obtained approximately 50% of the time. In VRA, as the staircase algorithm is executed, how many reversals should be required before identifying the hearing threshold? Too few reversals may sacrifice response accuracy. However, too many will increase test time, in turn reducing the number of stimulus presentations that could be spent obtaining thresholds to other stimuli. Assessing a desired stimulus may be stopped once the infant has exhibited between three and four response reversals (Eilers et al., 1991). Eilers and her colleagues found that using six rather than three response reversals before discontinuing the threshold search had minimal effect on threshold. Yet, tests with a three-reversal stopping rule were significantly shorter than those with six reversals. As stopping rules are increased from three to six, there is about a 50% increase in the number of test trials, with no improvement in response accuracy. These results suggest that, by using relatively few reversals to estimate threshold, a staircase algorithm may be shortened to increase efficiency without sacrificing accuracy. Thus, there is no need to continue testing beyond three or four reversals since the results obtained are not substantially better.

Thresholds obtained with the VRA procedure for infants 6 to 12 months of age have been shown to be within 10 to 15 dB of those obtained from older children and adults (Nozza and Wilson, 1984). In addition, VRA thresholds are similar across the age span (6 to 24 months) and show good reliability when compared to thresholds obtained from the same child at older ages.

Throughout testing, audiologists must consider that the next response to a test stimulus may be the child's last response. However, audiologists can influence attention and motivation by being flexible with their clinical decision making. Often, if a child begins to habituate to a specific stimulus, response behavior can be increased by using a different stimulus, a different transducer, or moving to the other ear. This approach to clinical assessment can optimize air conduction/bone conduction, another puretone versus speech, and switching ears. Thompson et al. (1992) also demonstrated that when 1-year-old children habituate to testing and are given a 10-minute break, the children return and are likely to provide a significant amount of additional information. Even a few additional responses in the same

session may provide just enough additional information to be confident that hearing loss is not a factor concerning speech/language development and communicative functioning, thereby eliminating the need for a costly follow-up clinic visit.

Conditioned Play Audiometry

Operant conditioning of behavioral responses to sound continues to be an effective approach for older children. What changes as children age, however, are the operant behaviors and the reinforcement that is used. Similar to operant conditioning in VRA, CPA uses positive reinforcement to increase response behavior. In CPA, children learn to engage in an activity (e.g., putting rings on a spindle, putting pegs in a board, dropping or stacking blocks, putting together simple puzzles) each time they hear a test signal. These activities are usually fun and appealing to children, are within their motor capability, and represent a specific behavior that is used to denote a deliberate response to a stimulus.

When teaching children to perform CPA, it is usually not difficult to select a response behavior that children are capable of performing, as long as the audiologist is intuitive in matching the child's motor skill with an appropriate play activity. CPA follows the traditional operant conditioning paradigm of stimulus → response → reinforcement, in which the play activity/motor activity is the response behavior, and social praise or other positive reward is the reinforcement. Three decisions are needed in play audiometry: First, the audiologist must select a response behavior that the child is capable of performing. Second, the audiologist must consider how to teach the child to wait, listen, and respond only when the auditory signal is presented. The third decision is what social reinforcement (the most common reinforcement with young children) the audiologist should give that is natural and genuine at the appropriate time and interval.

Separation of response behavior and reinforcement is essential in CPA. Although the play activity is fun for the child, it is not the reinforcement. A separate reinforcement is essential to minimize habituation and maximize repeated response behavior. In addition to social praise, other forms of reinforcement have been suggested. Tokens that can be traded for small toys at the end of the test session, unsweetened cereal, and a changing computer display screen all have been used successfully with play audiometry.

Children with multiple health concerns present unique challenges during audiometric evaluation using CPA. Challenges to consider include obtaining verifiable responses, choosing reinforcements that will interest the child, and response time. For children who have visual and hearing impairments, Holte et al. (2006) suggest using tactile cues (bone oscillator or simple touch) to train a child to the CPA task. For youngsters with limited gross motor/fine motor skills, a variety of responses (e.g., finger swing, hand motion, arm motion, eye motion, visual gaze) can be used to trigger an electronic switch, in turn activating a computer screen programmed for appropriate visual reinforcement. The goal is to select the most appropriate task and the most appropriate reinforcement while at the same time recognizing the physical limitations that may compromise the child's success. If the physical demands are too great, then the task will detract from maintaining a listening posture. If the task is too simple, the child will have less motivation to participate and will tire of the task. The critical decision for the audiologist is to select a specific operant behavior that is used to denote a specific response to a stimulus.

In general, the rate of success in obtaining detailed information with CPA is limited for children under the age of 30 months. However, some 2-year-olds can be conditioned to play audiometry (Thompson et al., 1989). In addition, when 2-year-olds are proficient with CPA, there is a greater likelihood that they will provide more responses before habituation than they would if tested by VRA. Because overlap exists between VRA and CPA as suitable techniques with children in this age range, the successful evaluation of a younger child with CPA ultimately depends on the following: The audiologist's observational skills of the child's developmental/maturational level, the interpersonal skills established between the audiologist and child, and the experience/comfort level of the audiologist with young children.

Striving to improve behavioral testing techniques is important because behavioral tests always are ultimately expected to define the response profile of young children. In addition, behavioral tests provide the critical link between AEPs and the child's demonstration of functional hearing.

TESTING SPEECH THRESHOLDS AND RECOGNITION

Speech Thresholds

Because language and vocabulary are emerging in infants and young children, it may not be feasible to establish a traditional speech reception threshold (SRT). An alternative approach is the determination of a speech detection threshold (SDT).

The SRT and SDT represent different criteria (intelligibility vs. detectability). The SRT is recognized as the intensity at which an individual is able to identify simple speech materials approximately 50% of the time. The SDT may be defined as the level at which a listener may just detect the presence of an ongoing speech utterance (e.g., bai-bai-bai presented with an overall duration of approximately 2 to 4 seconds). Speech can be detected at intensity levels lower than it can be understood, on the order of 8 to 12 dB.

The child who is ready for play audiometry typically has a communication strategy to express needs and wants at a more sophisticated level, whether with oral speech, signs, or a communication board. Family members often describe

various communication skills that the child possesses, such as following commands, pointing to body parts or pictures in a storybook, or identifying colors. The audiologist is then able to expand the test battery to include an SRT rather than an SDT. Additionally, at an age where play audiometry is successful, the SRT should be accomplished with insert earphones. The lighter weight of insert transducers coupled with increased comfort facilitates placement of the bone-conduction transducer for obtaining bone-conducted SRTs for each ear.

A spondee picture board can be very helpful in obtaining an SRT from a child who may be reluctant to respond in an unfamiliar test situation. Regardless of whether the response is verbal or pointing to a picture, it is recommended that a preliminary step in determining an SRT for young children is familiarizing the child with the test stimuli and eliminating those words that are not within the child's receptive vocabulary. The use of either picture or object pointing rather than a verbal response will require that the number of items be limited to less than 10. Otherwise, the visual scanning task and the demands placed on memory and attention become contaminating variables.

The utilization of a carrier phrase, such as "point to" or "show me," will often serve to focus the child's attention to the auditory task at hand. Moreover, since a child's attention span is limited and test time can be a factor, it is often more expedient to work in 10-dB rather than 5-dB steps when establishing an SRT.

The bone-conducted SRT can be extremely useful in obtaining additional data from children, and although it is typically underused, it is readily available to audiologists. The bone oscillator will deliver clear speech stimuli without any need for calibration correction or modification.

A bone-conducted SRT can offer valuable information in a very short period of time. Often the child will tolerate the bone oscillator during the more entertaining speech reception task but will not tolerate it for tonal testing. A frequently asked question regarding the use of the bone oscillator for speech reception testing relates to the potential for a false threshold that results in a vibratory response rather than a hearing response. It is true that the bone oscillator will vibrate for a speech stimulus, as well as low-frequency tonal stimuli, as the maximum output of the bone oscillator is approached. However, an important distinction must be made. A child will not be able to select the appropriate picture or item on the basis of a tactile sensation alone. If the child can complete the SRT, then a true hearing threshold by bone conduction has been obtained, and concerns regarding simply a vibratory response can be eliminated.

The value of the bone-conducted SRT becomes even greater with the introduction of masking. Many youngsters become confused when masking is introduced during puretone testing. With the bone-conducted SRT, it is relatively easy to introduce masking into the nontest ear without interruption of the SRT procedure. Confirmation of a bilateral conductive component to a hearing loss is possible for many children who will not cooperate for masked puretone testing. Similarly, a unilateral sensory/neural or conductive hearing loss can be confirmed.

Speech Recognition

The measurement of speech recognition with the pediatric population must consider the selection of test materials within a child's receptive vocabulary competency. Haskins (1949) developed phonetically balanced (PB) lists composed of monosyllabic words selected from the spoken vocabulary of kindergartners (PB-K). Clinicians must exercise caution in administering this test unless there is a relatively good assurance that the receptive vocabulary age of the child approaches at least that of a normal-hearing kindergartner (i.e., 6 years of age or older) (see more detailed information below).

To bypass this problem, Ross and Lerman (1970) developed the Word Intelligibility by Picture Identification (WIPI) test. The WIPI test includes picture plates with six illustrations per plate. Four of the illustrations have words that rhyme and the other two illustrations are presented as foils to decrease the probability of a correct guess. The use of WIPI materials is appropriate for those children with receptive vocabulary ages of 4 years and greater.

There are differences between the PB-K words and WIPI test approach to speech perception testing besides the evident fact that the latter is pictorially represented. PB-K words represent an open response paradigm in which the child is forced to give a response from an unlimited set of possibilities, whereas the WIPI is a closed response set with the child's response being a forced choice. As such, the use of the WIPI as a closed-set test improves the discrimination scores by about 10%.

The Northwestern University-Children's Perception of Speech (NU-CHIPS) test by Elliott and Katz (1980) was developed as a speech perception test appropriate for younger children. Test materials are limited to monosyllabic words that are documented to be in the recognition vocabulary of children with normal hearing as young as age 3 years. Additionally, the authors report that children with hearing loss and a receptive language age of at least 2.6 years demonstrate familiarity with the words and pictures on the test.

Historically, several criteria were considered essential in selecting test items for measuring children's speech recognition including word familiarity, homogeneity of audibility, and phonetic balancing (i.e., to have phonemes within a word list represented in the same proportion as in English). When test item construction is constrained by phonetic balancing, the resulting word lists may contain words that are unfamiliar to children with hearing loss. A lexical approach to test construction, sensitive to the frequency of occurrence of words in the language and to the lexical similarity of target words, may result in measuring spoken word recognition with greater accuracy in children with hearing loss.

The Lexical Neighborhood tests (LNTs) (Kirk et al. 1995) assess word recognition and lexical discrimination in children with hearing loss. A primary goal in the development of these perceptual tests was to select words that were likely to be within the vocabulary of children with profound hearing losses. These tests approach speech perception from the perspective that word recognition performance is influenced by the lexical properties of the stimulus words.

Kirk et al. (1995) examined the effect of lexical characteristics on a group of pediatric cochlear implant users' spoken word recognition and compared their performance on the LNT and Multisyllabic Lexical Neighborhood Test (MLNT) with scores on the traditional, PB-K. Word recognition was significantly higher on the lexically controlled lists than on the PB-K. In fact, only 30% of the words on the PB-K were contained within the childhood language database from where the words for the LNT and MLNT were derived. It may be that the restrictions imposed by creating a PB word list result in the selection of test items that are unfamiliar to children with hearing loss.

SUMMARY

The standard of care in the United States for EHDI is founded on hearing screening by 1 month of age, audiologic diagnosis by 3 months of age, and intervention by 6 months of age. The accuracy and precision of our audiologic test battery is critical in achieving valid diagnostic outcomes in a timely manner. Important and fundamental decisions in management and intervention depend on the audiometric outcomes and diagnosis provided by audiologists. Clearly, information must be accurate, precise, timely, and cost-effective to provide optimal service and help families move forward with intervention. When achieved, the goal of optimizing a child's communication behavior and global development is positively influenced, and a family's empowerment is significantly enhanced.

FOOD FOR THOUGHT

1. What kind of information should be collected for a child's case history?
2. What is an age-appropriate, cost-effective, and efficient diagnostic protocol for a 12-month-old normal developing and cooperative child referred for suspected hearing loss?
3. What is the impact of receptive vocabulary on the measurement of speech perception with the pediatric population?

REFERENCES

American Speech-Language-Hearing Association. (2004) Guidelines for the audiologic assessment of children from birth to 5 years of age. Available online at: http://www.asha.org/members/deskref-journals/deskref/default

Bayley N. (1969) *Bayley Scales of Infant Development: Birth to Two Years.* San Antonio, TX: Psychological Corp.

Condon MC. (1991) Unique challenges: children with multiple handicaps. In: Feigin J, Stelmachowicz P, eds. *Pediatric Amplification.* Omaha, NE: Boys Town National Research Hospital.

Culpepper B, Thompson G. (1994) Effects of reinforcer duration on the response behavior of preterm 2-year olds in visual reinforcement audiometry. *Ear Hear.* 15, 161–167.

Dent KM, Kenneson A, Palumbos JC, Maxwell S, Eichwald J, White K, et al. (2004) Methodology of a multistate study of congenital hearing loss: preliminary data from Utah newborn screening. *Am J Med Genet.* 125 (1), 28–34.

Dorn PA, Konrad-Martin D, Neely ST, Keefe DH, Cry E, Gorga MP. (2001) Distortion product otoacoustic emission input/output functions in normal-hearing and hearing-impaired human ears. *J Acoust Soc Am.* 110, 3119–3131.

Eilers RE, Widen J, Urbano R, Hudson TM, Gonzales L. (1991) Optimization of automated hearing test algorithms: a comparison of data from simulations and young children. *Ear Hear.* 12, 199–204.

Eilers RE, Wilson WR, Moore JM. (1977) Developmental changes in speech discrimination in infants. *J Speech Hear Res.* 70, 766–780.

Elliott LL, Katz D. (1980) *Development of a New Children's Test of Speech Discrimination (Technical Manual).* St. Louis, MO: Auditec.

Gallaudet Research Institute. (2005) *Regional and National Summary Report of Data from the 2004–2005 Annual Survey of Deaf and Hard of Hearing Children and Youth.* Washington, DC: Author.

Gorga MP, Neely T, Dierking DM, Dorn PA, Hoover BM, Fitzpatrick D. (2003) Distortion product otoacoustic emission tuning curves in normal-hearing and hearing-impaired human ears. *J Acoust Soc Am.* 114, 262–278.

Greenburg D, Wilson WR, Moore JM, Thompson G. (1978) Visual reinforcement audiometry (VRA) with young Down syndrome children. *J Speech Hear Disord.* 43, 448–458.

Haskins H. (1949) A phonetically balanced test of speech discrimination for children. Master's thesis, Northwestern University, Evanston, IL.

Holte L, Prickett JG, Van Dyke DC, Olson RJ, Lubrica P, Knutson CL, et al. (2006) Issues in the evaluation of infants and young children who are suspected of or who are deaf-blind. *Infants Young Child.* 19, 213–227.

Institute of Medicine. (2002) Unequal treatment: what health care providers need to know about racial and ethnic disparities in healthcare. Available online at: http://hospitals.unm.edu/health_literacy/pdfs/unequaltreatmenthcprovider.pdf

Jerger J., Hayes D. (1976) The cross-check principle in pediatric audiometry. *Arch Otolaryngol.* 102, 614–620.

Joint Committee on Infant Hearing. (2007) Year 2007 position statement: principles and guidelines for early hearing detection and intervention programs. *Pediatrics.* 120, 898–921.

Katbamna B, Homnick DN, Marks JH. (1999) Effects of chronic tobramycin treatment on distortion product otoacoustic emissions. *Ear Hear.* 20, 393–402.

Kirk KI, Pisoni DB, Osberger MJ. (1995) Lexical effects on spoken word recognition by pediatric cochlear implant users. *Ear Hear.* 16, 470–481.

Kopp C. (1974) Fine motor abilities of infants. *Dev Med Child Neurol.* 16, 629–636.

Lowery KJ, von Hapsburg D, Plyler EL, Johnstone P. (2009) A comparison of video versus conventional visual reinforcement in 7- to 16-month old infants. *J Speech Lang Hear Res.* 52 (3), 723–731.

Margolis RH, Bass-Ringdahl S, Hanks WD, Holte K, Zapala DA. (2003) Tympanometry in newborn infants–1 kHz norms. *J Am Acad Audiol.* 14, 383–392.

Matkin ND. (1977) Assessment of hearing sensitivity during the preschool years. In: Bess FH, ed. *Childhood Deafness: Causation, Assessment & Management.* New York: Grune & Stratton; pp 127–134.

Meredith R, Stephens D, Sirimanna T, Meyer-Bisch C, Reardon W. (1992) Audiometric detection of carrier of Usher's syndrome type II. *J Audiol Med.* 1, 11–19.

Moeller MP. (2000) Early intervention and language outcomes in children who are deaf and hard of hearing. *Pediatrics.* 106, 1–9.

Moore JM. (1995) Behavioral assessment procedures based on conditioned head-turn responses for auditory detection and discrimination with low-functioning children. *Scand Audiol Suppl.* 41, 36–42.

Moore JM, Thompson G, Folsom R. (1992) Auditory responsiveness of premature infants utilizing visual reinforcement audiometry (VRA). *Ear Hear.* 13, 187–194.

Moore JM, Wilson WR, Thompson G. (1977) Visual reinforcement of head-turn responses in infants under 12 months of age. *J Speech Hear Disord.* 40, 29–34.

Nozza R, Wilson WR. (1984) Masked and unmasked pure tone thresholds of infants and adults: development of auditory frequency selectivity and sensitivity. *J Speech Hear Res.* 27, 613–622.

Picton TW, Dimitrijevic A, Perez-Abalo M, Van Roon P. (2005) Estimating audiometric thresholds using auditory steady-state responses. *J Am Acad Audiol.* 16, 140–156.

Primus M. (1992) Operant response in infants as a function of time interval following signal onset. *J Speech Hear Res.* 35, 1422–1425.

Primus M, Thompson G. (1985) Response strength of young children in operant audiometry. *J Speech Hear Res.* 18, 539–547.

Purdy SC, Williams MJ. (2000) High frequency tympanometry: a valid and reliable immittance test protocol for young infants? *N Z Audiol Soc Bull.* 10, 12–21.

Renshaw JJ, Diefendorf AO. (1998) Adapting the test battery for the child with special needs. In: Bess FH, ed. *Children with Hearing Impairment.* Nashville, TN: Vanderbilt Bill Wilkerson Press; pp 83–103.

Ress BD, Sridhar KS, Balkany TJ, Waxman GM, Stagner BB, Lonsbury-Martin BL. (1999) Effects of cis-platinum chemotherapy on otoacoustic emissions. The development of an objective screening protocol. *Otolaryngol Head Neck Surg.* 121, 693–701.

Ross M, Lerman J. (1970) Picture identification test for hearing-impaired children. *J Speech Hear Res.* 13, 44–53.

Rubin R, Rosenblatt C, Balow B. (1973) Psychological and educational sequelae of prematurity. *Pediatrics.* 52, 352–363.

Schmida MJ, Peterson HJ, Tharpe AM. (2003) Visual reinforcement audiometry using digital video disc and conventional reinforcers. *Am J Audiol.* 12, 35–40.

Skinner BF. (1953) *Science and Human Behavior.* New York: Macmillan.

Stapells DR, Herdman A, Small SA, Dimitrijevic A, Hatton J. (2004) Current status of the auditory steady-state responses for estimating an infant's audiogram. In: Seewald RC, Bamford J, eds. *A Sound Foundation Through Early Amplification.* Basel: Phonak AG; pp 43–59

Starr A, Picton TW, Sininger Y, Hood LJ, Berlin CI. (1996) Auditory neuropathy. *Brain.* 119, 741–753.

Tharpe AM. (2009) Individuals with multiple disabilities. In: Katz J, Medwetsky L, Burkard R Hood L, eds. *Handbook of Clinical Audiology.* 6th ed. Philadelphia, PA: Lippincott, Williams & Wilkins; pp 666–677.

Thompson G, Thompson M, McCall A. (1992) Strategies for increasing response behavior of 1- and 2-year-old children during visual reinforcement audiometry (VRA). *Ear Hear.* 13 (4), 236–240.

Thompson MD, Thompson G, Vethivelu S. (1989) A comparison of audiometric test thresholds for 2-year-old children. *J Speech Hear Disord.* 54, 174–179.

Turner RG. (2003) Double checking the cross-check principle. *J Am Acad Audiol.* 14, 269–277.

Widen JD. (1990) Behavioral screening of high-risk infants using visual reinforcement audiometry. *Semin Hear.* 11, 342–356.

Wilson WR, Folsom RC, Widen JE. (1983) Hearing impairment in Down's syndrome children. In: Mencher G, Gerber S, eds. *The Multiply Handicapped Hearing Impaired Child.* New York: Grune & Stratton.

Yoshinaga-Itano C. (2003) From screening to early identification and intervention: discovering predictors to successful outcomes for children with significant hearing loss. *J Deaf Stud Deaf Educ.* 8, 11–30.

Yoshinaga-Itano C, Sedey A, Coulter DK, Mehl AL. (1998) Language of early and later identified children with hearing loss. *Pediatrics.* 102, 1161–1171.

CHAPTER 25

Genetic Hearing Loss

Carmen Brewer and Kelly King

INTRODUCTION

Although familial hearing loss has long been recognized, at the time of the first edition of the *Handbook of Clinical Audiology* (1972), our knowledge of hereditary hearing loss was limited to phenotypic descriptions and inheritance patterns. In his landmark manuscript, Hereditary Deafness in Man, Konigsmark (1969) described a classification system for hereditary hearing loss based on associations with deficits in other body systems (what we now call *syndromic* hearing loss). In 2003, the Human Genome project successfully sequenced the approximately 23,000 *protein* coding *genes* contained within human *chromosomes,* which provided a reference for normal genetic structure and function, and consequently, improved understanding of damaged genes and genetic mutations. These and other milestones, including identification of the first location for *nonsyndromic* hearing loss along a chromosome (Guilford et al., 1994) and the causative gene (Kelsell et al., 1997), have greatly influenced both research and clinical practice surrounding hereditary hearing loss.

The science of genetics now plays a significant role in our understanding of the auditory system. Genetics, quite simply, plays a part in most biologic aspects of living, and as our understanding of this branch of biology evolves, our application of this information in the diagnosis and management of patients becomes more commonplace. It is rapidly becoming apparent that the clinical audiologist must be knowledgeable about the fundamentals of genetics and hereditary hearing loss, including the common terminology, multitude and array of causative hearing loss genes, variety of associated syndromes, and usefulness of genetic diagnosis in patient counseling and management.

An introduction to genetics can feel like learning a new language, and although fundamental concepts are often accessible, assimilating new terminology may feel daunting. In an effort to facilitate learning, we provide a glossary of common terminology associated with hereditary hearing loss, available at the end of the book. The reader is referred to this glossary for any italicized term they are not familiar with, although most of these will be defined within the body of the text as well.

EPIDEMIOLOGY OF HEREDITARY HEARING LOSS

Based on extensive surveys conducted in schools for the deaf (Morton 1991) and newborn hearing screening statistics (Mehra et al., 2009), it is estimated that 2 to 3 of every 1,000 newborns has significant permanent hearing loss, and 90% of these children are born to hearing parents. Of these, 1:1,000 born in the United States will develop profound hearing loss in early childhood.

Etiologies of congenital or early-onset hearing loss can be environmental or genetic (Figure 25.1), of which at least 50% has a genetic origin. Of those children with a genetic hearing loss, approximately 30% have a recognized syndrome and the remaining 70% have nonsyndromic hearing loss. Approximately 80% of hereditary, nonsyndromic, prelingual hearing loss is inherited in an *autosomal recessive* pattern and 15% to 20% in an *autosomal dominant* pattern (see glossary and discussion below for definitions of *autosomal recessive* and *autosomal dominant*). Additionally, 1% to 2% of hereditary hearing loss is linked to genes on the sex chromosomes (*sex-linked inheritance*). An even smaller percentage is due to mutations in *mitochondrial DNA* (Arnos, 2013).

Epidemiologic figures describing the incidence and prevalence of hereditary hearing loss will no doubt evolve over the coming years as more children are identified through early detection programs and as our ability to identify and understand complex genetic conditions expands.

REVIEW OF BASIC HUMAN GENETICS

In humans, the repository of genetic information is the molecular material deoxyribonucleic acid, or *DNA*. DNA is passed from parent to offspring and contains the instructions necessary for development and survival. It is found in the nucleus and mitochondria of nearly all cells of an organism and is composed of long strands of *nucleotides,* made up of sugar, phosphate, and four chemical bases: *adenine* (A), *thymine* (T), *guanine* (G), and *cytosine* (C). These molecular building blocks are woven into strands that form the now widely recognized double helix shape. The organization of

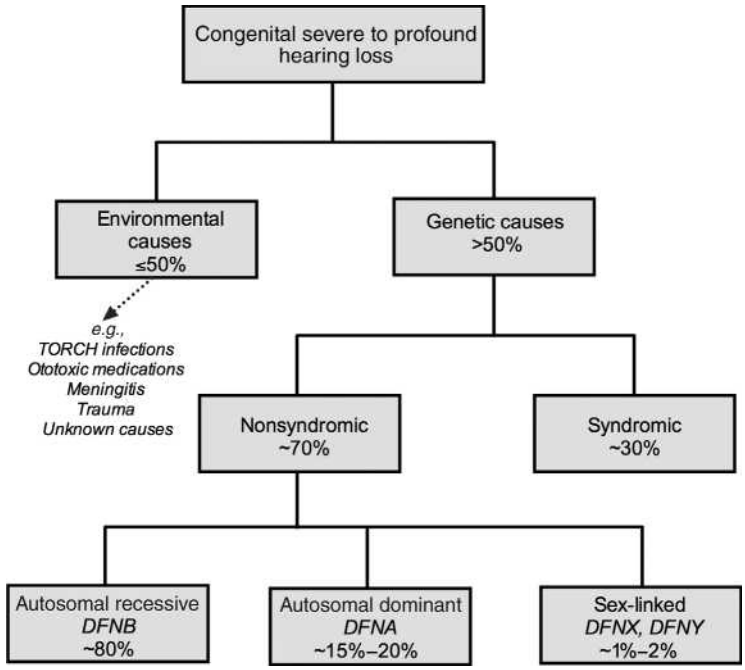


FIGURE 25.1 Causes of congenital severe-to-profound hearing loss in children.

these strands is dictated by a specific pairing of the bases (referred to as *base pairs*); A always pairs to T, and G always pairs to C. For example, a strand of DNA may look like AATGGGCTACTA, and its complementary, paired strand would be TTACCCGATGAT.

The chemical instructions contained within a strand of DNA are determined by the order, or sequence, in which the bases are arranged. A segment of coding DNA that contains enough information for the production of a protein or proteins is called a gene, the basic unit of *heritability.* The DNA contained within the human *genome* holds approximately 23,000 genes, which, collectively, control a great variety of biologic functions necessary for life.

Genes

Specific regions of DNA within a gene can be defined by their function; *exons* contain sequences of DNA that instruct (encode) for the arrangement of *amino acids,* which link and form proteins through a process known as *translation.* The nucleotides in a coding region are arranged into groups of three, forming *codons* each of which command production of a specific amino acid. There are a total of 20 amino acids used to form proteins in humans, and the beginning and end of the chemical translation into a protein is determined by specific coding regions, called *start* and *stop codons.* Noncoding segments of DNA (e.g., *introns*) are interspersed among exons and, although the function of these regions is not entirely understood, they are removed, or spliced, during the process of *transcription.* Additionally, there are regulatory segments of DNA that control aspects of transcription and genetic expression.

Chromosomes

To pack the nearly 3 billion base pairs of the human genome into the nucleus of a cell, DNA is tightly organized into chromosomes. Humans have 46 chromosomes, arranged into 23 *homologous* (corresponding) pairs. One copy of the chromosome pair is inherited from the female parent's egg cells and the other copy is inherited from the male parent's sperm cells. The first 22 pairs of chromosomes, called *autosomes,* are the same in males and females and are numbered (1 to 22) from largest to smallest, based on their relative size. The remaining pair of chromosomes, the sex chromosomes, determines a person's gender. In females, there are two X chromosomes, whereas males have one X and one Y. As DNA replicates in preparation for cell division, chromosomes play a critical role in ensuring that molecular information is copied accurately and carried into the new cell. Chromosomes can be viewed using a light microscope, and collectively, their number, structure, and organization is known as a *karyotype.* Notably, genes associated with either syndromic or nonsyndromic hearing loss have been identified on all 23 chromosomes in humans (Figure 25.2).

Except for the Y chromosome, which has only a few genes, there are thousands of genes contained within each chromosome. Each gene has a specific physical location on a chromosome, called a *locus* (pl. *loci*). To understand the strategy for identifying a gene and its position on a chromosome, one must appreciate how chromosomes are organized. Each chromosome has a short and a long extension, or arm (identified as *p* and *q arms,* respectively). The p and q arms are connected by a *centromere.* The ends of chromosomes are called *telomeres.* In *cytogenetics,* the branch of biology concerned primarily with studying chromosomes,

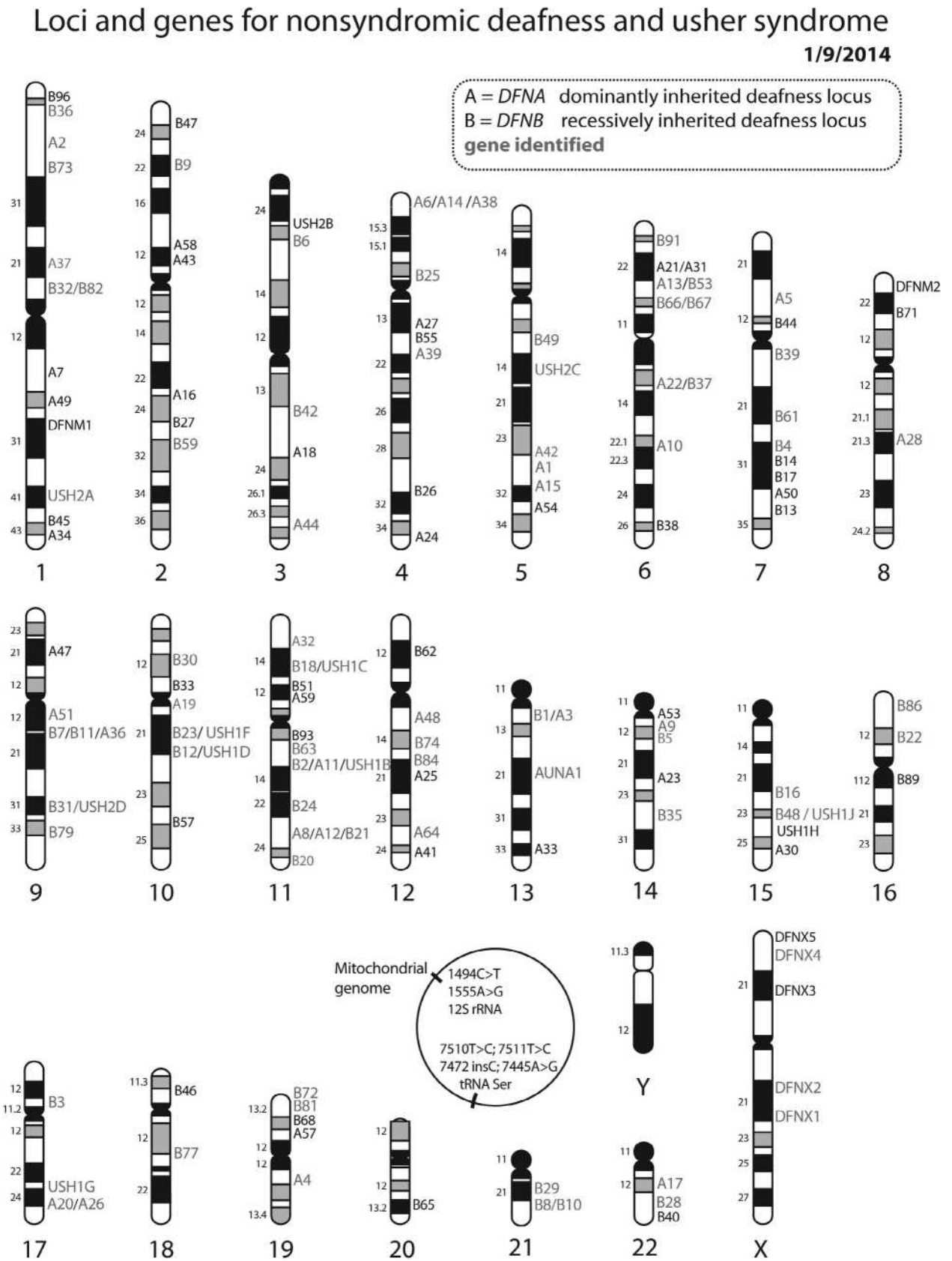


FIGURE 25.2 Nonsyndromic deafness loci and genes shown by chromosome (autosomes and sex chromosomes) as well as the mitochondrial genome, updated from Friedman and Griffith (2003).

staining is used to examine the chromosome, which results in light and dark horizontal segments, or bands. Each chromosome has a unique pattern of banding and each band is numbered. Therefore, regions along a chromosome can be mapped, or identified, by providing the chromosome number, the arm of the chromosome, the region along the arm, and the corresponding band and subband, if applicable. For example, the locus for the gene *GJB2* is 13q12.11. This gene is located on chromosome 13, on the long arm (q) at region 1, band 2, subband 11 (pronounced as "thirteen q one two point one one").

Genotype–Phenotype

In humans, the percentage of DNA that is the same from person to person is 99.5%, and yet as a species we contain a large degree of variation in how we look and how our bodies function. An individual's *genotype* is their specific genetic composition, that is, the combination of *alleles,* or variations in genes, that make each person unique. The manifestation of these genes into observable traits (e.g., eye color) is called a *phenotype.* Many genes have a variety of normal alleles associated with them, resulting from *polymorphisms,* or variations in the DNA sequence that do not have an adverse effect. The sum of this normal genetic variation in each person specifies our anatomy and physiology (e.g., height, metabolism). When the alleles of a gene on homologous chromosomes are the same, the genotype is described as being *homozygous.* When the alleles of a gene are different on each chromosome, they are described as *heterozygous.* In men, most genes on the X chromosome do not have a counterpart on the Y chromosome, meaning they are *hemizygous* (having only one copy) for those genes.

Genetic Mutations

Genetic *mutations* occur when the nucleotide sequence of a gene is altered in such a way that it changes the protein output, often affecting the structure and function of an organism. Mutations can result from substitutions*, insertions,* or *deletions* of nucleotide bases, which then alter the normal allele and the associated *wildtype,* or naturally occurring phenotype (these are described in Table 25.1). When the substitution of a single nucleotide occurs but the total number of nucleotides in the sequence remains unchanged it is known as a *point mutation.* In some cases, there is no effect on the protein product and the mutation is considered silent. Alternatively, a point mutation may cause the substitution of a different amino acid, in which case it is called a *missense mutation,* or it may change the sequence to instruct for a premature stop codon, known as a *nonsense mutation.* This latter type of substitution often renders the protein nonfunctional.

When nucleotides are inserted or deleted into a sequence in multiples of three (recall that proteins are coded by groupings of three base pairs) an amino acid(s) may be added or missing, or it may produce an abnormal stop codon. Insertions or deletions in multiples of three only affect the involved codon and subsequent codons will be unaffected. If the insertion or deletion of nucleotides occurs by some multiple other than three, all subsequent codons will be affected, thus shifting the entire remaining sequence, or reading frame. This is known as a *frameshift mutation.* Sometimes, short sequences of DNA are incorrectly repeated within a sequence, known as an *expansion.* See Table 25.1 for an analogy of the common types of mutations that can occur at the molecular level.

TABLE 25.1

Analogy of Mutations at the Molecular Level

Coding Sequence	Mutation Type
The boy ate one hot dog	Wildtype
The boy ate one *n*ot dog	Missense
The boy ate	Nonsense
The boy ate one *big* hot dog	Insertion
The boy ate ____ hot dog	Deletion
The boy *u*at eon eho tdo g	Frameshift–insertion
The boy teo neh otd og	Frameshift–deletion
The boy ate *ate ate* one hot dog	Expansion

Mendelian Inheritance

Mutations that occur at the level of a single gene may follow one of three inheritance patterns: Autosomal dominant, autosomal recessive, or sex linked. These patterns of *Mendelian inheritance* are distinguished from one another by which type of chromosome the mutation occurs on (autosome or sex chromosome) and by how many mutated alleles are necessary for the affected individual to express the trait (one or two). A thorough family history will help identify the mode of inheritance in many cases and even complex histories can be efficiently and effectively captured using a charting tool called the pedigree. A *pedigree* is the visual representation of a family's health history and its use in distinguishing heritable conditions has become common practice across medical disciplines.

Pedigrees are created using a common set of symbols to catalog the occurrence and presentation of phenotypes within a group of related individuals. Each person is depicted by a symbol (e.g., circle indicates female and square indicates male), and their genetic relationship to other individuals is traced through connecting lines and across generations. Filling or shading the symbol identifies affected individuals, and each row within the pedigree represents a different generation within the family. The first affected family member to come to medical attention is known as the *proband* and is depicted on the pedigree by an arrow next to their symbol.

For a complete review of current standard nomenclature, including common symbols, definitions, and abbreviations for human pedigrees, see Bennett et al. (2008).

Autosomal Dominant Inheritance

In autosomal dominant inheritance, an individual only needs one mutated allele to express the trait. In such cases, an affected child typically will have one affected parent from whom they inherited the mutated gene, and they will have a 50% chance of passing on the mutation to their offspring. Conversely, an unaffected child of a parent with the trait will have no risk for passing on the condition. It is important to remember that the risk of inheritance for any condition does not change based on the number of pregnancies and is calculated in the same way for each pregnancy. *Obligate carriers* within a family are members who have one copy of the gene mutation in question based on the pattern of inheritance. In autosomal dominant inheritance, it is relatively easy to identify the obligate carriers because they most often express the phenotype; any member carrying the dominant gene for hearing loss will have hearing loss (assuming 100% *penetrance*—see next paragraph for a discussion of penetrance). In these cases we see a vertical transmission pattern where every generation has at least one affected individual, and males are equally as likely to express the trait as females. See Figure 25.3 for an example pedigree of a family displaying an autosomal dominant inheritance pattern.

An exception to the common vertical transmission pattern associated with autosomal dominant inheritance occurs in instances when there is a *de novo mutation,* or the first occurrence in the transmission line. In such cases affected individuals will not have an affected parent; however, the risk for recurrence in their offspring remains the same (50%) as someone with an inherited autosomal dominant mutation. Similarly, there are examples of autosomal dominant gene mutations where not every person who has the mutation expresses the trait. This is known as the penetrance of a gene, or the percentage of individuals carrying a dominant mutation who actually express the trait. When a dominant mutation has incomplete penetrance, not every obligate carrier will have an associated phenotype. In a similar fashion, an autosomal dominant gene may be completely penetrant, but vary in how the phenotype is expressed among individuals (e.g., varying degrees of hearing loss). This is known as *variable expressivity.* The penetrance and expressivity of a heritable disorder can be described for all patterns of Mendelian inheritance and do not apply just to autosomal dominant traits.

Autosomal Recessive Inheritance

When the inheritance pattern is identified as autosomal recessive it means an individual must have two copies of a mutated gene to express the associated phenotype. Classically, this signifies an affected child with two heterozygous unaffected parents who each have one copy of the mutated

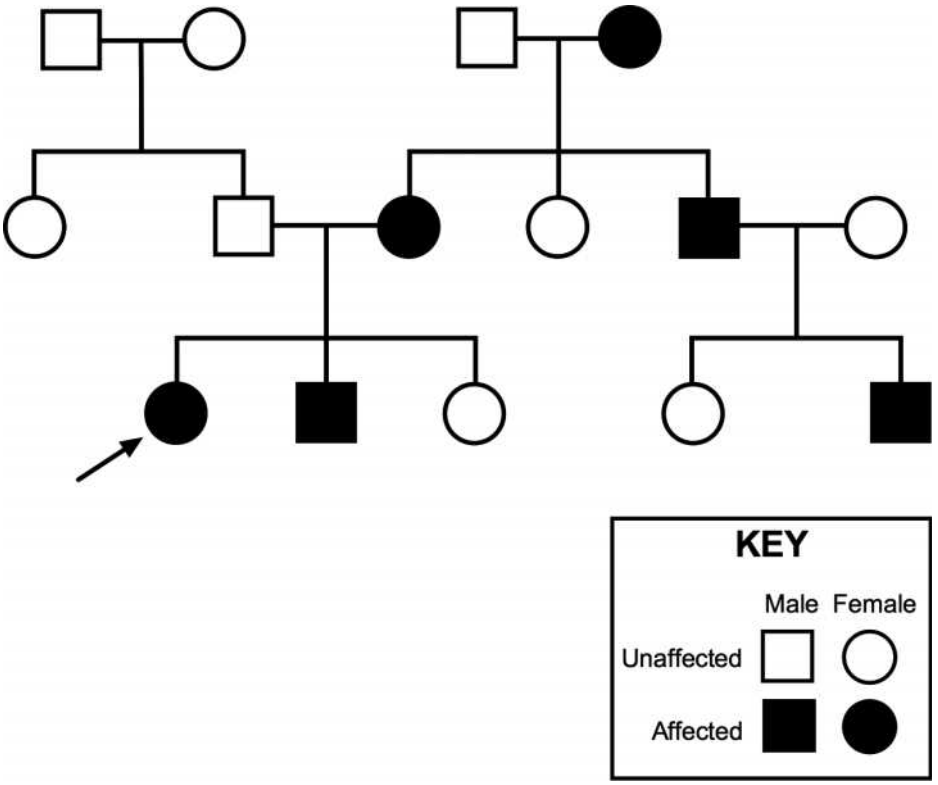


FIGURE 25.3 Three-generation pedigree of a family segregating an autosomal dominant trait. The *arrow* indicates the proband. Note the vertical transmission pattern across generations and father-to-son transmission.

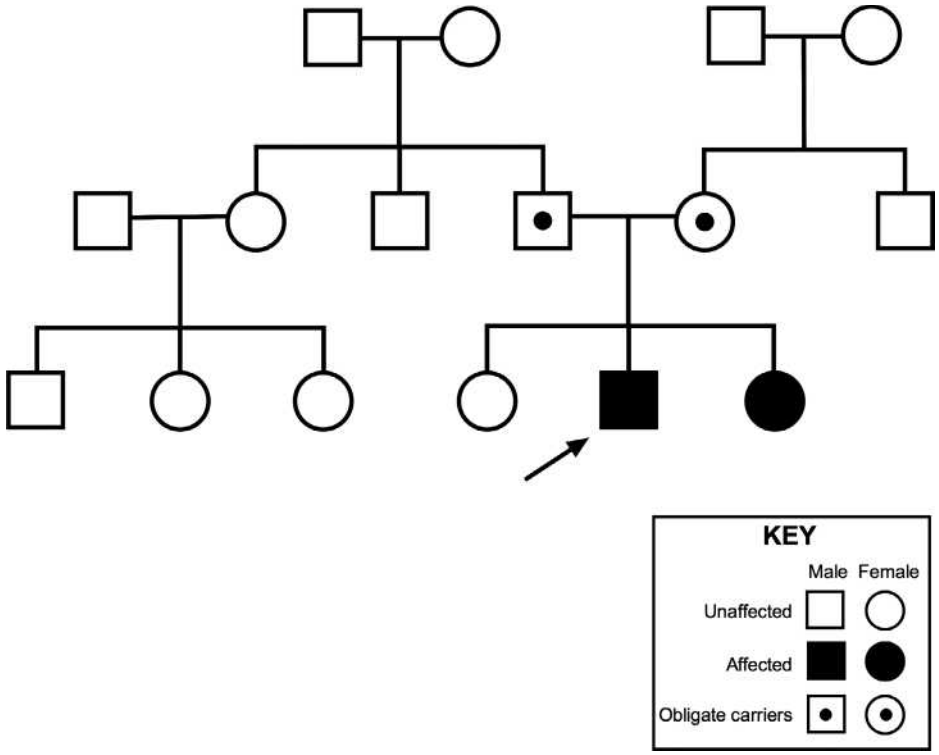


FIGURE 25.4 Three-generation pedigree of a family segregating an autosomal recessive trait. The *arrow* indicates the proband. Note the horizontal transmission pattern and obligate carriers.

allele. There is a 25% chance that the offspring of two heterozygous carriers will acquire a double dose of the mutated gene, one from each parent and, thus, express the trait. There is a 50% chance that each offspring will be a heterozygous carrier, and a 25% chance that the child will inherit no mutated allele. As in autosomal dominant transmission, males and females are equally likely to express the trait, but in the case of autosomal recessive inheritance there can be generations of unaffected individuals and, within a single generation, either a sole affected individual or multiple family members who express the trait. See Figure 25.4 for an example pedigree of a family displaying an autosomal recessive inheritance pattern. The probability of expression for recessive genes increases when parents are related individuals, which is known as *consanguinity.*

Sex-Linked Inheritance

When a genetic mutation occurs on either the X or Y chromosome, the inheritance is considered sex linked. Females pass on one of their X chromosomes to their offspring and males pass on either an X or a Y, which determines the sex of the child. Sex-linked conditions are more likely to involve the X chromosome because it contains many more genes than the Y chromosome. These conditions can be inherited in either a dominant or recessive manner; however, because males are hemizygous for most genes on the X chromosome, the occurrence of X-linked recessive traits is far more common in males than females. In X-linked recessive inheritance, normally there can be no father-to-son transmission of the mutation, but all female offspring of an affected male will be carriers of the mutated allele. Those carrier females will have a 50% chance of having a daughter who will carry the gene and a 50% chance of having a son who will express the trait. See Figure 25.5 for an example pedigree of a family displaying an X-linked recessive inheritance pattern. In cases of X-linked dominant inheritance, males and females are more equally affected. All female offspring of an affected male will express the trait, and an affected female has a 50% chance of having an affected child of either gender. Y-linked disorders are less common conditions that only occur in males as the result of a mutated gene on the Y chromosome. In such cases, affected males will pass on the trait to their male offspring.

Non-Mendelian Inheritance

Sometimes, the inheritance pattern of a disorder does not follow one of the more common Mendelian patterns. These genetically complex conditions are considered rare currently, but in fact are likely common and simply underidentified. We review several examples of non-Mendelian inheritance here, although the reader should note that this is not a comprehensive list.

Mitochondrial Inheritance

Although the cell nucleus contains the majority of DNA in humans, a small amount is also present outside the nucleus in the mitochondria of the cell. Although nuclear DNA is inherited from both parents, because sperm cells lose their

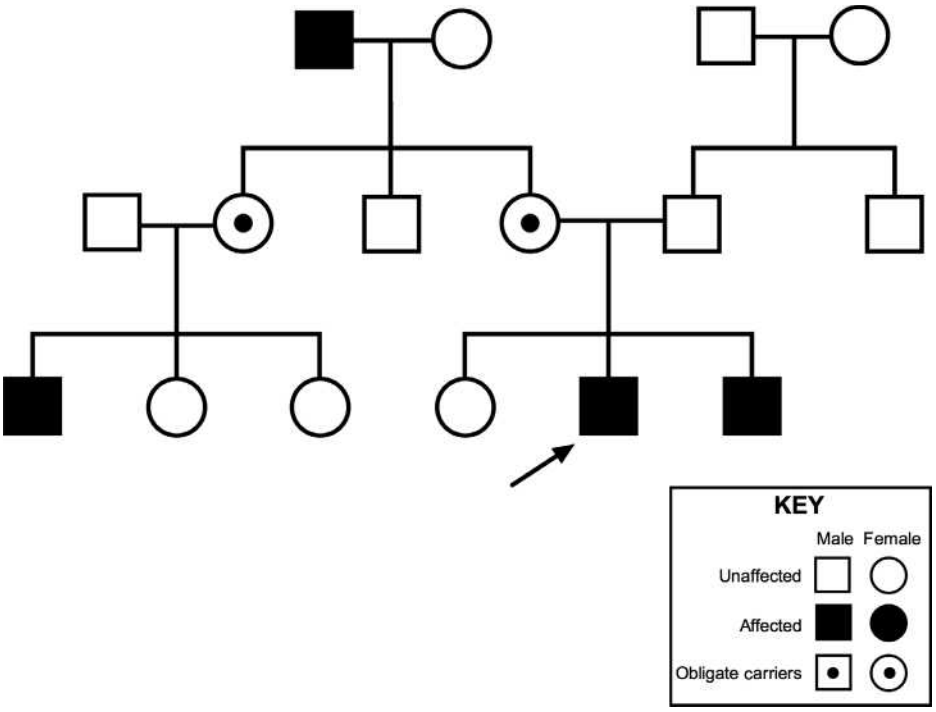


FIGURE 25.5 Three-generation pedigree of a family segregating an X-linked recessive trait. The *arrow* indicates the proband. Note that there is no father-to-son transmission and that all female offspring of affected males are carriers.

mitochondria during fertilization, mitochondrial DNA (mtDNA) is inherited only from the mother (*matrilineal inheritance*) and passed on to all of her offspring. Consequently, when a mutation occurs in mtDNA, males and females are equally affected. All offspring of an affected female will express the trait, and there can be no transmission between affected males and their offspring. Mitochondrial disorders are often characterized by reduced penetrance, variable expressivity, and the observation of multiple, seemingly unrelated phenotypes that result from a single genetic mutation(s), known as *pleiotropy.*

Polygenic Inheritance

Polygenic inheritance refers to the cumulative effect of many genes on a phenotype, in contrast to effects from a single gene or pair of genes, known as *monogenic inheritance.* Most traits in humans are inherited in a polygenic fashion, although our understanding of these complex interactions and ability to identify them are just evolving. In the case of *digenic inheritance,* an individual is heterozygous for mutations in two different genes at different loci. In such instances, neither mutation alone would result in an altered phenotype, but the combined effect from the interaction of the independent mutations is deleterious.

Modifier Genes

Another complex aspect of heritability involves the influence of genetic background on phenotypic expression. There is growing circumstantial and direct evidence for the existence of *modifier genes,* which are independent genes that alter, or modify, the expression of traits, such as the onset, progression, and severity of disease. Just as modifiers of the English language can enhance or change the quality or meaning of a sentence, *genetic modifiers* can enhance or inhibit the expression of other autonomous genes. Indeed, much of the phenotypic variability (e.g., expressivity, penetrance, and pleiotropy) observed in single gene (monogenic) disorders may be explained by variations in genetic background. For example, the phenotypic spectrum associated with mutations in the cadherin 23 gene (*CDH23*) ranges from age-related hearing loss, to nonsyndromic prelingual hearing loss, to the occurrence of Usher syndrome (McHugh and Friedman, 2006). The most common effect of modifier genes is an increased risk for disease (e.g., hearing loss) by the interaction of two or more alleles at different loci; in this case, the risk for disease is higher than the risk associated with either allele individually. There is also evidence that some modifier genes exert their influence in a protective fashion by decreasing susceptibility for disease (Nadeau, 2003; Riazuddin et al., 2002). Going forward, the ability to quantify the influence of genetic background on normal and aberrant structure and function will refine our understanding of heritability and susceptibility, clarify fundamental properties of auditory function, and guide future therapeutic designs.

Multifactorial Inheritance

Many clinicians have observed variations in the phenotypic expression of hearing loss between individuals with similar environmental exposures (e.g., noise, pharmacologic intervention). What explains significant ototoxicity documented in one patient given an equivalent dose of the same

cisplatin-based chemotherapy as another patient who exhibits no change at all? Certainly, comorbid factors such as preexisting hearing loss, age, and renal function, among others, are associated. But these cases also support an underlying genetic influence. Ongoing research in animal models and humans is aimed at delineating the complicated relationship between genes and our environment, known as *multifactorial inheritance.* In multifactorial inheritance individuals have genetic susceptibility for certain diseases or disorders, but do not express the phenotype until they are exposed to a particular environmental trigger. A well-described example of this is a mitochondrial mutation involving an adenine (A) to guanine (G) single nucleotide transition at position 1555 on the 12S *ribosomal RNA* gene (A1555G). Individuals with this mutation have an increased risk for ototoxicity from aminoglycoside exposure. Studies in animal models, mainly mice, have identified several genes associated with increased susceptibility to noise-induced hearing loss, including but not limited to *Ahl1,* which is a gene that is also associated with age-related decline in hearing (e.g., Davis et al., 2001), and several candidate genes in humans (*GRHL2, KCNQ4, KCNE1, CAT, PCDH15, MYH14, HSP70*) have shown promising evidence for a multifactorial interaction with noise (Sliwinska-Kowalska and Pawelczyk, 2013).

Chromosomal Abnormalities

Abnormalities that affect the number or structure of chromosomes result in a loss, gain, or altered location of segments of genetic material. Consequently, multiple body systems may be affected. These are rarely inherited conditions and most often stem from an error in *meiosis* or *mitosis,* processes that take place during cell division. The incidence of chromosomal abnormalities is approximately 1:150 live births, although they account for a significant number of spontaneous abortions, or miscarriages (Carey, 2003).

The normal number of 46, XX or 46, XY chromosomes in females and males, respectively, is known as *euploidy.* When there is an extra copy of a single chromosome (*trisomy*) or when one copy is lost (*monosomy*), it is known as *aneuploidy.* Generally, the gain of genetic material is tolerated more than the loss of a chromosome, and monosomy of any of the autosomal chromosomes is lethal. The most common viable trisomy syndrome is trisomy 21, which causes Down syndrome (e.g., 47, XY +21 for a male with Down syndrome). This extra copy of all or a part of the 21st chromosome accounts for nearly one-third of all infants born with a chromosomal abnormality. Down syndrome is characterized by craniofacial anomalies, varying degrees of intellectual disability, delayed growth, and hearing loss. An abnormality in one of the sex chromosomes occurs in approximately 1:300 live births, and the most common sex chromosome disorder in females is monosomy 45, X, which causes Turner syndrome (described later in this chapter). Excluding trisomy 21 and disorders affecting the sex chromosomes, the incidence of aberrations in number or structure of all remaining chromosomes occurs in less than 1:1,000 births (Carey, 2003). Occasionally, the entire set of chromosomes is abnormally copied, known as *polyploidy.* An example karyotype for a female with three paired sets of each chromosome (triploidy) would be 69, XXX. Although common in some species, polyploidy is lethal in humans.

It is rare that the loss or gain of an entire chromosome results in a viable fetus. More often, *duplications* or deletions of segments of the chromosome are observed. The term duplication indicates contiguous DNA has been erroneously copied onto the same chromosome resulting in extra genetic material. Similarly, a deletion indicates a region of the chromosome is missing, which often involves the loss of multiple genes. In some cases a portion of a chromosome will break and reverse its order within the same chromosome, known as an *inversion.* Other times, a portion of one chromosome will break and attach to another nonhomologous chromosome, which is known as a *translocation.* Errors in cell division that occur after fertilization has taken place result in an individual with two or more cell lines that contain different genetic information. This is known as *mosaicism.* In many cases, the severity of the phenotype is correlated with the number of abnormal cells present.

Nomenclature

Genes associated with hearing loss have been localized to every autosome and both sex chromosomes. The Human Genome Organization (HUGO) Gene Nomenclature Committee oversees the standardized naming process of genes in humans to ensure that nomenclature is unambiguous and uniform. Gene names are meant to convey the specific character or function of the gene. The standard gene symbol (typically, an abbreviated version of the gene name) for humans is italicized and written in capitalized letters (e.g., *GJB6* is the symbol for the gene "gap junction protein, beta 6, 30 kDa"). The standard nomenclature for loci associated with nonsyndromic hearing loss is DFN (for deafness) followed by a letter that denotes the mode of inheritance: A (autosomal dominant), B (autosomal recessive), X (X-linked), or Y (Y-linked). The number that follows identifies the order in which a locus designation was requested from the nomenclature committee and may reflect when it was mapped or discovered. For example, DFNA1 is the first nonsyndromic autosomal dominant locus for hearing loss that was identified, where DFNX4 is the fourth locus along the X chromosome associated with nonsyndromic hearing loss. Loci for modifier genes for hearing loss are classified as DFNM (Riazuddin et al., 2000).

ROLES OF GENES INVOLVED IN HEREDITARY HEARING LOSS

Identification of genes causing hearing loss has facilitated understanding many different proteins and *ribonucleic acids*

TABLE 25.2

Examples of the Roles of Genes Involved in Hereditary Hearing Loss

Role	Gene	Cytogenetic Locus	Hereditary Hearing Loss
Gene regulation			
Transcription factor	*EYA4*	6q23	DFNA10
Fluid homeostasis			
Gap junctions	*GJB2*	13q11–q12	DFNB1, DFNA3A, KID (keratosis, ichthyosis, and deafness) syndrome, Vohwinkel syndrome
Ion-channels	*KCNQ4*	1q34	DFNA2A
	KCNQ1		Jervell and Lange-Nielsen Syndrome
Transporters	*SLC26A4*	7q31	DFNB4, Pendred syndrome, EVA (enlarged vestibular aqueduct)
Junctional complex and tight junctions			
Tight barriers	*CLDN14*	21q22.13	DFNB29
Structural integrity			
Hair bundle organization	*MYO7A*	11q13.5	DFNA11, DFNB2, Usher syndrome type 1B
Structural proteins	*TECTA*	11q23.3	DFNA8/12, DFNB21
Synaptic transmission			
Synaptic vesicle exocytosis	*OTOF*	2p23.3	DFNB9

(*RNAs*) that are necessary for hearing and has also unveiled signaling pathways and protein complexes in the inner ear. Hearing loss genes can be classified by their role in development and function of the ear. These roles include gene regulation, fluid homeostasis, mechanotransduction, and structure (Table 25.2). Regulatory genes primarily function to regulate or transcribe other genes and are important in the development and maturation of the ear. Genes encoding proteins critical for transportation of ions across membranes and fluid homeostasis include those involved in gap junctions, ion-channels, and transporters. Genes contributing to the structural integrity of the inner ear include cytoskeletal proteins, such as the myosins that have an important role in organization of stereocilia and tip-links, and structural proteins that form and organize the tectorial membrane. Additional genes encode proteins important for synaptic transmission between sensory cells and their dendritic connections (Dror and Avraham, 2010; Jones and Jones, 2013).

HEARING LOSS PHENOTYPE

Knowledge of auditory phenotypes observed in hereditary hearing loss is a valuable component of the patient's diagnostic assessment. Familiarity with the presentation and natural history of the hearing loss is essential for etiology-specific counseling, anticipating future hearing-related needs, and establishing a baseline for any current or future interventions. In some cases, the audiologic information alone may help to guide genetic diagnosis.

What we know or understand about the phenotype of some hereditary hearing losses is well defined; however, the auditory and especially the vestibular phenotypes of many conditions remain incomplete. This is due, in part, to the fact that some conditions are rare and difficult to study. However, it also reflects conclusions derived from limited, at times anecdotal clinical assessments, as well as the current limitations of our diagnostic measures to demonstrate the complex nature of the auditory system.

Use of the term "deafness" by the nonaudiology medical community to describe hereditary hearing loss is vague and often misleading. Observations of self-reported hearing loss or dizziness and cursory screenings (e.g., watch tick or finger-rubbing "tests") are still found within the method sections of some current papers. The concern raised is that not only do these casual findings fail to adequately characterize auditory function, but they may misrepresent the true course of a disease and delay efforts to advance therapy or identify at-risk populations. There is clearly a role for audiology among these clinical research teams.

A European working group has proposed recommendations for the content and description of audiologic data for nonsyndromic hereditary hearing loss (Mazzoli et al., 2003). This includes specification of hearing loss degree, type, configuration, involved frequencies, laterality, symmetry, estimated age of onset, progression, presence/absence of tinnitus, and assessment of vestibular symptoms and/or function. Within this framework, it is important that the audiologic assessment include testing to specify the type of hearing loss as fully as possible, including (a) differentiation of sensory from neural with tests, such as otoacoustic emissions (OAEs), the auditory brainstem response (ABR), and acoustic reflex measures, and (b) description of middle ear function in addition to 226-Hz

tympanometry using tests such as wideband reflectance and multifrequency tympanometry when there is a conductive component or other evidence of middle ear dysfunction. It is imperative for the audiologist to conduct a comprehensive, consistent, and informative test battery.

NONSYNDROMIC HEARING LOSS

Autosomal Dominant Nonsyndromic Hearing Loss

Nonsyndromic hearing loss inherited as a dominant (DFNA) trait and mapping to one of the 22 autosomal chromosomes (Figure 25.2) is genetically *heterogeneous* with at least 64 loci and more than 25 known genes (Van Camp and Smith, 2013). All autosomal dominant nonsyndromic hearing losses are sensory/neural, with the exception of DFNA23. This gene was reported for a single family, half of whom had a conductive component to their hearing loss, although there was insufficient data to rule out active middle ear disease at the time of the audiometric evaluation (Smith, 2013).

The severity of autosomal dominant nonsyndromic hearing loss is variable, ranging from mild to profound, and in general, it is less severe than that of autosomal recessive nonsyndromic hearing loss. The hearing loss most commonly begins in the high frequencies and progresses to include the mid and eventually the low frequencies. However, there are a variety of configurations which include hearing loss that begins in the low frequencies (e.g., DFNA1 and DFNA6/14/23) or mid frequencies with a "cookie-bite" configuration (e.g., DFNA10). In some cases, the hearing loss can be limited to the high frequencies (e.g., DFNA2).

Onset of the hearing loss is typically postlingual and progressive, beginning in the first or second decade of life; however, there are several loci associated with congenital or prelingual onset, stable, or slowly progressive hearing loss (e.g., DFNA6/14/38). A number of loci are associated with progressive hearing loss that begins during the third decade of life or later (e.g., DFNA9). Fluctuating hearing loss has been observed with four loci (e.g., DFNA9). Hearing loss in persons with DFNA16 has a sudden onset and fluctuations that respond to treatment with steroids (Fukushima et al., 1999).

Vestibular manifestations, ranging from subjective reports to positive findings on tests of vestibular function, have been reported for more than 10 autosomal dominant nonsyndromic loci, although this area of the phenotype in most cases has not been thoroughly explored.

Because of the delayed onset, most people with autosomal dominant nonsyndromic hearing loss will not be identified by newborn hearing screenings, and in many cases not even by early school screenings for hearing loss. In some cases it is difficult to differentiate a late onset autosomal dominant nonsyndromic hearing loss from one caused by environmental factors and aging (e.g., DFNA2B).

DFNA2 (Genes: *KCNQ4* and *GJB3;* Cytogenetic Locus: 1q34)

There are two genes, *KCNQ4* and *GJB3,* at the *DFNA2* locus, which are labeled as *DFNA2A* and *DFNA2B,* respectively. Both encode proteins that form channels important for fluid homeostasis. Mutations in *KCNQ4* are one of the more common causes of autosomal dominant nonsyndromic hearing loss.

Hearing loss at the *DFNA2A* locus is typically progressive with postlingual onset in the high frequencies during the first or second decade of life, which may progress to the mid and low frequencies with a sloping configuration. There is phenotypic variability between affected families; some have hearing loss confined to the high-frequency regions, and others have hearing loss spanning the frequency range. Word recognition ability is typically proportionate to the degree of hearing loss. Many experience tinnitus. Although vestibular function is generally normal, there has been at least one report of vestibular hyperactivity on rotary chair testing. Hearing loss associated with *DFNA2B* has a later onset, around the fourth or fifth decade of life, and manifests as a progressive, sloping high-frequency sensory/neural hearing loss (De Leenheer et al., 2002; Smith, 2013).

DFNA6/14/38 (Gene: *WFS1;* Cytogenetic Locus: 4p16.1)

DFNA6, DFNA14, and *DFNA38* are considered mutations in the same gene, *WFS1.* This gene encodes the protein wolframin, which is expressed in many cells in the body including hair cells and other inner ear structures. Its exact function in the ear is unknown, but it is thought to have a role in ion homeostasis within the cochlea.

The hearing phenotype associated with mutations in *WFS1* at the *DFNA6/14/38* locus is one of very few hereditary hearing losses with a low-frequency configuration that progresses slowly. Age of onset is in the first and second decades of life. The hearing loss is typically symmetrical and initially involves 250 and 500 Hz before 10 years of age, making it unlikely that it will be identified on newborn or early school hearing screenings. It gradually progresses to include 1,000 to 2,000 Hz in a low-to-high–frequency progression with puretone thresholds, on average, exceeding 50 dB HL by age 50 years. In the fifth and sixth decades, the audiometric configuration flattens as a concomitant high-frequency hearing loss develops, sometimes with preservation of mid-frequency hearing. Other audiologic characteristics include preserved word recognition ability and absent distortion product OAEs (DPOAEs) commensurate with puretone thresholds. There is frequently nonbothersome tinnitus. Although there are no reports of subjective vestibular complaints, several cases of vestibular hyperactivity are reported in one cohort (Lesperance et al., 2003; Smith, 2013).

Homozygous mutations in *WFS1* can result in Wolfram syndrome, an autosomal recessive disease with a constellation of clinical manifestations including diabetes insipidus, diabetes mellitus, optic atrophy, hearing loss, and neurologic symptoms. Over half of those with Wolfram syndrome experience sensory/neural hearing loss that is typically greater in the high frequencies but with a wide range of severity and configurations. Onset of hearing loss is most often postlingual and in the first decade of life, but cases of congenital and prelingual hearing loss have been reported. Vestibular dysfunction in Wolfram syndrome is possible, but not common (Karzon and Hullar, 2013).

DFNA9 (Gene: *COCH;* Cytogenetic Locus: 14q12)

The hearing loss gene at the *DFNA9* locus is *COCH,* which encodes the protein cochlin. *COCH* is expressed in the cochlea and the vestibular labyrinth. Its exact role in the ear is unknown, but it is thought to contribute to structural integrity of the cochlea and susceptibility to inner ear infection (Hildebrand et al., 2009; Smith, 2013).

The phenotype associated with mutations in *COCH* includes both auditory and vestibular dysfunction, and some individuals may have symptoms suggestive of Meniere disease, including hearing loss fluctuations and asymmetry with accompanying episodes of vertigo or imbalance (Smith, 2013). Age of hearing loss onset ranges from the second or third decade for some, to as late as the fifth decade for others, depending on the specific mutation. The hearing impairment typically begins as a moderate-to-severe high-frequency (3,000 Hz and above) hearing loss with progression to a severe-to-profound degree across the entire test frequency range (Hildebrand et al., 2009). Word recognition may be disproportionately reduced relative to puretone thresholds (Bischoff et al., 2005).

Vestibular symptoms occur in most persons with DFNA9 and include imbalance, especially in the dark, and episodic vertiginous attacks ranging from paroxysmal to several hours in duration without aural fullness. Results of velocity step testing indicate that vestibular dysfunction starts at a younger age and progresses more rapidly than hearing loss; in some cases vestibular *areflexia* (absence of vestibular function) may be an early finding. Endolymphatic hydrops has been observed on histopathology. Several people with DFNA9-associated auditory dysfunction have been diagnosed with atypical Meniere disease (Smith, 2013), one with autoimmune inner ear disease, and another with superior semicircular canal dehiscence (Bischoff et al., 2005; Hildebrand et al., 2009).

DFNA10 (Gene: *EYA4;* Cytogenetic Locus: 6q23)

Mutations in the *EYA4* gene cause hearing loss at the *DFNA10* locus on chromosome 6q23. *EYA4* is a transcription regulator expressed in the embryonic cochlea that may be involved in inner ear development; its continued role in the cochlea later in life is unknown (Makishima et al., 2007).

Postlingual sensory/neural hearing loss starts during the second to fourth decade of life, often with an initial cookie-bite configuration or with involvement of the middle and high frequencies. There is progression to moderate-to-severe levels across the entire frequency range, with variable expressivity within affected families as the hearing loss progresses. Word recognition scores and acoustic reflex thresholds are typically commensurate with the degree of puretone hearing loss. Vestibular symptoms have been reported for six individuals. Unilateral vestibular hyporeactivity was documented in three of these cases on caloric testing, and benign positional vertigo was observed for one case with a positive Dix Hallpike test (Makishima et al., 2007).

Autosomal Recessive Nonsyndromic Hearing Loss

Autosomal recessive nonsyndromic hearing loss (DFNB) is associated with at least 100 known loci and over 40 known genes (Van Camp and Smith, 2013; Figure 25.2). The audiologic phenotype of most autosomal recessive nonsyndromic hearing losses is congenital or prelingual, severe to profound, stable, and sensory/neural. However, several loci are associated with a delayed onset, and although the puretone configuration typically involves all frequencies, a sloping, progressive high-frequency configuration has been reported as well (e.g., *DFNB8/10*). There may be inter- and intrafamilial variability in the audiologic phenotype (e.g., *DFNB1*). Vestibular dysfunction has been reported for approximately 10 loci (e.g., *DFNB8/10*) and auditory neuropathy has been observed for 2 loci (*DFNB9* and *DFNB14*). Some autosomal recessive hearing loss loci are also associated with autosomal dominant hearing loss (e.g., *DFNB1* and *DFNA6*), and some are associated with syndromic forms of hearing loss (e.g., *DFNB12* and Usher syndrome type 1).

Because most autosomal recessive nonsyndromic hearing loss is congenital, it will most often be detected by newborn hearing screenings.

DFNB1 A (Gene: *GJB2,* Cytogenetic Location: 13q11–12)

The first locus described for nonsyndromic hearing loss, *DFNB1,* contains the gene *GJB2,* which encodes gap junction beta-2 (also referred to as connexin 26 or CX26), a member of the connexin family of proteins. Connexin proteins assemble to form docking stations between adjacent cells known as gap junctions that allow intercellular flow of small molecules.

GJB2 has particular clinical significance because of the high proportion of hearing loss caused by related mutations at the *DFNB1* locus in many different populations. *Biallelic* (referring to both paired alleles) mutations in

GJB2 account for the majority of moderate-to-profound nonsyndromic recessive hearing loss in some populations. There are over 200 known disease-causing mutations in *GJB2,* some of which are very common. These mutations include 35delG in the United States and Europe, 167delT in Ashkenazi Jewish populations (Morell et al., 1998), and 235delC in Japanese Asians (Smith, 2013). Genetic testing for *GJB2* mutations in newly identified prelingual deafness is a first-line standard of care.

The DFNB1 hearing loss phenotype associated with *GJB2* mutations is highly variable, even within a family, and ranges from mild to profound in degree, with a congenital onset in approximately 95%. The hearing loss is sensory/neural, typically symmetric with a flat or sloping configuration, and there are no known vestibular manifestations. There is evidence for a genotype–phenotype correlation. For example, biallelic nonsense mutations (premature stop codon) are associated with more severe and earlier onset hearing loss than nontruncating mutations (Snoeckx et al., 2005). Another molecular variation is associated with a milder phenotype characterized by high-frequency hearing loss with delayed onset (Griffith et al., 2000).

DFNB 8/10 (Gene: *TMPRSS3;* Cytogenetic Location: 21q22.3)

The causal gene at *DFNB8/10, TMPRSS3* codes for the protein transmembrane protease serine 3. The function of *TMPRSS3* in the inner ear is poorly understood, but it likely contributes to normal development and maintenance. The *DFNB8/10* locus is of interest because of the wide variety of phenotypic expressions and a genotype–phenotype correlation. Initial reports were from large, *consanguineous* Pakistani kindred segregating severe-to-profound hearing loss. In this context, *segregation* refers to the separation of phenotypic elements within a population. DFNB8 hearing loss was postlingual with onset during childhood and DFNB10 hearing loss was prelingual. These two loci were later found to be on the same gene. Subsequently, eight Dutch families with postlingual onset of progressive, bilateral sensory/neural hearing loss were described. The hearing loss began as a precipitously sloping high-frequency loss, with ensuing progression to the mid and then the low frequencies. Those with homozygosity for the more severe mutations of *TMPRSS3* were more likely to have severe-to-profound hearing loss, and those with two different mutations of the same gene (*compound heterozygote*), including one allele with a less severe mutation, were more likely to have later onset and sharply sloping hearing loss (Weegerink et al., 2011).

DFNB9 (Gene: *OTOF,* Cytogenetic Location: 2p23.3)

The *DFNB9* locus is associated with mutations in *OTOF* that encodes for the protein otoferlin, which is believed to play an important role in synaptic function. The initial phenotypic description of DFNB9 reported prelingual, severe-to-profound sensory/neural hearing loss and absent ABRs in children in a Lebanese family (Chaïb et al., 1996). Subsequently, mutations in *OTOF* were shown to be the major cause of nonsyndromic recessive auditory neuropathy (Varga et al., 2003). In these latter cases, puretone hearing loss ranged from mild to profound in degree with intact OAEs and abnormal ABRs. Notably, the OAEs were present in young children, but often disappeared with age. It is possible that all persons with *OTOF* mutations have auditory neuropathy, but tests of cochlear function (e.g., OAEs) were not conducted on the early cohorts or at young enough ages.

Another nonsyndromic recessive locus (*DFNB59, PJVK,* 2q31.2) is associated with auditory neuropathy in some kindreds but not in others. The corresponding hearing loss can be prelingual, stable, and severe to profound, or it can be progressive (Mujtaba et al., 2012).

DFNB12 (Gene: *CDH23;* Cytogenetic Location: 10q22.1)

The *DFNB12* locus is associated with mutations in *CDH23,* which codes for an adhesion protein involved in stereociliary bundle cohesion and tip-link formation. Missense mutations in *CDH23* result in DFNB12-related hearing loss, and more severe nonsense mutations result in Usher syndrome (type ID) (Friedman et al., 2011), which is reviewed later in this chapter. Nonsyndromic hearing loss at the *DFNB12* locus is most often congenital or prelingual, but postlingual onset in the first decade has also been reported. The hearing loss can be progressive with the final severity ranging from moderate to profound. Vestibular function is normal (Astuto et al., 2002). The *homolog* of *CDH23* in the mouse, $Cdh23^{ahl}$, is also associated with heritable forms of presbycusis.

X-Linked Nonsyndromic Hearing Loss (*DFNX*)

Five loci are assigned for X-linked hereditary hearing loss (Figure 25.2). There is no unifying pattern of presentation, with the exception that the auditory phenotype is more severe in males than in females. The most common and distinct X-linked hearing loss locus is *DFNX2,* which encodes *POU3F4* (Xq21.1). In males, the hearing loss is congenital and mixed with a conductive component of 30 to 40 dB in the low and mid frequencies and narrowing of the air–bone gap in the high frequencies. The acoustic reflex is frequently present in early stages of the hearing loss, despite air–bone gaps. Over time, there is progression of the sensory component to severe or profound levels. Two anatomic features, dilation of the lateral aspect of the internal auditory canal and enlargement of the vestibule, may contribute to the conductive aspect of the

hearing loss. Attempted stapedectomy has resulted in perilymphatic gushers and subsequent further loss of hearing and vestibular function. This makes it important to consider the possibility of *DFNX2* in males with congenital mixed hearing loss prior to stapedectomy. Female heterozygotes have a similar but milder audiologic phenotype (Cremers et al., 2002; Smith, 2013).

Y-Linked Nonsyndromic Hearing Loss (*DFNY*)

A single locus assigned for Y-linked hearing loss, *DFNY1,* is based on *patrilineal inheritance* of bilateral, symmetrical sensory/neural hearing loss in a nine-generation Chinese family. The degree of hearing loss ranges from mild to severe, and audiometric configurations include sloping, flat, and U-shaped. Age of onset is postlingual and ranges from 5 to 27 years, with a mean of 11.5 years. ABR findings are consistent with a peripheral origin of the hearing loss, and caloric irrigations suggest normal vestibular function in at least a subset of affected individuals. High-resolution CT scans of the temporal bones show no apparent inner ear abnormalities (Wang et al., 2009). Recent evidence suggests that *DFNY1* may be associated with insertion of genetic sequences from chromosome 1 into the Y chromosome rather than mutation of a Y chromosomal gene as the putative cause of hearing loss (Wang et al., 2013).

Deafness Modifier Genes (*DFM*)

The *DFNB26* locus was mapped to chromosome 4q31 in a large, consanguineous Pakistani family. Fifty-three percent of the family members with homozygous *genetic markers* linked to the *DFNB26* region had prelingual, severe-to-profound sensory/neural hearing loss and the other 47% had normal hearing. This led to the discovery of the first deafness modifier locus, *DFNM1,* mapped to a region on chromosome 1q24 (Figure 25.2). All unaffected family members with homozygosity for *DFNB26* had a dominant modifier, *DFNM1,* which suppressed the associated hearing loss (Riazuddin et al., 2000).

Auditory Neuropathy, Autosomal Dominant (*AUNA*)

Currently, there is one known locus for autosomal dominant auditory neuropathy, *AUNA1,* that maps to chromosome 13q21–q24 (Figure 25.2); the causative gene is *DIAPH3* (Schoen et al., 2010). This locus and gene were identified in a four-generation American family of European descent. Age at onset of the auditory symptoms ranged from 7 to 45 years. The puretone hearing loss was symmetrical, worse in the high frequencies, and typically progressed to a profound degree over a 10- to 20-year period. In the younger family members with moderate sensory/neural hearing loss, the phenotype included absent or grossly abnormal ABRs in the presence of intact DPOAEs, typical of auditory neuropathy. As the puretone hearing loss progressed, there was a loss of DPOAEs, indicating a partial sensory site of lesion, and a loss of the ABR, if it was present to begin with. Some affected family members benefited from cochlear implantation (Kim et al., 2004).

Nonsyndromic Mitochondrial Hearing Loss (*MTRNR1*)

Nonsyndromic sensory/neural hearing loss caused by mutations in mitochondrial genes shows a pattern of matrilineal inheritance. There is considerable heterogeneity in both penetrance and phenotype of the hearing loss. The most common nonsyndromic hearing loss results from A1555G mutation in the ribosomal RNA (*MTRNR1*). This mutation can cause nonsyndromic, congenital, severe-to-profound sensory/neural hearing loss. Additionally, in some families and individual patients with this same mutation, hearing loss occurs only after aminoglycoside exposure (Fischel-Ghodsian, 2003).

Otosclerosis (*OTSC*)

Otosclerosis is a common cause of progressive hearing loss with a prevalence of 0.2% to 1% among white adults. Most audiologists are familiar with the clinical presentation of a mixed hearing loss with air–bone gaps that narrow in the mid frequencies, normal tympanograms, and absent acoustic reflexes. Age of clinical onset ranges from the second to the sixth decade of life or later and penetrance averages about 40% with considerable interfamilial variability. Currently eight loci for clinical otosclerosis (*OTSC*) have been identified and more will likely emerge. No causative genes have been sequenced to date. Each of the known *OTSC* loci segregates as an autosomal dominant trait. Because of the variable penetrance and large range in the age of clinical onset, it is likely that there are modifier genes or environmental factors that impact the expression of hearing loss (Schrauwen et al., 2011).

SYNDROMIC HEARING LOSS

Hundreds of syndromes include hearing and vestibular disorders, and the list is growing. Often, issues of comorbidity and multisensory involvement can affect the diagnostic process and re/habilitation strategies. For example, visual reinforcement audiometry with a visually impaired child will be difficult if not impossible. A child with craniofacial abnormalities may have structural anomalies of the outer ear that limit amplification options. The presence of more than one disability has a multiplying effect in hindering communication and learning that is greater than any single occurring disorder, which underscores the importance of early

TABLE 25.3

Online Resources for Hereditary Hearing Loss

Website	Content/Use	Sponsors/Hosts
Online Mendelian Inheritance in Man (OMIM) http://www.ncbi.nlm.nih.gov/omim	Catalog of genetically based human diseases; describes clinical phenotype, causative gene, and function of the causative gene when known; extensive lists of related references. Allows searches by clinical signs and symptoms, disorder name, gene, chromosomes	McKusick-Nathans Institute of Genetic Medicine, Johns Hopkins University School of Medicine
Hereditary Hearing Loss Homepage http://hereditaryhearingloss.org/	Overview of genetics of hereditary hearing loss, designed for both clinicians and researchers. Provides information about nonsyndromic hearing loss, monogenic hearing loss, syndromic hearing loss, and gene expression in the cochlea	Guy Van Camp, University of Antwerp, and Richard Smith, University of Iowa
Genetics Home Reference http://ghr.nlm.nih.gov/	Consumer information including summaries of genetic conditions, genes, gene families, and chromosomes, *Help Me Understand Genetics Handbook,* and a glossary of genetics terminology	National Library of Medicine
Gene Reviews http://www.ncbi.nlm.nih.gov/books/NBK1116/	Expert-authored, peer-reviewed disease descriptions presented in a standardized format and focused on clinically relevant and medically actionable information on the diagnosis, management, and genetic counseling of patients and families with specific inherited conditions, and a glossary of genetics terminology	University of Washington National Center for Biotechnology (NCBT)
GeneTests http://www.genetests.org	Resource for healthcare providers and researchers that includes a directory of laboratories offering genetic testing and genetics clinics providing services to patients and their families with known or suspected inherited disorders	Bio-Reference Laboratories Inc

diagnosis and intervention for these children. Moreover, in some cases the audiologist may be in the unique position to identify possible syndromic forms of hearing loss and make critical referrals for medical confirmation and multidisciplinary management.

Syndromic hearing loss is classically categorized by its association with other affected systems of the body including the external ear, integumentary system (skin, hair, nails), eye, nervous system, skeletal system, renal system, and other abnormalities. Here, we present several syndromes that represent the range of systems most often associated with hearing loss. Examples of Mendelian inheritance are provided, as well mitochondrial inheritance and chromosomal abnormality. The reader is referred to the resources outlined in Table 25.3 for more expansive and up-to-date information on these and the many other syndromes associated with hearing loss. It is worth noting that although eponyms are routinely used in the naming of syndromes, the current standard is for use of the nonpossessive form (e.g., Usher syndrome instead of Usher's syndrome).

Alport Syndrome

Alport syndrome is characterized by progressive renal disease, ocular anomalies, and sensory/neural hearing loss. It occurs in approximately 1:50,000 live births, and 85% of cases are inherited in an X-linked transmission pattern, because of mutations in the *COL4A5* gene (Xq22). The remaining cases have an autosomal recessive inheritance pattern resulting from biallelic mutations in either *COL4A3* (2q36–q37) or *COL4A4* (2q35–q37). All three of these genes contribute to the production of a protein known as type IV collagen. Type IV collagen is a critical component in the network of proteins that make up basement membrane structures, a thin framework that supports and separates cells in many tissues throughout the body. Type IV collagen appears to be especially critical for the basement membrane structures that are found in the cochlea, as well as the kidney and eye.

The renal disease observed in Alport syndrome is characterized by blood and high levels of protein in the urine (hematuria and proteinuria, respectively), and progressive

renal failure that can result in end-stage renal disease. Eye anomalies include a bulging lens, typically in the anterior direction (*anterior lenticonus*) that is so rarely observed outside of Alport syndrome, it is considered, practically, a *pathognomonic* finding. Additional ocular manifestations, including cataract and retinal flecks, can also occur (Kimberling et al., 2011).

The hearing loss associated with Alport syndrome is most often late onset, occurring in older children or adolescents, but congenital hearing loss has also been reported. It is bilateral and sensory/neural in origin and may be more severe in the higher frequencies. Most males (80% to 90%) and some females (28%) with X-linked transmission will have hearing loss, as will most males and females with an autosomal recessive inheritance pattern. The hearing loss can vary in degree and may be progressive in the first or second decade of onset.

Individuals with Alport syndrome and functionally significant hearing loss can usually benefit from hearing aids. Dysfunction is typically localized to the cochlea, although ABR disturbance has been reported. Vestibular function has not been comprehensively evaluated.

Branchio-oto-renal Syndrome (Melnick–Fraser Syndrome; Branchio-oto Syndrome)

Branchio-oto-renal syndrome (BOR) is one of the more common syndromic conditions associated with hearing loss and inherited in an autosomal dominant transmission pattern. Named for the triad of branchial arch remnants, ear and hearing abnormalities, and renal dysfunction, BOR is estimated to occur in 1:40,000 live births. Approximately 40% of cases are due to a mutation in *EYA1* (8q13.3), but BOR may result from mutations in *SIX1* (14q23.1) and *SIX5* (19q13.32) as well. Not all causative genes have been identified. All three of the known causative genes play important roles in embryologic development and regulate the activity of other genes.

The manifestation of branchial remnants results from disrupted development of the second branchial arch, which contributes to the formation of tissues in the front and sides of the neck. This leads to branchial cleft cysts and fistulas associated with BOR. The renal phenotype may include a variety of kidney abnormalities that affect structure and function and, in severe cases, end-stage renal disease may develop, requiring dialysis or kidney transplant. A variation of BOR without renal dysfunction has also been described, known as branchio-oto syndrome, and can be observed in the same family as someone with BOR (Kochhar et al., 2007).

Hearing loss is the most common phenotypic manifestation of BOR, estimated to occur in more than 70% and as much as 93% of affected individuals (Kochhar et al., 2007). Additional ear-related anomalies include preauricular pits, pinna deformities (e.g., cupped auricle), and stenosis of the external auditory canal. A less frequent (<20%) manifestation is preauricular tags. In addition to structural anomalies of the outer ear, middle and inner ear anatomy may be compromised (e.g., ossicular fixation, cochlear hypoplasia). The hearing loss can range from mild to profound and may be conductive, sensory/neural, or, most often, mixed. Notably, hearing loss onset is not always congenital and can range from birth to young adulthood and it may be stable or progressive. Vestibular involvement has neither been confirmed nor ruled out as an associated phenotype.

The expressivity of BOR is highly variable, both within and between families, and the signs and symptoms may differ significantly within the same individual between the right and left sides across affected systems. A comprehensive audiologic evaluation in all patients suspected of BOR is necessary, in combination with otologic management. Re/habilitation strategies will vary depending on the type and degree of hearing loss, but some form of amplification and educational accommodation(s) is necessary for many children with BOR. These patients will likely present initially to an otologist and/or audiologist, and early identification of BOR is important to expedite diagnosis and management of the urologic and renal abnormalities and to establish appropriate surveillance of affected individuals.

CHARGE Syndrome

CHARGE syndrome is a multisystem congenital disorder characterized by the co-occurrence of anomalies represented in the mnemonic: **C***oloboma,* **H**eart defects, **A**tresia of *choanae,* **R**etarded growth and development with or without central nervous system involvement, **G**enital hypoplasia, and **E**ar anomalies with or without hearing loss. It occurs in 1:8,500 to 10,000 births and is inherited in an autosomal dominant fashion, although the majority of individuals with CHARGE syndrome represent simplex cases (single affected member of the family). Dominant mutations in *CHD7* (8q12.2) result in CHARGE syndrome, but *SEMA3E* (7q21.11) is also associated. The *CDH7* gene is believed to play an important role in the organization and packaging of DNA into chromosomes.

Clinical diagnosis of CHARGE syndrome is based on the presence of major and minor diagnostic features. Major clinical features are coloboma, atretic or stenotic choanae, cranial nerve involvement (I, VII, VIII, IX/X), and structural anomalies of the auditory system. Minor diagnostic findings include underdeveloped genitals, developmental delay, cleft palate or lip, tracheoesophageal fistulas, growth deficiency, and structural abnormalities in the cardiovascular system. Many of these features present as life-threatening conditions during the neonatal period (Edwards et al., 2002).

Almost all individuals with CHARGE syndrome will present with pinnae deformity, which is often asymmetrical, with or without hearing loss. Auricles can be short and wide, possibly protruding, with triangular concha and a missing

helical fold that gives a "snipped off" appearance. Lobes may be absent or small. The external auditory canal is usually unaffected, but middle ear ossicles may be malformed. Temporal bone abnormalities occur in most patients and have been suggested for inclusion as a major diagnostic finding. Mondini dysplasia and underdeveloped or absent semicircular canals are common, and vestibular areflexia has been reported.

At least 80% of individuals with CHARGE syndrome have hearing loss. Mixed hearing loss is most common, and the sensory/neural component is believed to be congenital. The conductive contribution is likely because of either malformation of the ossicles or high rates of chronic/recurrent middle ear disease secondary to craniofacial anomalies, or both. The hearing loss may range from mild to profound, but in the majority of cases it will be sufficiently impaired to affect speech and language development, and it may be progressive.

The multisensory involvement and developmental delay associated with CHARGE syndrome make diagnosing hearing loss and habilitation difficult and often necessitates use of physiological and electrophysiological measures of auditory function. These objective measures can reduce the age at which the loss is diagnostically confirmed and expedite early intervention. Children with CHARGE syndrome can benefit from air- or bone-conducted hearing aids, assistive listening devices, or cochlear implants, depending on the type and severity of the hearing loss and their medical candidacy. Serial audiologic monitoring is warranted throughout the lifetime.

Jervell and Lange-Nielsen Syndrome

Jervell and Lange-Nielsen syndrome (JLNS) is a rare (1.6 to 6:1,000,000) autosomal recessive disorder resulting from mutations in either the *KCNE1* (21q22.12) or *KCNQ1* (11p15.5) genes. It is associated with congenital bilateral profound sensory/neural hearing loss and a heart arrhythmia characterized by a long QT interval. When the electrical activity of the heart is measured during an electrocardiogram (EKG or ECG), the distance between two of the waveforms is known as the QT interval. In JLNS, the QT interval is prolonged, which increases the risk for fainting episodes and, in some cases, sudden death. This aspect of the phenotype is treatable with medication, which underscores the importance of early diagnosis.

The auditory phenotype is uniform. These children present with profound bilateral sensory/neural hearing loss and are audiologic candidates for cochlear implantation. Because of the advent of newborn hearing screening, most children with JLNS will be identified during the neonatal period, prior to recognition of cardiovascular symptoms. Although the syndrome is extremely rare, because of the potential for lethal cardiac events, which have been associated with anesthesia and auditory stimulation, cardiac work-up is recommended in any child with profound congenital sensory/neural hearing loss, especially in cases of unconfirmed etiology, and prior to surgical intervention. Cochlear implantation is not contraindicated in these patients, necessarily, but special precautions during surgery and activation may be necessary (Siem et al., 2008). There is limited anecdotal evidence in humans for a vestibular phenotype in JLNS, but this is currently poorly described and requires further study to confirm.

KCNE1 and *KCNQ1* regulate the formation and function of potassium channels that control the flow of potassium ions across cell membranes, which is a critical component to normal function in both the heart and inner ear (Schwartz et al., 2006). Heterozygous carriers of mutations in either gene have normal hearing but may have the long QT phenotype (Romano–Ward syndrome). Because hearing loss is detected before the cardiovascular phenotype, the audiologist may be in the position to identify and refer a child with congenital bilateral hearing loss and a history of possible cardiac events.

Mitochondrial Encephalomyopathy (or Myoclonic Epilepsy) with Ragged Red Fibers

Mitochondrial *encephalomyopathy* with ragged red fibers (MERRF) results from mutations in mtDNA and is an exceptionally rare condition, although the exact prevalence is unknown. The majority of cases are associated with mutations in *MT-TK,* which encodes for the production of *transfer RNAs* that help to build functional proteins, produce energy within cells, and process oxygen. The most common mutation is A8334G.

The initial presenting symptom typically observed in MERRF is an involuntary twitching in a muscle or group of muscles, called myoclonus. Epilepsy, ataxia, and muscle weakness follow and may include additional central nervous system symptoms such as dysarthria, neuropathy, and dementia. The onset of symptoms may range from childhood to adulthood and, as such, young children with MERRF almost always reach early developmental milestones. As the phenotype progresses, clumps of mutated mitochondria collect in muscle tissue throughout the body, which can be stained and viewed through a microscope where they appear as red, ragged fibers. Because of *heteroplasmy,* a common condition in mitochondrial disorders in which there are varying percentages of mitochondria containing mutated DNA within a cell, there is variable expressivity in the MERRF phenotype.

Hearing loss associated with mitochondrial encephalopathy disorders may be cochlear or retrocochlear in origin, or both. Absent OAEs with additional retrocochlear findings (prolonged interpeak latencies) on ABR measures have been reported in patients with MERRF (Tsutsumi et al., 2001). Differentiating MERRF from other progressive mitochondrial encephalopathy disorders can be challenging, and MERRF should be considered as a possible etiology

in any patient with myoclonic epilepsy and sensory/neural hearing loss.

Pendred Syndrome (*DFNB4*)

Pendred syndrome is an autosomal recessive disorder most often resulting from biallelic mutations in *SLC26A4* (7q31). *SLC26A4* codes for production of the protein pendrin, which transports ions across cell membranes in the inner ear, thyroid, and kidneys, as well as other organs in the body. Pendred syndrome is one of the more common syndromic forms of hearing loss, with estimates accounting between 5% and 10% of all early-onset hereditary hearing loss. Pendred syndrome was originally defined as goiter and profound congenital sensory/neural deafness, but it is now known to include variable thyroid and auditory phenotypes.

The most penetrant feature of Pendred syndrome is enlargement of the vestibular aqueduct (EVA), although additional structural anomalies of the bony labyrinth can also occur (e.g., incomplete partition of the cochlea, also known as a "Mondini" cochlea). The hearing loss is often congenital, but can develop during the prelingual and perilingual periods. It is typically bilateral but may not be symmetrical and can be associated with fluctuating or progressive changes in hearing, or both. When hearing loss occurs, progression may be stepwise and head trauma is often reported as a precipitating event. Consequently, patients are encouraged to avoid contact sports or barotrauma (e.g., scuba diving). The configuration of the hearing loss is gradually sloping or flat, but it may also present initially as a high-frequency loss or with sparing of the mid-frequency test region (inverted U). The hearing loss is traditionally described as sensory/neural; however, because EVA is purported to be associated with a third mobile window in the labyrinth, air–bone gaps in the presence of normal tympanometry are often observed (King et al., 2009). In such cases, placement of pressure-equalization tubes to ameliorate suspected conductive hearing loss is not beneficial. Vestibular dysfunction of varying severity is reported in Pendred syndrome, but has not been fully characterized to date.

Diagnosis of Pendred syndrome can be challenging and difficult to differentiate from nonsyndromic forms of EVA, including DFNB4, which is radiologically indistinguishable from Pendred syndrome. The thyroid phenotype has a later onset in childhood or early adulthood and goiter is incompletely penetrant and, thus, not a good diagnostic requirement. Moreover, goiter is common in the population, increasing the potential for possible *phenocopies* (when an environmentally caused trait mimics an inherited one) of Pendred syndrome in patients with severe-to-profound early-onset hearing loss. Even within the same family, there can be large variability in the expression of Pendred syndrome (Madeo et al., 2006).

The audiologist should be aware of the variable auditory phenotype associated with Pendred syndrome beyond what is classically described, including asymmetries in hearing and even unilateral presentations, milder degrees of loss, air–bone gaps with normal tympanometry, and the risk for sudden or stepwise progressive changes in hearing, all of which can impact intervention and re/habilitation strategies. Some children with Pendred syndrome will pass newborn hearing screenings, and most will be good candidates for hearing aids or cochlear implantation.

Stickler Syndrome

Stickler syndrome is an autosomal dominant connective tissue disorder that affects the eye, ear, development of facial structures, and musculoskeletal system. It occurs in 1:7,500 to 9,000 births and is a clinically and genetically heterogeneous condition. Mutations in one of five causative collagen genes have been identified to date. A mutation in *COL2A1* is found in 80% to 90% of cases. This gene contributes to the production of type II collagen, which adds structure and strength to connective tissues and is found in the inner ear. *COL11A1* mutations occur in 10% to 20% of Stickler syndrome cases, and mutations in the remaining three causative genes are rare. All of the genes associated with Stickler syndrome contribute to the normal production of different types of collagen throughout the body.

Stickler syndrome is associated with a flattened facial profile, which is more pronounced during childhood, because of midface hypoplasia involving the maxilla and nasal bridge. Other craniofacial findings can include underdeveloped jaw and cleft palate. Early onset of osteoarthritis, hypermobile joints, and short stature are associated. Ocular anomalies are most commonly observed with mutations in *COL2A1* and *COL11A1* and include early-onset progressive myopia, retinal abnormalities, cataract, and risk for retinal detachment. A nonocular phenotype is related to mutations in *COL11A2.*

Hearing loss can be observed with all forms of Stickler syndrome, regardless of the underlying genotype. When the causative mutation is *COL2A1* the hearing loss is usually mild, confined to the higher frequencies, and progression of the sensory/neural component appears to be no greater than that associated with typical age-related decline. Mutations in *COL11A2* and *COL11A1* are associated with a more severe degree of hearing loss, affecting a broader frequency range, and the loss may be progressive. Conductive hearing loss is common in children with Stickler syndrome in whom chronic/recurrent otitis media occurs frequently. Many patients with Stickler syndrome will present with hypermobile middle ear systems, which may be related to sequela of chronic/recurrent otitis media or because of reduced amounts of type II collagen in the tympanic membrane (Szymko-Bennet et al., 2001). Vestibular dysfunction has not been reported. Serial audiologic monitoring is recommended for patients with Stickler syndrome, with fitting of amplification for functionally significant hearing loss, as necessary. Educational accommodations for these children, who are

at risk for cosensory loss of vision and hearing, should be emphasized.

Treacher Collins Syndrome

Treacher Collins syndrome is an autosomal dominant disorder marked by underdeveloped bony structures and tissues of the face and surrounding areas. It occurs in approximately 1:50,000 live births and the majority of individuals have a single mutation in *TCOF1* (5q32) encoding the protein treacle, which appears to be vital for normal embryologic development of the face. Causative mutations in *POLR1 C* (6q21.1) and *POLR1D* (13q12.2) have also been identified. Notably, over half of patients with Treacher Collins syndrome represent a de novo mutation.

Dominant clinical features of Treacher Collins syndrome include midface hypoplasia because of underdeveloped zygomatic bones, a small chin and jaw (micrognathia), down-slanting eyes, coloboma of the lower eyelid, and abnormalities in the structures of the external ear, including microtia or severe malformation of the pinnae and atresia of the external auditory canal. In addition, the middle ear cavity and ossicular structures may be underdeveloped or missing, and cleft palate with or without cleft lip can also occur. Inner ear anatomy is typically unaffected.

Approximately 50% of patients with Treacher Collins syndrome have congenital conductive hearing loss. It is usually bilateral and can be severe in degree. Sensory/neural hearing loss is not often reported, but because of the severity and complexity of the external and middle ear anomalies, those born with hearing loss are likely to have some degree of permanent auditory dysfunction. Management often involves a combination of surgeries that are medically necessary (e.g., cleft palate repair) and cosmetic (e.g., construction of an artificial pinna, known as an aural episthesis). Surgical reconstruction cannot be carried out safely during the first few years of life and outcomes rarely restore hearing to within normal limits. Consequently, many patients with Treacher Collins syndrome will be lifelong users of some form of amplification. Traditional hearing aids may not be appropriate, depending on the degree to which the ear anatomy is compromised. Bone conduction hearing aids, either removable or implantable, are essential for many of these children to develop normal speech and language (Marsella et al., 2011). There are no cognitive delays associated with Treacher Collins syndrome; however, because of the *dysmorphic* facial features (i.e., structural defects) and poor speech articulation, persons with Treacher Collins syndrome may be inaccurately stereotyped this way.

Turner Syndrome (Ullrich–Turner Syndrome)

Monosomy 45, X, known as Turner syndrome is a chromosomal disorder resulting from the loss of either an X or Y chromosome. It is the most common sex chromosome disorder in females (1:2,500 live births), and the characteristic phenotype includes short stature and premature ovarian failure leading to infertility. The majority of fetuses with Turner syndrome spontaneously abort in the first or second trimester because of developmental abnormalities in the cardiovascular and lymphatic systems. Fetuses that survive are phenotypically female and have additional risks beyond heart disease and lymphedema that include urinary system dysfunction, vision loss, autoimmune conditions, and skeletal abnormalities, among other issues related to health and development (Bondy and Turner Syndrome Study Group, 2007).

Hearing loss is present in approximately 50% of females with Turner syndrome, and it is characterized by transient, recurrent middle ear pathology and progressive sensory/neural loss. Pinna deformities such as low set, posteriorly rotated, cupped, and protruding ears and narrow external auditory canals are common. Heightened monitoring for and aggressive treatment of otitis media is recommended, and whether because of active otitis media or sequelae from recurrent disease, middle ear dysfunction remains an issue for many women with Turner syndrome throughout their lifetime.

Sensory/neural hearing loss is present in approximately one-third of those with Turner syndrome and can be greater in degree in the mid frequencies, especially for those with complete monosomy 45, X karyotypes. Women with Turner syndrome are at risk for progressive changes in sensory hearing, particularly in the high frequencies, at an accelerated rate beyond typical age-related decline. Routine audiologic monitoring is warranted throughout the lifetime regardless of prior documentation of normal hearing. Many can benefit from hearing aids.

About one half of patients with Turner syndrome have a total monosomy 45, X karyotype. Alternatively, mosaicism is common (approximately one-third of cases), and the frequency and severity of sensory/neural hearing loss and auricular anomalies is greater in women with a larger percentage of monosomy 45, X cells. Patients with deletions involving the short arm of the X chromosome have a greater degree of conductive hearing loss than patients with deletions affecting only the long arm (45, XdelXq) (King et al., 2007). Many girls with Turner syndrome are not diagnosed until absence of menstruation in the early teenage years. Because of the high rate of middle ear disease and hearing loss, the otologist or audiologist may be the entry point into the healthcare system and early identification may accelerate important medical intervention(s).

Usher Syndrome

Although many syndromes are associated with both vision and hearing loss, the most common of these is Usher syndrome, which occurs in 4 to 5:100,000 births in the United States. Usher syndrome is an autosomal recessive condition

TABLE 25.4

Classical Hearing, Vision, and Vestibular Phenotypes Reported in the Three Subtypes of Usher Syndrome

	Usher Syndrome Type I	Usher Syndrome Type II	Usher Syndrome Type III
Hearing	Congenital profound bilateral hearing loss	Congenital moderate-to-severe bilateral hearing loss	Normal at birth; progressive bilateral loss starting in childhood or teenage years
Vision	Onset of RP prior to age 10	Onset of RP in late childhood or teenage years	Onset of RP in the second to fourth decade, which may vary in severity
Vestibular function	Vestibular areflexia or significant hypofunction	Normal	Varying degrees of dysfunction, from normal to areflexia; may be progressive

RP, retinitis pigmentosa.

that is clinically and genetically heterogeneous and characterized by three distinct subtypes (Table 25.4) based on hearing loss, a progressive loss of vision (*retinitis pigmentosa* (RP)), and varying degrees of vestibular dysfunction. RP manifests initially as difficulty seeing in the dark, followed by a progressive degeneration in the peripheral field of vision and, in end-stage disease, a loss of visual acuity. The prevalence of some types of Usher syndrome is higher among certain ethnic groups (e.g., Ashkenazi Jewish ancestry, Acadian populations in Louisiana) (Friedman et al., 2011).

There are 15 known loci for Usher syndrome and 11 causative genes identified, to date (Figure 25.2). Many of these genes are associated with autosomal recessive forms of nonsyndromic hearing loss and at least one mutant allele has a nonsyndromic dominant transmission (*DFNA11*). The most common mutations reported occur in *MYO7A* (11q13.5) and *CDH23* (10q22.1), both observed with Usher syndrome type I, and *USH2A* (1q41) which is associated with Usher syndrome type II, and which is also related to a nonsyndromic form of RP. The proteins encoded for by each of these genes (usherin, cadherin 23, and myosin VIIA, respectively) are all present in the inner ear and retina and contribute to the development, organization, and maintenance of the hair cells, and in particular the stereociliary bundle.

The manifestation of RP occurs in all three types of Usher syndrome and its presentation is not specific enough as a clinical measure to reliably distinguish phenotypes. Clinical evaluation of hearing and vestibular function is most useful to differentiate the type of Usher syndrome, in the absence of and before obtaining genetic information. All three subtypes are associated with hearing loss, but with variable onset, degree, and progression of the loss.

Children with Usher syndrome type I and type II will not pass a newborn hearing screening based on our current understanding of the onset of the disease. Patients with Usher syndrome type I may identify with the deaf community or benefit from cochlear implantation, or both. Most patients with Usher syndrome type II will benefit from hearing aids, as will those with Usher syndrome type III when the hearing loss progresses. Any child with congenital severe-to-profound hearing loss for whom the etiology of their loss has not been identified should be evaluated for possible Usher syndrome, especially if the child is a late walker or has delays in motor milestones. As is the case with any congenital or prelingual significant hearing loss, early identification and intervention is critical for language development. In the case of Usher syndrome, knowledge of the etiology and associated outcomes, which includes progressive loss of vision, has important prognostic and counseling implications for managing teams and families, including which communication mode and habilitation strategies are most appropriate. Comprehensive vestibular assessments are warranted for any patient diagnosed with Usher syndrome. Computerized dynamic platform posturography can serve as an especially useful tool, as the complex interaction of somatosensory, visual, and vestibular systems in the maintenance of balance is of concern in these patients for whom one or two of these systems are compromised.

Waardenburg Syndrome

Waardenburg syndrome is a genetically heterogeneous, rare (1:40,000 to 100,000) condition that causes pigmentary anomalies in the skin, hair, and eyes and is associated with sensory/neural hearing loss. There are four subtypes of Waardenburg syndrome, distinguished by their clinical presentation and mode of inheritance. Types I and II are the most common clinical subtypes and have similar phenotypes, but are differentiated by the presence or absence of *dystopia canthorum,* which is observed in type I and not in type II. This lateral displacement of the inner canthus of the eyes gives the appearance of a wide nasal bridge. Type III, also known as Klein–Waardenburg syndrome, has the same presentation as type I with the addition of upper limb

abnormalities (e.g., hypoplasia of limb muscles; contracture of elbows or fingers). Type IV, also known as Waardenburg–Shah syndrome, is similar to type II but includes an intestinal disorder called Hirschsprung disease (Friedman et al., 2003).

Types I, II, and III exhibit an autosomal dominant mode of transmission, but type IV segregates as an autosomal recessive disorder. Eight known Waardenburg syndrome loci and six genes have been identified to date. Waardenburg syndrome types I and III result from mutations in *PAX3* (2q35), which regulates the expression of several genes. Types II and IV are heterogeneous, with causative mutations associated with genetic expression (e.g., *MITF,* 3p14.1-p12.3) and genes involved with the development of pigment-producing cells called melanocytes (e.g., *SOX10,* 22q13). Melanocytes produce melanin that helps promote skin and eye color, but they are also important to the normal development and function of the inner ear, notably in the stria vascularis and vestibular dark cells.

Waardenburg syndrome may account for approximately 2% of congenital hereditary hearing loss. Much of the phenotype is associated with marked inter- and intrafamilial variable expression, including the hearing loss, which can range from normal to profound in degree. It is usually stable, but may be progressive, and can be unilateral or bilateral. Hearing loss appears to be more common in type II than type I, although it is possible some individuals with type II and less severe phenotypes may be diagnostically unrecognized because of the absence of dystopia canthorum. When the hearing loss is not profound, the configuration can reveal a greater loss in the low frequencies, or an inverted U, with sparing of mid-frequency hearing.

Additional phenotypic features of Waardenburg syndrome involve pigmentary anomalies that can include partial or complete *iris heterochromia* (differently colored areas of the same eye, or each eye being a different color) or strikingly blue irises. Distinctive patterns in hair color, often involving a congenital or premature white forelock, are common. Other facial features may include a wide, high nasal root, broad confluent eyebrow, and a square jaw.

Depending on the severity of the auditory phenotype, patients with Waardenburg syndrome may benefit from hearing aids or cochlear implantation. In addition to cochlear dysfunction, dysplasia of the semicircular canals and saccular degeneration have been observed on temporal bone study, and patients may present with vestibular complaints and dysfunction with or without accompanying hearing loss.

GENETIC EVALUATION AND DIAGNOSIS

As the professional who oversees newborn hearing screening programs and conducts confirmatory hearing tests, the audiologist is often the first to inform parents of their infant's hearing loss and initiate the referral process that begins a multidisciplinary partnership between healthcare professionals and the family. In addition to requisite otolaryngology referrals, a genetic evaluation and counseling should be offered to families of all infants with newly identified hearing loss (AAP and JCIH, 2007). It is appropriate for any person with hearing loss of unknown etiology, regardless of their age, to consider a genetic evaluation. The audiologist who suggests a genetic evaluation should have knowledge of the process and an understanding of the value and limitations of genetic testing.

Benefits and Limitations of Genetics Testing

Genetic testing is conducted for a variety of reasons, including carrier screening to assist in reproductive decisions, prenatal screening to detect the presence of a genetic condition in an embryo or fetus, newborn screening for current disorders (e.g., *biotinidase deficiency* which, if left untreated, may lead to hearing loss), presymtomatic predictive testing for hearing loss and other conditions that occur later in life, predispositional testing for genetic mutations that increase the risk of developing a condition (e.g., the well-known mutations in *BRCA1* or *BRCA2* associated with increased risk for breast or ovarian cancer), and diagnostic testing to determine the etiology of a disease (Arnos, 2003). In some cases, a genetic diagnosis may result in avoidance of expensive and more invasive tests. Establishing an etiologic diagnosis of congenital hearing loss provides answers to parents and may raise additional questions. Knowing the inheritance pattern contributes to understanding recurrence risks. In some cases, hearing loss may be the first manifestation of a syndrome for which critical medical intervention is necessary (e.g., JLNS) or that may have a significant impact on habilitation strategies (e.g., Usher syndrome). A genetic diagnosis may facilitate timely referrals to appropriate specialists and management of associated conditions. Predictive information about hearing loss progression is useful in planning management and making amplification and educational choices. In persons with an increased risk for age-related, noise-induced, or drug-induced hearing loss, a genetic diagnosis may lead to avoidance of environmental causes of hearing loss.

Although genetic testing can be beneficial, there are limitations. Some families may find the process and information emotionally upsetting. Genetic testing will not lead to a diagnosis in all cases; the etiology of congenital hearing loss may remain unknown in as many as 30% to 40% (AAP and JCIH, 2007). Negative findings on genetic testing do not mean that the hearing loss is not hereditary, but may occur when the causative gene or the specific mutation has not been previously identified. Ultimately, the decision to pursue genetic evaluation should be based on an informed family decision.

Diagnostic Evaluation of Hereditary Hearing Loss

A multidisciplinary team comprising audiologists, otolaryngologists, medical geneticists, geneticists, and genetic counselors is necessary for the diagnosis and management of hereditary hearing loss and family support. Guidelines for genetic evaluation to determine the etiology of congenital hearing loss include comprehensive review of (1) the patient's family history of hearing loss and other medical conditions, (2) the patient's medical history and risk factors for hearing loss, and (3) examination of the patient for physical features of a syndrome and other concomitant conditions (Table 25.5) (ACMG, 2002).

Construction of a pedigree based on at least three generations, identification of other medical problems, physical characteristics, or known genetic conditions in the family provides insight regarding the possibility of a syndromic form of hearing loss. Careful inspection and interpretation of family member audiograms and hearing loss history, including age of onset, comorbid conditions, and environmental exposures, assists in assuring that the auditory phenotype is correctly identified.

TABLE 25.5

Components of a Comprehensive Genetics Evaluation for the Etiologic Diagnosis of Congenital Hearing Loss

- Family history
  - Pedigree (three to four generations), attention to consanguinity, paternity, and hearing status
  - Ethnicity and country of origin
  - Inheritance pattern of the hearing loss
  - Audiometric characteristics of deaf and hearing impaired family members–age of onset, progression, degree, and type
  - Evidence of vestibular dysfunction
- Syndromic versus nonsyndromic features
  - Visual anomalies
  - Facial/cervical dysmorphology
  - Endocrine abnormalities
  - Cardiac signs or symptoms
  - Renal abnormalities
  - Integumentary changes
- Patient history–review of risk factors
  - Intrauterine infections (TORCH)
  - Prenatal exposure to alcohol or drugs
  - Postnatal infections (meningitis, varicella, herpes)
  - Extracorporeal membrane oxygenation (ECMO)
  - History of hypoxia
  - Exposure to ototoxic drugs
  - Prolonged NICU stay
- Physical examination
  - Otologic examination–Ear pits or cysts, pinna, ear canals, tympanic membrane, temporal bone
  - Craniofacial dysmorphisms
  - Airway examination
  - Other dysmorphisms or syndromic manifestations

The medical evaluation begins with a thorough history that reviews risk factors for hearing loss. These include in utero exposures such as maternal infections (cytomegalovirus, herpes, syphilis, and toxoplasmosis) and maternal drug or alcohol use. Neonatal risk factors comprise extracorporeal membrane oxygenation (ECMO), assisted ventilation, exposure to ototoxic medications, hyperbilirubinemia requiring exchange transfusion, neonatal intensive care stay lasting more than 5 days, or culture-positive postnatal infections such as meningitis which is associated with sensory/neural hearing loss (AAP and JCIH, 2007). For older children, a review of speech and language development, including vocal play, gives insight as to the time of hearing loss onset. Vestibular function in young children and toddlers can be indirectly assessed by factors such as age at independent walking, nystagmus, clumsiness, and torticollis. The patient should be examined for ear pits, tags or cysts, defects of the pinna, patency of the ear canals, and status of the tympanic membrane, as well as other craniofacial structural abnormalities.

Additionally, the comprehensive physical examination looks for system-wide features known to be associated with a syndrome that includes hearing loss. Johnston et al. (2010) recommend a comprehensive ophthalmologic evaluation on every child with a confirmed sensory/neural hearing loss to evaluate visual acuity and rule out other ocular disorders. Referrals to other medical specialists, including cardiologists, neurologists, and nephrologists, may be required based on clinical findings. Imaging studies (CT or MRI) of the temporal bone may be indicated for diagnostic purposes (e.g., enlarged vestibular aqueduct) and for assessment of surgical rehabilitative candidacy (e.g., cochlear patency).

It is also suggested that siblings of children identified with hearing loss be evaluated for hearing loss themselves and that this not wait until the etiology of the proband's hearing loss is identified, which may take time and which may never be fully confirmed.

Genetics Professionals

Geneticists and genetic counselors are important members of the multidisciplinary team involved in the diagnosis and management of someone with hereditary hearing loss. Geneticists participate in evaluation, diagnosis, and management of hereditary disorders. Genetic counselors are trained to (1) interpret family and medical histories and assess occurrence and recurrence risk; (2) educate patients and families regarding basic concepts of inheritance, available testing, and resources; and (3) provide

counseling to enable families to make informed decisions (NSGC, 2013). Typically, the family interacts with the genetic counselor to collect and review family medical histories and to ensure that the family understands the benefits and limitations of genetic testing. Additionally, results of genetic testing and the implications of a genetic diagnosis, or lack of a genetic finding, are explained and discussed with the family.

Genetic Testing

In 2002, genetic triage paradigms recommended testing for specific genes if a hearing loss syndrome was suspected. In the case of possible nonsyndromic hearing loss, testing for cytomegalovirus and genetic testing for a *GJB2* mutation served as the starting point (ACMG, 2002) for what amounted to a one-gene-at-a-time methodology. Recent technologies now make the process of identifying causative mutations more efficient, less expensive, and more accurate than ever before, allowing for the screening of thousands of genetic loci at one time. Hearing loss panels for simultaneously sequencing as many as 66 genes known to cause nonsyndromic hearing loss, and genes associated with common hearing loss syndromes such as Usher and Pendred syndromes are currently available at a number of facilities.

FUTURE DIRECTIONS

The proportion of infants with congenital or prelingual heritable hearing loss is expected to increase because of the successful development of vaccines for infectious causes of hearing loss (AAP and JCIH, 2007). In combination with new technologies such as whole *exome* sequencing, where all exons in the genome are examined, we will witness rapid and thorough testing for known hereditary hearing loss genes and the identification of new genes associated with auditory function.

Gene therapy for hearing loss is beginning to receive attention in research laboratories. It is a method that uses genetic material to halt, reverse, or prevent disease by replacing or stopping the function of a mutated gene or inserting a gene. Gene therapy is considered an experimental treatment and is used currently in very limited situations for humans with diseases that have no other cures. The application of gene therapy to treatment of sensory/neural hearing loss is in the early stages of discovery in animal models, but holds promise for eventual use in humans. The potential use of gene therapy in cases of hereditary hearing loss includes (1) delivery of functional copies of the gene to the cochlea to overcome the genetic defect, (2) delivery of genes that will initiate hair cell regeneration, and (3) delivery of genes capable of providing a protective effect on the cells of the organ of Corti and spiral ganglion to minimize the loss of these cells (Chien et al., submitted).

The ability to explore complex polygenic conditions, unravel the interactions between single mutations and their genetic background, and explain the dynamic relationship between genes and the environment is at a critical stage of advancement. Our understanding of all of this in relation to hearing loss is emerging, and future therapies, targeted measures aimed at preventing disease, and routine genetic screening will all be impacted by this new frontier of genetic research. This also means that any number of individuals with hearing loss previously thought to be of unknown origin will be identified as having a hereditary etiology.

ONLINE RESOURCES

Information about hereditary hearing loss continues to expand at a rapid rate. In addition to the information presented in this chapter, it is important for the audiologist to access reliable and up-to-date information regarding hereditary hearing loss. Table 25.3 presents a list of useful websites and a brief description of the website's content.

FOOD FOR THOUGHT

1. How much does the audiologist need to know about genetics? How has the answer to this question changed in recent decades, and how might it continue to evolve in the future?
2. Universal screenings at birth to identify mutations in genes that can affect development and health, including hearing loss, are on the horizon. What are the ethical implications associated with this testing, and will these data reduce or complement the need for universal newborn hearing screenings?
3. How is knowledge of the heritability of a patient's hearing loss incorporated in clinical practice currently, and how might this change in the coming years?

REFERENCES

ACMG. (2002) Genetics evaluation guidelines for the etiologic diagnosis of congenital of hearing loss. Genetic evaluation of congenital hearing loss expert panel. ACMG statement. *Genet Med.* 4, 162–171.

American Academy of Pediatrics, Joint Committee on Infant Hearing. (2007) Year 2007 position statement: principles and guidelines for early hearing detection and intervention programs. *Pediatrics.* 120, 898–921.

Arnos KS. (2003) The implications of genetic testing for deafness. *Ear Hear.* 24, 224–231.

Arnos KS. (2013) Epidemiology, etiology, genetic mechanisms, and genetic counseling. In: Toriello HV, Smith SD, eds. *Hereditary Hearing Loss and Its Syndromes.* 3rd ed. New York: Oxford University Press; pp 4–12.

Astuto LM, Bork JM, Weston MD, Askew JW, Fields RR, Orten DJ, et al. (2002) CDH23 mutation and phenotype heterogeneity: a profile of 107 diverse families with Usher syndrome and nonsyndromic deafness. *Am J Hum Genet.* 71, 262–275.

Bennett RL, French KS, Resta, RG, Doyle, DL. (2008) Standardized human pedigree nomenclature: update and assessment of the recommendations of the National Society of Genetic Counselors. *J Genet Counsel.* 17, 424–433.

Bischoff AM, Huygen PL, Kemperman MH, Pennings RJ, Bom SJ, Verhagen WI, et al. (2005) Vestibular deterioration precedes hearing deterioration in the P51 S COCH mutation (DFNA9): an analysis in 74 mutation carriers. *Otol Neurotol.* 26, 918–925.

Bondy CA, Turner Syndrome Study Group. (2007) Care of girls and females with Turner syndrome: a guidance of the Turner Syndrome Study Group. *J Clin Endocrinol Metab.* 92, 10–25.

Carey JC. (2003) Chromosomal disorders. In: Rudolph CD, Rudolph AM, eds. *Rudolph's Pediatrics.* 21st ed. New York: McGraw Hill; pp 731–741.

Chaïb H, Place C, Salem N, Chardenoux S, Vincent C, Weissenbach J, et al. (1996) A gene responsible for a sensorineural nonsyndromic recessive deafness maps to chromosome 2p22–23. *Hum Mol Genet.* 5, 155–158.

Chien WW, Monzack EL, McDougald D, Cunningham LL. Gene therapy for sensorineural hearing loss, in submission.

Cremers CW, Snik AF, Huygen PL, Joosten FB, Cremers FP. (2002) X-linked mixed deafness syndrome with congenital fixation of the stapedial footplate and perilymphatic gusher (DFN3). *Adv Otorhinolaryngol.* 61, 161–167.

Davis RR, Newlander JK, Ling X, Cortopassi GA, Krieg EF, Erway LC. (2001) Genetic basis for susceptibility to noise-induced hearing loss in mice. *Hear Res.* 155, 82–90.

De Leenheer EM, Ensink RJ, Kunst HP, Marres HA, Talebizadeh Z, Declau F, et al. (2002) DFNA2/KCNQ4 and its manifestations. *Adv Otorhinolaryngol.* 61, 41–46.

Dror AA, Avraham KB. (2010) Hearing impairment: a panoply of genes and functions. *Neuron.* 68, 293–308.

Edwards BM, Kileny PR, Van Riper LA. (2002) CHARGE syndrome: a window of opportunity for audiologic intervention. *Pediatrics.* 110, 119–126.

Fischel-Ghodsian N. (2003) Mitochondrial deafness. *Ear Hear.* 24, 303–313.

Friedman TB, Griffith AJ. (2003) Human nonsyndromic sensorineural hearing deafness. *Annu Rev Genomics Hum Genet.* 4, 341–402.

Friedman TB, Schultz JM, Ahmed ZM, Tsilou ET, Brewer CC. (2011) Usher syndrome: hearing loss with vision loss. *Adv Otorhinolaryngol.* 70, 56–65.

Friedman TB, Schultz JM, Ben-Yosef T, Pryor SP, Lagziel A, Fisher RA, et al. (2003) Recent advances in the understanding of syndromic forms of hearing loss. *Ear Hear.* 24, 289–302.

Fukushima K, Kasai N, Ueki Y, Nishizaki K, Sugata K, Hirakawa S, et al. (1999) A gene for fluctuating, progressive autosomal dominant nonsyndromic hearing loss, DFNA16, maps to chromosome 2q23–24.3. *Am J Hum Genet.* 65, 141–150.

Griffith AJ, Chowdhry AA, Kurima K, Hood LJ, Keats B, Berlin CI, et al. (2000) Autosomal recessive nonsyndromic neurosensory deafness at DFNB1 not associated with the compound-heterozygous GJB2 (connexin 26) genotype M34 T/167delT. *Am J Hum Genet.* 67, 745–749.

Guilford P, Ben Arab S, Blanchard S, Levilliers J, Weissenbach J, Belkahia A, et al. (1994) A non-syndrome form of neurosensory, recessive deafness maps to the pericentromeric region of chromosome 13q. *Nat Genet.* 6, 24–28.

Hildebrand MS, Tack D, Deluca A, Hur IA, Van Rybroek JM, McMordie SJ, et al. (2009) Mutation in the COCH gene is associated with superior semicircular canal dehiscence. *Am J Med Genet A.* 149A, 280–285.

Johnston DR, Curry JM, Newborough B, Morlet T, Bartoshesky L, Lehman S, et al. (2010) Ophthalmologic disorders in children with syndromic and nonsyndromic hearing loss. *Arch Otolaryngol Head Neck Surg.* 136, 277–280.

Jones SM, Jones TA. (2013) *Genetics, Embryology, and Development of Auditory and Vestibular Systems.* San Diego, CA: Plural Publishing.

Karzon RK, Hullar TE. (2013) Audiologic and vestibular findings in Wolfram syndrome. *Ear Hear.* 34, 809–812.

Kelsell DP, Dunlop J, Stevens HP, Lench NJ, Liang JN, Parry G, et al. (1997) Connexin 26 mutations in hereditary non-syndromic sensorineural deafness. *Nature.* 387, 80–83.

Kim TB, Isaacson B, Sivakumaran TA, Starr A, Keats BJ, Lesperance MM. (2004) A gene responsible for autosomal dominant auditory neuropathy (AUNA1) maps to 13q14-21. *J Med Genet.* 41, 872–876.

Kimberling WJ, Borsad N, Smith RJH. (2011) Hearing loss disorders associated with renal disease. *Adv Otorhinolaryngol.* 70, 5–83.

King KA, Choi BY, Zalewski C, Madeo AC, Manichaikul A, Pryor SP, et al. (2009) SLC26A4 genotype, but cochlear radiologic structure, is correlated with hearing loss in ears with an enlarged vestibular aqueduct. *Laryngoscope.* 120, 384–389.

King KA, Makishima T, Zalewski CK, Bakalov VK, Griffith AJ, Bondy CA, et al. (2007) Analysis of auditory phenotype and karyotype in 200 females with Turner syndrome. *Ear Hear.* 28, 831–841.

Kochhar A, Fischer SM, Kimberling WJ, Smith RJH. (2007) Branchio-oto-renal syndrome. *Am J Med Genet Part A.* 143A, 1671–1678.

Konigsmark BW. (1969) Hereditary deafness in man. *N Engl J Med.* 281, 713–720, 774–778, 827–832.

Lesperance MM, Hall JW 3rd, San Agustin TB, Leal SM. (2003) Mutations in the Wolfram syndrome type 1 gene (WFS1) define a clinical entity of dominant low-frequency sensorineural hearing loss. *Arch Otolaryngol Head Neck Surg.* 129, 411–420.

Madeo AC, Pryor SP, Brewer C, Zalewski C, King K, Butman JA, et al. (2006) Pendred syndrome. *Semin Hear.* 27, 160–170.

Makishima T, Madeo AC, Brewer CC, Zalewski CK, Butman JA, Sachdev V, et al. (2007) Nonsyndromic hearing loss DFNA10 and a novel mutation of EYA4: evidence for correlation of normal cardiac phenotype with truncating mutations of the Eya domain. *Am J Med Genet A.* 143A, 1592–1598.

Marsella P, Scorpecci A, Pacifico C, Tieri L. (2011) Bone-anchored hearing aid (Baha) in patients with Treacher Collins syndrome: tips and pitfalls. *Int J Pediatr Otorhinolaryngol.* 75, 1308–1312.

Mazzoli M, Van Camp G, Newton V, Giarbini N, Declau F, Parving A. (2003) Recommendations for the description of genetic and audiological data for families with nonsyndromic hereditary hearing impairment. *Audiol Med.* 1, 148–150.

McHugh RK, Friedman RA. (2006) Genetics of hearing loss: allelism and modifier genes produce a phenotypic continuum. *Anat Rec A Discov Mol Cell Evol Biol.* 288, 370–381.

Mehra S, Eavey RD, Keamy DG Jr. (2009) The epidemiology of hearing impairment in the United States: newborns, children, and adolescents. *Otolaryngol Head Neck Surg.* 140, 461–472.

Morell RJ, Kim HJ, Hood LJ, Goforth L, Friderici K, Fisher R, et al. (1998). Mutations in the connexin 26 gene (GJB2) among

Ashkenazi Jews with nonsyndromic recessive deafness. *N Engl J Med.* 339, 1500–1505.

Morton NE. (1991) Genetic epidemiology of hearing loss. *Ann N Y Acad Sci.* 630, 16–31.

Mujtaba G, Bukhari I, Fatima A, Naz S. (2012) A p.C343 S missense mutation in PJVK causes progressive hearing loss. *Gene.* 504, 98–101.

Nadeau J. (2003) Modifier genes and protective alleles in humans and mice. *Curr Opin Genet Dev.* 13, 290–295.

National Society of Genetic Counselors. (2013) About genetic counselors. Available online at: http://nsgc.org/p/cm/ld/fid=46. Retrieved October 3, 2013.

Riazuddin S, Ahmed ZM, Friedman TB, Griffith AJ, Riazuddin S, Wilcox ER. (2002) Genetic modifiers of hereditary hearing loss. *Adv Otorhinolaryngol.* 61, 224–229.

Riazuddin S, Castelein CM, Ahmed ZM, Lalwani AK, Mastroianni MA, Naz S, et al. (2000) Dominant modifier DFNM1 suppresses recessive deafness DFNB26. *Nat Genet.* 26, 431–434.

Schoen CJ, Emery SB, Thorne MC, Ammana HR, Sliwerska E, Arnett J, et al. (2010) Increased activity of Diaphanous homolog 3 (DIAPH3)/diaphanous causes hearing defects in humans with auditory neuropathy and in Drosophila. *Proc Natl Acad Sci USA.* 107, 13396–13401.

Schrauwen I, Weegerink NJ, Fransen E, Claes C, Pennings RJ, Cremers CW, et al. (2011) A new locus for otosclerosis, OTSC10, maps to chromosome 1q41–44. *Clin Genet.* 79, 495–497.

Schwartz PJ, Spazzolini C, Crotti L, Bathen J, Amlie JP, Timothy K, et al. (2006) The Jervell and Lange-Nielsen Syndrome: natural history, molecular basis, and clinical outcome. *Circulation.* 113, 783–790.

Siem G, Früh A, Leren TP, Heimdal K, Teig E, Harris S. (2008) Jervell and Lange-Nielsen syndrome in Norwegian children: aspects around cochlear implantation, hearing, and balance. *Ear Hear.* 29, 261–290.

Sliwinska-Kowalska M, Pawelczyk M. (2013) Contribution of genetic factors to noise-induced hearing loss: a human studies review. *Mutat Res.* 752, 61–65.

Smith SD. (2013) Genetic hearing loss with no associated abnormalities, In: Toriello HV, Smith SD, eds. *Hereditary Hearing Loss and Its Syndromes.* 3rd ed. New York: Oxford University Press; pp 98–2011.

Snoeckx RL, Huygen PL, Feldmann D, Marlin S, Denoyelle F, Waligora J, et al. (2005) GJB2 mutations and degree of hearing loss: a multicenter study. *Am J Hum Genet.* 77, 945–957.

Szymko-Bennet YM, Mastroianni MA, Shotland LI, Davis J, Ondrey FG, Balog JZ, et al. (2001) Auditory dysfunction in Stickler syndrome. *Arch Otolaryngol Head Neck Surg.* 127, 1061–1068.

Tsutsumi T, Nishida H, Noguchi Y, Komatsuzaka A, Kitamura K. (2001) Audiological findings in patients with myoclonic epilepsy associated with ragged-red fibers. *J Laryngol Otol.* 115, 777–781.

Van Camp G, Smith RJH. (2013) Hereditary hearing loss homepage. Available online at: http://hereditaryhearingloss.org. Retrieved October 3, 2013.

Varga R, Kelley PM, Keats BJ, Starr A, Leal SM, Cohn E, et al. (2003) Non-syndromic recessive auditory neuropathy is the result of mutations in the otoferlin (OTOF) gene. *J Med Genet.* 40, 45–50.

Wang Q, Xue Y, Zhang Y, Long Q, Asan, Yang F, et al. (2013) Genetic basis of Y-linked hearing impairment. *Am J Hum Genet.* 92, 301–306.

Wang QJ, Rao SQ, Zhao YL, Liu QJ, Zong L, Han MK, et al. (2009) The large Chinese family with Y-linked hearing loss revisited: clinical investigation. *Acta Otolaryngol.* 129, 638–643.

Weegerink NJ, Schraders M, Oostrik J, Huygen PL, Strom TM, Granneman S, et al. (2011) Genotype-phenotype correlation in DFNB8/10 families with TMPRSS3 mutations. *J Assoc Res Otolaryngol.* 12, 753–766.

CHAPTER 26

Educational Audiology

Cheryl DeConde Johnson and Carrie Spangler

INTRODUCTION

Audiology services in schools have been clearly defined in the Individuals with Disabilities Education Act (IDEA) since the law was first implemented in 1975. IDEA 2004 (US Department of Education, 2006) contains the most significant changes in policy since the inception of the law. This chapter will address educational audiology services, focusing on the roles and responsibilities as defined in IDEA as well as models for service delivery, caseload guidelines, licensure considerations, participation in the Individual Education Program (IEP) team, program development and evaluation, and ethics and conduct considerations.

EDUCATIONAL AUDIOLOGY SERVICES ACCORDING TO THE INDIVIDUALS WITH DISABILITIES EDUCATION ACT

Audiology is a related educational service under IDEA along with other related services such as speech-language pathology, psychology, and occupational therapy. IDEA is organized into several parts. This chapter will discuss Part B, which pertains to children 3 to 21 years old, and Part C, which pertains to infants and toddlers from birth to age 3 years. Although both sections address hearing loss, they have slightly different definitions (see Appendix 26.1). The definition differences directly impact an audiologist's responsibilities, and so are reviewed here:

- *Agency Responsibility.* Part B is under the education system in all states, whereas Part C responsibility depends on the specified lead agency within each state. Common agencies for Part C are education, health, or human services. Provision of services under Part C depends on several variables including income, the family's insurance, Medicaid and other state insurance programs, state agency services such as those provided through a state school for the deaf, and available services in the family's community. However, under Part C, a family should never be denied services because of inability to pay; ultimately, the community and state lead agency must provide funding for the necessary services.
- *Identification.* Part C specifies the use of appropriate screening techniques as part of identification; Part B does not.
- *Assessment.* Part C includes the assessment of communication functions as determined by the use of audiologic procedures.
- *Habilitation.* Assistive listening device orientation is included as part of habilitation in Part C; it is not mentioned in the Part B audiology definition but is included under the definition of assistive technology.
- *Prevention.* Part C provides for direct provision of services, whereas Part B calls for creation and administration of programs to prevent hearing loss. Direct services include monitoring children at risk of developing late-onset hearing loss.
- *Counseling.* Counseling services are absent in the Part C definition.
- *Amplification.* Part C includes selecting and fitting appropriate devices.

The following sections will discuss each of the areas within these audiology definitions.

IDENTIFICATION

Identification of children with hearing loss suggests several roles for audiologists. Identification does not explicitly mean hearing screening, but screening could be a step in the process toward identification of hearing loss. Resources and regulations in each state generally dictate the level of involvement of the audiologist at this stage. States that have mandated hearing screening of children in schools usually have the associated regulations within health or education agencies that direct those services. Because these procedures apply to all children, basic screening is considered a population-based event that should be completed by nurses, health aides, volunteers, or other individuals designated by the responsible agency, rather than a special education service.

Audiologists, however, do have a significant role in hearing screening programs. They should work with the appropriate state or local agencies to establish screening procedures, referral criteria, and follow-up activities as well as provide training for those individuals who perform the

screening. Screening procedures should be consistent with professional practice guidelines and should include measures that target identification of hearing loss in specific populations of students. For example, acoustic immittance may be part of a screening protocol for young children to identify middle ear problems, whereas the addition of 6,000 and/or 8,000 Hz to a puretone protocol for middle and high school–age students might identify potential noise-induced hearing loss. Audiologists may also assist with establishing databases to ensure that all students are screened and that follow-up is completed.

Children who are very young or unable to respond with traditional puretone screening methods may require special procedures as well as the expertise of an audiologist to conduct the screening. Otoacoustic emissions as a screening procedure have enabled more widespread screening of children who are young and difficult to assess by nonaudiologists. With effective training and supervision, audiologists can manage these screening programs, leaving time for follow-up screening, audiologic assessment, and other audiologic activities. The American Academy of Audiology (2011b) provides specific guidance, based on evidence-based practices, for audiologists involved with developing and managing hearing screening programs. Appropriate roles for audiologists in school screening may include the following:

- Facilitate multiagency and community collaboration for hearing identification and referral including programs for early hearing detection and intervention (EHDI).
- Coordinate efforts with nurses, local deaf/hard of hearing services teams, and relevant community resources to implement hearing identification, assessment, and referral procedures that are consistent with professional practice guidelines.
- Manage required preschool, school-age, and Child Find screenings following state and local policies and procedures. Depending on state guidelines, screening may be conducted by trained paraprofessionals or volunteers under the supervision of the school nurse or educational audiologist.
- Provide technical support to the screening team including training; perform screenings for difficult to assess children/students.
- Conduct follow-up activities to ensure that those referred have received the prescribed service.

ASSESSMENT

Audiologic assessment that is focused on communication access in the school environment extends the traditional clinical evaluation. The goal in the educational setting is to not only define the parameters of the hearing loss, including the necessary referrals for diagnosis, treatment, and hearing instrument fittings, but also determine individual educational implications. To fulfill this objective, audiologic assessment must address classroom listening including classroom acoustics, functional listening, and communication access. Other areas that should be considered are general developmental and educational performance, listening skills, hearing loss adjustment, and self-advocacy. For teens, assessment should include areas that teens need to know to function independently with regard to their hearing loss and communication needs and accommodations. Some of these areas can be addressed broadly through functional skill surveys such as the Functional Skills Screening for Children with Hearing Loss (Appendix 26.2). Assessment should always include information directly from the child whether as a series of questions or through play using a counseling tool such as My World (www.idainstitute.com). Table 26.1 summarizes these individual and environmental assessment areas and recommended procedures.

Hearing Assessment

Audiologic assessment of hearing includes standard measures as well as additional ones that yield a comprehensive profile of a child's auditory abilities. Assessment should include speech recognition tests that address the variety of listening situations encountered in the communication and learning situations such as the ability to understand soft speech and speech in noise. Audiologists should also include measures that provide detailed information regarding speech perception including ones that analyze suprasegmental (e.g., duration, loudness, pitch) and phonetic features of speech, phonemes, words, sentences, and discourse. The added information gained from knowing the auditory perception capabilities of these components assists speech-language pathologists and deaf education teachers in speech and auditory development, planning, and intervention. Otoacoustic emissions are available in most educational audiology settings and should be used when additional information is needed about the integrity of a child's auditory system or used with children who are low functioning or difficult to assess. Assessment should also include testing with the child's personal hearing instruments to assure that they are providing the intended benefit. Educational audiologists and private audiologists should work collaboratively to ensure that the comprehensive assessment covers all areas and duplication is minimized.

Communication Access Assessment

Assessment of communication access provides information on how children are communicating in their classroom and school environments with teachers and peers (normal hearing and deaf or hard of hearing).

TABLE 26.1

Educational Audiology Assessment for Children with Hearing Loss

Individual Measures	Environment Measures
1. Hearing • Case history • Otoscopic • Air and bone conduction • Speech reception (unaided/aided) • Acoustic immittance • OAEs (as appropriate) • MCL/UCL (unaided/aided) • Word recognition at soft and average hearing levels and in noise (unaided/aided) • Aided verification procedures for hearing assistance technology (probe microphone) • Aided validation procedures for hearing assistance technology 2. Communication access • Ida Institute "My World" and "Living Well" tools • Classroom Participation Questionnaire (Antia et al., 2007) 3. Classroom listening • Functional listening measures (e.g., Functional Listening Evaluation (FLE); Johnson, 2013a) • Listening Inventory for Education–R (LIFE-R) (Anderson et al., 2011) 4. Hearing loss adjustment • Self-Assessment of Communication–Adolescents (Elkayam and English, 2011a) • Significant Other Assessment of Communication–Adolescents (Elkayam and English, 2011b) 5. Self-advocacy • Self-Advocacy Competency Checklist (Guide to Access Planning, www.phonak.com) 6. General development • Functional Skills Screening for Children with Hearing Loss (Johnson, 2013b)	1. Classroom acoustics • Noise measurements • Reverberation measurements • Critical distance 2. Classroom communication • Classroom observation/teacher interview 3. Instruction • Classroom observation/teacher interview 4. Administrative support • Teacher/administrator interview

OAEs, otoacoustic emissions; MCL, most comfortable loudness; UCL, uncomfortable loudness.

A self-assessment protocol such as the Classroom Participation Questionnaire (Antia et al., 2007) is useful in identifying preferred communication patterns and determining how well the child is able to understand and be understood by peers and teachers. The protocol also includes information about the children's feelings (positive and negative) regarding their ability to communicate. The audiologist should use this information to make adjustments to hearing assistance technology (HAT) and to serve as a basis for counseling to help students understand and identify strategies they might employ to improve or remediate challenging communication situations. The information may also be informative regarding the appropriateness of placement decisions, particularly components under "special consideration" [34 CFR 300.324(2)(iv)] in the development of the IEP in IDEA.

Classroom Listening Assessment

Assessment of classroom listening skills is an essential component in the evaluation of children with hearing loss. Functional assessments such as the Functional Listening Evaluation (FLE) (Johnson, 2013b); observation tools such as the Early Listening Function (ELF) (Anderson, 2002), the Children's Home Inventory for Listening Difficulties (CHILD) (Anderson, 2000), the Listening Inventory for Education Revised (LIFE-R) (Anderson et al., 2011), the Children's Auditory Performance Scale (CHAPS) (Smoski et al., 1998), and the Functional Auditory Performance Indicators (FAPI) (Stredler-Brown and Johnson, 2004); as well as self-assessments such as the student component of the LIFE-R provide critical information about the development of listening and communication skills. These findings support needed

accommodations, document benefits of those accommodations, and identify areas of needed skill development for the IEP.

Hearing Loss Adjustment and Self-advocacy

Classroom performance is often affected by one's level of self-esteem, confidence, and friendships. Including measures in the assessment process, such as English's (2002) Children's Peer Relationship (CPR) Scale (Note: The CPR is a set of discussion points, not a test) and the Self-Assessment of Communication—Adolescents (SAC-A) and Significant Other Assessment of Communication—Adolescents (SOAC-A) by Elkayam and English (2011a, 2011b), identifies levels of self-identification and adjustment to hearing loss and issues associated with lack of adjustment.

The audiologist should also determine how well a student is able to self-advocate for his or her communication needs. The Self-Advocacy Competency Checklist (www.phonak.com) defines self-advocacy development based on what students should know at each level of school (e.g., elementary, middle, high school) (Appendix 26.3).

Bullying and Hearing Loss

Bullying is a growing topic that audiologists should address as part of self-concept and hearing loss adjustment (Squires et al., 2013). Bullying, particularly among school-age children, is a major public health problem both domestically and internationally. Pacer's National Bullying Prevention Center (2012) has reported studies indicating that children with disabilities are two to three times more likely to be bullied than their nondisabled peers.

Bullying is defined as unwanted, aggressive behavior among school-age children that involve a real or perceived power imbalance. The behavior is repeated, or has the potential to be repeated, over time. There are three types of bullying: (1) Verbal bullying (saying or writing mean things); (2) social bullying (sometimes referred to as relational bullying and involves hurting someone's reputation or relationships); and (3) physical bullying (hurting a person's body or possessions) (www.StopBullying.gov).

As practitioners, healthcare professionals should be vigilant for possible signs of victimization or bullying behavior among children and youth, particularly among high-risk youth such as children with disabilities or children who display characteristics of bully-victims. Healthcare professionals should ask children about their experiences with bullying and discuss possible concerns with parents. They should be prepared to make referrals to appropriate mental health professionals within the school or community (Fleming and Towey, 2002).

Identifying possible signs of bullying in children with hearing loss is an important counseling aspect of the role of the audiologist. Although the exact prevalence of bullying in children with hearing loss is unclear, it is at least as prevalent as typical children (Bauman and Pero, 2011) and there is evidence that deaf and hard of hearing children may be the most likely victims among the disability groups (Whitney et al., 1994). Professional associations in health care and safety are firm advocates for change whenever evidence suggests that the well-being of children is imminently at risk (www.stopbullying.org). The statistics and prevalence of students with hearing loss being a target are strong evidences that audiologists need to be firm advocates for the well-being of children with hearing loss who are imminently at risk.

Materials to guide audiologists with this issue are available on a website hosted by the American Academy of Audiology (www.audiology.org). A Bullying Decision Tree for Audiologists is a resource that can help audiologists begin the process of incorporating screening techniques into their audiology practice (Squires et al., 2013). The main focus of the website materials is to help audiologists identify the warning signs of bullying, investigate community and school resources, and provide ongoing support. Other tools include Bullying Screening Dialogue: Student Probes and Bullying Screening Dialogue: Parent Probes. These tools have been adapted from the "Roles for Pediatricians in Bullying Prevention and Intervention" (www.stopbullying.gov/resources-files/roles-for-pediatricians-tipsheet.pdf). The dialogue questions can help audiologists prepare themselves for this conversation, start the conversation, respond appropriately if there is a problem, and give proactive help if the child/family does not see a problem at the present time. The tools are intended to be used at frequent intervals to promote preventative practices in all stages of childhood and family life.

If a student is a victim of bullying, audiologists can help direct families to the educational and community resources that are available. Hands & Voices (wwwhandsandvoices.org) and Pacer's National Bullying Prevention Center (www.pacer.org) websites offer suggestions on how to address bullying specifically for children who have disabilities. Students with disabilities who are eligible for special education under the IDEA will have an Individualized Education Program (IEP). "The IEP can be a helpful tool in a bullying prevention plan. Remember, every child receiving special education is entitled to a free, appropriate public education (FAPE), and bullying can become an obstacle to that education" (www.pacer.org/bullying/resources/students-with-disabilities).

Instruments such as those mentioned in this section often provide counseling opportunities. The audiologist must be prepared in advance to address issues that are identified by the student during the assessment or interview process. Sufficient time should be scheduled during the assessment period or shortly thereafter to give students the opportunity to at least briefly talk about their problems and for the audiologist to begin to skillfully guide them through a problem-solving process. Anytime a student divulges sensitive information, it deserves at least acknowledgment and a response, even if brief. Time for more in-depth counseling can be scheduled once the "door has opened" (English, 2002).

Classroom Acoustics

Assessment includes measuring classroom noise, reverberation, and critical distance (CD) and making recommendations for improvement of the listening environment. Depending on the equipment used by the audiologist and the problems identified, the noise measurements may be sufficient for determining the need for acoustic treatment. The measurements may also be considered a screening that can be used as a basis for referral to an acoustic engineer. The American National Standards Institute/Acoustical Society of America (ANSI/ASA) classroom noise standards (ASA/ANSI S12.60-2010) provide the impetus for making acoustic modifications. These standards specify that, for typical classrooms (under 10,000 cubic feet), unoccupied noise levels should not exceed 35 dBA for the greatest 1-hour average and reverberation time (RT) should be ≤0.6 seconds. They further recommend that rooms be readily adaptable to allow reduction of RT to 0.3 seconds for children with special listening needs. Recent emphasis on educational outcomes for all students has highlighted the importance of the learning environment. Furthermore, it has been shown that poor classroom acoustics can also lead to voice fatigue and, subsequently, increased absences by teachers (Allen, 1995).

Classroom acoustics should be evaluated to provide evidence for HAT as well as the type of HAT that will best meet the child's communication needs. Rooms with high reverberation levels may preclude the use of classroom audio distribution systems (CADS also referred to as sound field systems) because sound distribution in highly reverberant areas may exacerbate speech intelligibility problems.

Audiologists in school settings should have a sound level meter (SLM) or smart phone app to conduct immediate screening of classrooms and other spaces. The app should be calibrated against a Type 2 or 3 SLM so that it can be adjusted, or corrections can be made for minor calibration differences. RT can also be measured with an app. If an RT app is not available, an estimated RT can be calculated using known sound absorption coefficients of typical materials used in school construction. CD is the maximum distance between the talker and listener before reflective sound begins to degrade speech transmission. CD is calculated based on room size and RT. Teachers need to know this distance and use this measure to maintain optimal speech communication for students with hearing loss. The American Academy of Audiology (2011a) has created a helpful worksheet for measuring classroom acoustics (see Supplement B).

Classroom Communication, Instruction, and Administrative Support

Interviews and observation provide important information about the appropriateness of the classroom context (e.g., general classroom physical environment, communication and instructional styles, the teacher's ability and flexibility in addressing the individual learning styles of students, and classroom management). These variables require careful attention when determining the classroom placement for a child as well as when making a recommendation for HAT. Administrative support is also critical in ensuring that the needs of the students are consistently met. School principals and special education administrators set the tone for their school's acceptance of students with diverse learning needs. Both school administration and teacher

- support for students with disabilities,
- knowledge about hearing loss,
- commitment to making the required accommodations for children with hearing loss,
- willingness to use and support assistive technology,
- willingness to work with specialists, and
- willingness to provide opportunities for individualized attention in the classroom.

Auditory Processing Assessment

Another component of audiologic assessment is the evaluation of central auditory processing abilities, an area of audition that should not be overlooked. The student's ability to understand what the ear hears is essential to the development of communication skills and for learning in school. The school setting is a common environment for identifying children with learning difficulties that may be auditory in nature. Educational audiologists should establish a multidisciplinary process with speech-language pathologists, school psychologists, and learning disabilities specialists to consider children who may have central auditory processing problems. The process should include screening and diagnostic procedures as well as intervention and treatment options. For more information on this topic, the reader is referred to Chapters 27 to 30 of this book.

Assessment Considerations for Eligibility for Services

To be eligible for special education, IDEA requires that there be an adverse impact of the disability on learning. Analysis of the findings must show that, without special education and related services, the child cannot reasonably benefit from a free and appropriate public education (FAPE). Therefore, audiologic assessment must include the procedures required by individual states for eligibility determination. Assessments are required for initial eligibility and re-evaluations which must occur minimally every 3 years. However, all children with hearing loss on IEPs should have annual audiologic assessments to monitor hearing thresholds, speech perception, listening abilities, use and performance of hearing technologies, and the child's functional performance. Even

though auditory sensitivity may be stable, the classroom listening environments change and therefore the accommodations and HAT, if used, need adjustment to assure the child has full communication access. The annual audiologic assessment should be written as a service in the child's IEP.

For children who are not eligible for special education services, the audiologist has a greater role because there is not a teacher of the deaf or other specialist monitoring them. In addition to audiologic assessment, educational performance should also be monitored at least annually to ensure academic level is maintained. This monitoring can be accomplished by the audiologist under a 504 Plan or as part of provisions under IDEA, Early Intervening Services or Response to Intervention (RTI). RTI is a general education program that focuses on high-quality instruction matched to student needs and monitoring of student progress to make decisions regarding educational programming. Audiologists play a critical role in this special education prevention program insuring that accommodations for students with hearing loss are implemented appropriately as part of monitoring student progress. Audiologists should be aware of their options for these programs and procedures according to their state regulations.

Interpretation of test results and recommendations should be detailed in a written report. It is helpful to include background information, test results, implications, and recommendations. A specific section of recommendations for the teacher and other school professionals often helps to highlight the most critical components and accommodations they need to know. Reviewing the report information with the student's teachers provides a forum for discussion as well as an opportunity for reinforcement of the issues, challenges, and necessary accommodations. Teachers appreciate when information is distilled to the essential elements they need to know. Therefore, including a short list of accommodations on a 4×6 card can be very helpful. By about third grade, the student should participate in the discussion, gradually taking the lead and developing his or her own list of accommodations. One tool to help a student take the lead is the Personal Profile and Accommodations Letter (PPAL) found within the Guide to Access Planning (GAP) program (www.phonakpro.com/us/b2b/en/pediatric/GAP.html).

AMPLIFICATION

Amplification and services that relate to both personal instruments and HAT are a major responsibility for audiologists in the school setting. IDEA details in its regulations provisions for amplification devices and monitoring the function of personal hearing instruments as well as HAT (Appendix 26.1).

The exclusion for cochlear implants was added in IDEA 2004 to limit the growing demands on schools for cochlear implant programming. IDEA 2004 also strengthened the responsibility of schools to monitor device function. As a result, schools should always include a monitoring plan that specifies who monitors the device, when it is conducted, the procedures used, and what will happen if a problem is identified. It is recommended that this plan be included in the IEP. See Supplement B of American Academy of Audiology (2011b) for a sample amplification monitoring plan.

HAT that is required for the child to receive FAPE must be designated in the IEP and provided by the school as well as the accompanying services that are indicated. These services begin with a functional assessment in the child's classroom or other "customary" environment. Tools such as functional listening evaluations, observation checklists, and self-assessments provide this essential information. These same tools are also used to validate the effectiveness of recommended devices to assure that they provide the desired benefit. Fitting of HAT should be conducted using professional practice standards including probe microphone measurements (American Academy of Audiology, 2011b) with the specific goal of enhancing audibility of the desired speech signal (usually the teacher's voice), while maintaining access to the discourse of classmates. Educational audiologists must have access to appropriate hardware and software to complete these fittings. Device selection considerations are based on environmental, individual, and technologic factors such as conditions within the learning environment, the age of the student, developmental abilities, device wearability and ease of use, compatibility with other technologies, and potential interference issues. Audiologists must also plan and implement orientation and education programs to assure realistic expectations and to improve the acceptance of, adjustment to, and benefit from HAT as well as from hearing aids and cochlear implants. These programs should include the student, the student's classmates, relevant teachers, and school support staff, as well as the parents when devices are used at home. To summarize, selecting and fitting HAT should include the following steps:

- Determination of candidacy for HAT
- Considerations of device options and device selection
- Fitting and verification procedures
- Orientation and training activities
- Validation procedures
- Monitoring procedures

HABILITATION

Educational audiologists provide a wide range of habilitation services depending on their specific responsibilities in the schools. Regardless of the specific services delivered, the educational audiologist should support and advocate for appropriate intervention methods that address state standards and include

- auditory skill development and listening skill training;
- speech skill development, including phonology, voice, and rhythm;

TABLE 26.2

Audiology Individual Education Program (IEP) Services and Suggestions for Where to Include them in the IEP

Service	Where to Include Service in the IEP
Training students regarding use of their hearing aids, cochlear implants, and hearing assistance technology	• IEP goals and objectives–counseling, assistive technology services
Counseling and training for students regarding self-determination and self-advocacy skills	• IEP goals and objectives–counseling
Recommending acoustic modifications based on classroom acoustic evaluations that structure or modify the learning environment	• Accommodations
Educating and training teachers, other school personnel, and parents, when necessary, about the student's hearing loss, communication access needs, amplification, and classroom and instructional accommodations and modifications	• Related services–counseling • Related services–parent counseling and training • Assistive technology needs and services
Monitoring the functioning of hearing aids, cochlear implants, and hearing assistance technology (by who, how often, where, procedures used to monitor, and what will occur when a problem is identified)	• Related services • Monitoring plan addendum

- visual communication systems and strategies, including speechreading, manual communication, and cued speech;
- language development (expressive and receptive) of oral, signed, cued, and/or written language, including pragmatic skills;
- selection and use of appropriate instructional materials and media;
- use of assistive technologies, such as those necessary to access television, and telephones, as well as pagers and alerting devices;
- case management and care coordination with family/parent/guardian, school, and medical and community services;
- habilitative and compensatory skill training to reduce academic deficits as related to, but not limited to, reading and writing;
- social skills, self-esteem, and self-advocacy support and training;
- transition skills for self-determination and integration into postsecondary education, employment, and the community;
- the transition between, but not limited to, levels, schools, programs, and agencies; and
- support for a variety of education options for children with hearing loss and other auditory disorders.

To provide input regarding the associated communication and educational implications of the hearing impairment and the needed services, the audiologist must attend all IEP meetings for students with educationally significant hearing loss. Specific habilitation activities that are the responsibility of the audiologist are identified in Table 26.2, along with suggested ways to document them in the IEP. Since the IEP is the contract that assures services, they must be included in the IEP, along with the frequency with which they are provided and who provides them.

COUNSELING

Counseling services can be provided for the student and the student's family, as well as school staff. Counseling students about their hearing loss provides information, emotional support, and the opportunity to develop self-advocacy skills. Students need to be able to generally describe their hearing loss and understand the necessary accommodations they need in various learning and communication situations. As discussed earlier in the chapter, the Self-Advocacy Checklist (www.phonak.com) suggests a framework for addressing some of these self-advocacy skills, whereas English (2012) provides a curriculum for self-advocacy activities. Audiologists should work with the educational team (school psychologist, speech-language pathologist, educational interpreter, and deaf education teachers as well as the students' classroom teachers) to assure that goals are consistently supported and developing skills are reinforced.

The incidence of emotional disturbance in students with hearing loss is low, about 2% according to the 2010 Gallaudet Annual Survey (Gallaudet Research Institute (GRI), 2010). The GRI also reports that 10% of students participating in its survey received counseling services. Whenever students exhibit significant problems, audiologists should also

refer and defer to the school counselor or school psychologist; preferably, this professional has expertise with children who are deaf and hard of hearing. When providing counseling services, the audiologist should

- assure that parents/guardians receive comprehensive, unbiased information regarding hearing loss, communication options, educational programming, and amplification options, including cochlear implants in cases of severe to profound hearing loss;
- demonstrate sensitivity to cultural diversity and other differences, including those found among individuals and within family/guardian systems and deaf culture; and
- demonstrate reflective listening and effective interpersonal communication skills.

Parent counseling and training is a separate, related service in IDEA. The law specifies that support should be provided to families if it is needed for their children to meet their IEP goals and to receive FAPE. Parents can choose whether or not they desire the support. Unfortunately, this service is underused and can be difficult to implement because of confusion about how to include the service in the IEP, how to provide the service, and how to promote and monitor parent compliance. IDEA states the following:

From the Federal Code of Regulations, Title 34, Section.300.24(c)(8)

i. Parent counseling and training means assisting parents in understanding the special needs of their child;
ii. Providing parents with information about their child's development; and
iii. Helping parents to acquire the necessary skills that will allow them to support the implementation of their child's IEP or IFSP.

Counseling for school staff is focused on information that teachers and others need to understand the implications of hearing loss and implement appropriate accommodations. Ongoing services should be included in the child's IEP as discussed in the previous section on habilitation.

PREVENTION

Based on the results of the Third National Health and Nutrition Examination Survey (NHANES III), Niskar et al. (2001) estimated that about 12% of children 6 to 19 years old have noise-induced hearing threshold shifts in one or both ears. Shargorodsky et al. (2010) reported data from 1998 to 2006 indicating that the incidence of noise-induced hearing loss among adolescents had increased significantly. In recognition of this evidence, a report called *Healthy People 2020* (US Department of Health and Human Services, 2010) includes the following objectives related to hearing loss prevention in adolescents:

Objective ENT-VSL 6.2. Increase the proportion of adolescents 12 to 19 years who have ever used hearing protection devices (earplug, earmuffs) when exposed to loud sounds or noise.

Objective ENT-VSL7. Reduce the proportion of adolescents who have elevated hearing thresholds, or audiometric notches, in high frequencies (3, 4, or 6 kHz) in both ears, signifying noise-induced hearing loss.

Prevention of hearing loss, even though required under IDEA, usually receives the least emphasis of all of the audiology services in school audiology practices. Concern in this area is growing based on some of the following issues:

- Schools, as government entities, are exempt from the US Occupational Safety and Health Administration's (OSHA) standards unless there are state OSHA-like requirements. Yet, shop class noise levels have been reported to range from 85 to 115 dB (Langford and West, 1993).
- Noise regulations that do exist in schools apply primarily to classified staff (e.g., grounds, facility, print shop, cooking staff).
- Insurance companies for schools have limited knowledge of noise exposure hazards.
- School hearing screening is not mandated in all states; thus, a mechanism to identify children with potential noise-induced hearing loss is not consistently available; when screening programs do exist, they generally are not designed to identify students with noise-induced hearing loss.

Although there are many resources available that provide hearing loss prevention education (e.g., Dangerous Decibels, www.dangerousdecibels.org; Crank it Down, www.hearingconservation.org; Wise Ears, www.nidcd.nih.gov/health/wise/index.htm), the difficulty lies in coordinating efforts for implementing a systematic hearing loss prevention education program within the curriculum. With the required effort that is necessary to address this area for all students, it is imperative this service be part of a larger agenda shared by health and general education services. Thus, noise-induced hearing loss needs a national focus as a preventable health condition. Given this information, educational audiologists should support such an effort by promoting the following activities (Johnson & Meinke, 2008):

- Noise education activities that are embedded in the school health and science curriculums at multiple grade levels
- Identification of "at-risk" and "dangerous" noise sources
- Mandatory noise safety instruction for classes with potentially hazardous noise exposure, including strategies to minimize noise exposure in those settings
- Mandatory use of hearing protection for all individuals who work in noise-hazard areas
- Mandatory monitoring of hearing levels of classified employees and teachers who work in noise-hazard areas
- Training for school employees in hearing loss prevention, proper use of ear protection, noise control strategies, and interpretation of hearing test results

- School policies to limit decibel levels and exposure time at school-sanctioned events
- Required hearing screening of students that includes protocols targeted to identification of noise-induced hearing loss

MODELS OF SERVICE PROVISION, CASELOADS, AND LICENSURE

Models of Service Provision

There are two primary methods that schools may use to deliver audiology services: (1) Employment directly by the local education agency (LEA) responsible for providing special education and related services or (2) through a contract with an individual, organization, or agency for specified audiology services. The LEA is either the local school district or a consortium established by the state that provides special education services for a group of school districts. These state consortia are usually referred to as Boards of Cooperative Educational Services, Intermediate Units, or Area Education Agencies, and are structured under the respective state department of education to provide special education services.

Although both models can be effective, educational audiologists hired by LEAs usually provide more comprehensive services. As employees of the education agency, educational audiologists are peers of the teachers and other staff and therefore may be more effective working within the system. Their schedules are designed around the school day resulting in more availability and flexibility for meeting student needs and providing teacher support, particularly for students who are not served under special education. Contracted services are often more limited because they are restricted to the specific services that are negotiated. School districts usually prioritize these services to the minimum necessary to provide follow-up to screenings, audiologic assessment, and HAT management. Contracts must assure that services are in compliance with state and federal requirements and that timelines are met. One of the most significant challenges with contracted services is supervision. In these situations, the school administrator providing oversight often has little knowledge of the roles and responsibilities of audiologists, and it is not uncommon for these audiologists to be less familiar with the scope of practice in the schools when they are from private practice or other noneducational settings. For more information about contracting audiology services, see the Educational Audiology Association's Guidelines for Developing Contracts for School-based Audiology Services at www.edaud.org.

Another employment setting that carries unique responsibilities for the educational audiologist is schools for the deaf. In this environment, the audiology role must support the communication systems that are used by the students. Still, it is the audiologist who bears much of the responsibility to assure auditory communication access to those students who utilize hearing and listening whether as a primary means of communication or to supplement visual systems (e.g., sign language). It often requires a slightly different approach, one that is sensitive to the preferences of the child, his or her family, and the culture of the school.

Caseloads and Workloads

Caseloads for audiologists are recommended at one audiologist for every 10,000 students (American Speech-Language Hearing Association, 2002; Educational Audiology Association, 2009). This ratio assumes a caseload of children with hearing loss and central auditory processing problems based on current prevalence rates. Workload analysis is another factor in determining the number of students served (Figure 26.1). Workload factors that may influence this ratio for a school system include but are not limited to

- the geographic area and travel time, such as within the LEA;
- the number of students with hearing loss served by the LEA:
  - the number of students with additional disabilities,
  - service provision to regional and/or self-contained programs for students who are deaf/hard of hearing,
  - involvement with hearing loss prevention programs and school-age hearing screening programs,
  - role in follow-up diagnostic audiologic assessment of hearing screening program,
  - involvement with RTI,
  - meeting federal, state, and local mandates for the child's IEP,
  - documentation of activities and Medicaid,
- the quantity and diversity of FM and other HAT;
- the quantity of special tests that are performed such as electrophysiological tests, auditory skill development, and auditory processing;
- the amount of in-house equipment calibration, test-check, and maintenance activities that are performed; and
- involvement with local newborn and early childhood screening follow-up, and early intervention.

When direct services to students are provided by the audiologist, the ratio must be further adjusted using caseload guidelines for consultant and itinerant teacher service delivery models. Figure 26.1 illustrates core components of the educational audiologist's workload. More information on workload analysis for audiologists is available from Johnson and Seaton (2012). ASHA also has workload guidelines for SLPS that can serve as a framework for educational audiologists.

Licensure

Audiologists who provide services in the schools must adhere to state licensing requirements. Several states have

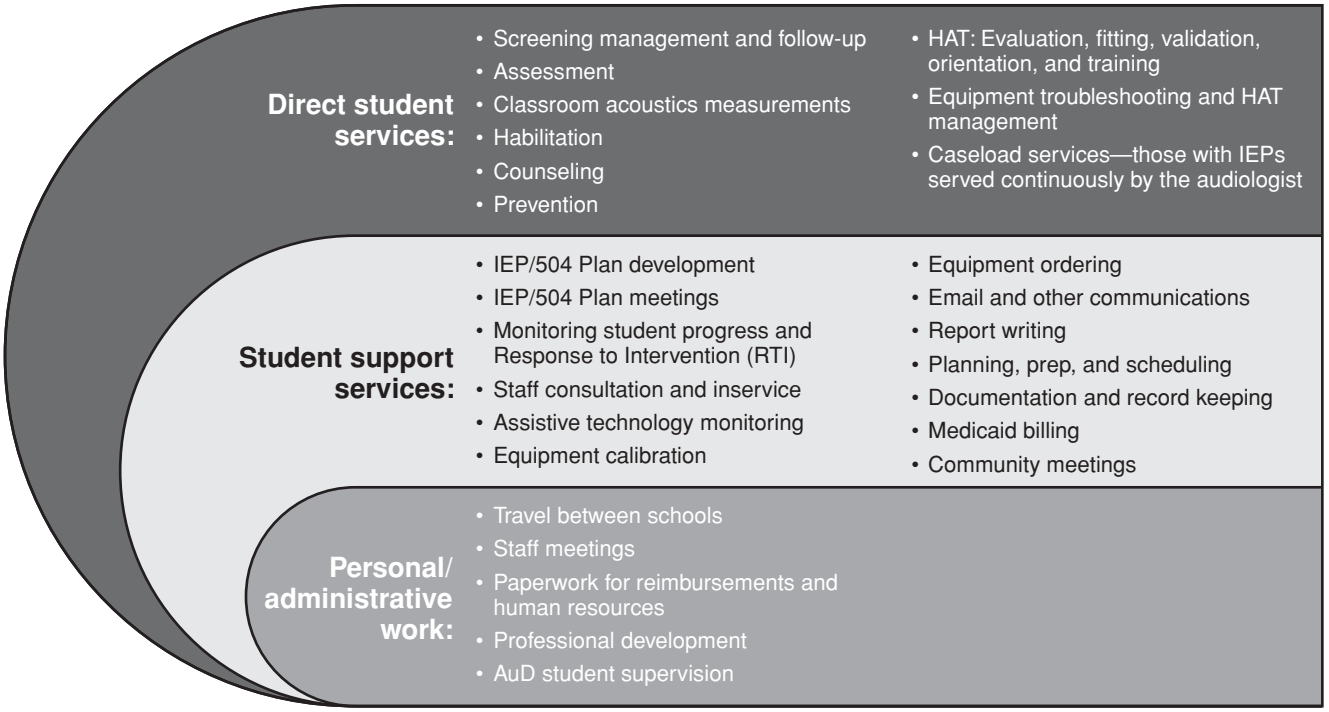


FIGURE 26.1 Workload model of educational audiology responsibilities.

specific certification or licensure for audiologists who are employed in school settings, which is administered through the state department of education. For example, audiology graduate programs in Colorado must demonstrate that their curricula meet knowledge and skill standards and complete a school-based practicum in audiology so that the graduates are eligible to meet the state's Department of Education qualifications in school-based audiology services.

Recognition of advanced specialty practice for pediatric and educational audiologists through the Pediatric Audiology Specialty Certification (PASC) by the American Board of Audiology (ABA) is another opportunity for audiologists to demonstrate and market their expertise. The PASC requires a 2-year postgraduate professional experience including 600 pediatric contact hours and a passing score in the PASC examination. The examination is built on knowledge and skills in seven domain areas: laws and regulations, general knowledge about hearing and hearing loss, child development, screening and assessment procedures, counseling, communication enhancement technology, and habilitation/rehabilitation strategies and educational supports. For more information, see http://www.americanboardofaudiology.org/ specialty/pediatric.html.

ROLE IN THE INDIVIDUAL EDUCATION PROGRAM TEAM

Special education eligibility and IEP development require input from the educational audiologist to ensure that information about the implications of the child's auditory, listening, and communication skills are understood. To assist in planning for the learning and communication of children with hearing loss, the audiologist uses data from a variety of sources (e.g., teacher, student, and parent interviews; observations; informal and formal assessments; classroom acoustic evaluations), explains the implications of hearing and listening on the child's ability to communicate and learn, and recommends appropriate HAT.

After a comprehensive profile of a child is developed, the educational audiologist is a resource for planning and implementing evidence-based interventions to increase academic, communication, and social performance. The audiologist's goal as an IEP team member is to collaborate with educators, parents, and other related service professionals to create supportive, communication-accessible learning environments for all students. When children are not eligible for special education, the audiologist should guide the development of an accommodations plan under another educational support system called a Section 504 plan (Johnson and Seaton, 2012) and should serve as the case manager to monitor implementation of the recommendations and the ongoing child's performance trajectories.

PROGRAM DEVELOPMENT AND EVALUATION

An effective educational audiology program undergoes continuous evaluation to determine whether the services continue to meet the needs of the students, staff, and others it serves. Mechanisms for identifying program gaps and

updating technology and services require systematic review and, in turn, help to prioritize future needs. Program components should focus on the impact of audiology services on student outcomes. With teacher evaluation increasingly tied to student performance, educational audiologists need to articulate how the services they provide also impact student performance. Data obtained through program service reviews, workload analysis, and teacher and parent input can be very powerful for affecting change.

ETHICS AND CONDUCT IN EDUCATION SETTINGS

The ethical responsibility for implementing the audiology requirements of IDEA based on the scope of practice discussed in this chapter can be challenging. Given the resource limitations that exist, educational audiologists frequently struggle with doing the right thing. The following questions are just some examples of the ethical dilemmas faced daily by educational audiologists:

- Are you able to evaluate students as often as best practices recommend?
- Are you able to conduct comprehensive assessment procedures that evaluate the functional aspects of hearing ability such as speech in noise, soft speech, speech with and without visual cues, and listening skills?
- Do you have access to current technology for conducting hearing evaluations and providing habilitation?
- Are you able to provide hearing assistive technology to *all* students who would benefit?
- Are you able to recommend the hearing technology that best suits a child's hearing needs?
- Are you able to meet with teachers and staff to discuss the results of each student's audiologic assessment and describe the implications of the loss on hearing and learning?
- Are you able to attend all IEP meetings for students with auditory deficits?
- Are you able to consult or teach students about the effects of noise exposure and requirements for hearing protection?
- Are you able to advocate for appropriate classroom acoustics?

Educational audiologists are often balancing the services they should provide with the resources (mostly time) available to do them. As a result, they often have to make decisions regarding which services should continue and which services need to be eliminated or modified. Reconciling the responsibility for providing "adequate" services versus what is often referred to as "the Cadillac model" can be challenging. Audiologists should be prepared to justify all of their activities under the scope of audiology that is mandated by IDEA to avoid the issue of their administrators perceiving that they are doing more than the law requires. An ethical question that is often faced is, "How does one balance the necessary audiologic services with what individual school settings allow?" Given the emphasis on accountability that is currently present in the education system, all audiologists should have an ongoing evaluation process for their program that provides data on student numbers, types of services, use of amplification, and student performance, coupled with a plan for addressing unmet needs. Sufficient evidence is needed before a special education director can justify supporting an audiologist's request for additional funds, time, or resources.

Effective audiology support and services are critical to all children and youth with auditory disorders. School-based audiologists have distinct roles and responsibilities to assure that these students are identified and properly assessed and managed so that they have the same opportunity to access their educational program as all students. They are simply children who have an extra communication challenge because of their hearing loss or processing issues; it is the job of the audiologist and the other school team members that support these students to minimize the impact of their impairments. The Educational Audiology Association (www.edaud.org) provides additional information and resources for school-based audiology services.

FOOD FOR THOUGHT

1. Compare the differences in Part B and Part C of IDEA. How do those differences impact the audiologist's responsibilities?
2. Consider the student enrollment (caseload size) for educational audiologists. What factors influence the audiologist's ability to provide compliance-based audiologic services? How might workload factors affect alter the caseload model?
3. How would your services as an educational audiologist shift as the student moves from elementary, middle, and high school?
4. Identify strategies the educational audiologist can use to increase student self-determination of accommodations to improve communication access at school.
5. The numbers of students with hearing loss who are not served by IEPs are increasing. Discuss how the educational audiologist might monitor and support these students.

APPENDICES for this chapter can be found at the end of the book.

REFERENCES

Acoustical Society of America. (2010) ANSI S12.60-2010 American National Standard acoustical performance criteria, design requirements, and guidelines for schools, Part 1: Permanent schools, and Part 2, relocatable classroom factors. Available online at: http://acousticalsociety.org/about_acoustics/acoustics_of_classrooms.

Allen L. (1995) The effect of sound-field amplification on teacher vocal abuse problems. *Paper presented at the Educational Audiology Association Conference,* Lake Lure, NC.

American Academy of Audiology. (2011a) Childhood hearing screening guidelines. Available online at: http://www.audiology.org/resources/documentlibrary/Documents/ChildhoodScreeningGuidelines.pdf.

American Academy of Audiology. (2011b) Clinical practice guidelines: Remote microphone hearing assistance technologies for children and youth from birth to 21 years. Supplement B: Classroom audio distribution systems—selection and verification. Available online at: http://www.audiology.org.

American Speech-Language Hearing Association. (2002) A workload analysis approach for establishing speech-language caseload standards in the schools: Guidelines. Available online at: http://www.asha.org/policy/GL2002–00066/#sec1.2.1.

Anderson K. (2000) Children's Home Inventory of Listening Difficulties (CHILD). Available online at: http://successforkidswithhearingloss.com.

Anderson K. (2002) Early Listening Function (ELF). Available online at: http://successforkidswithhearingloss.com.

Anderson K, Smaldino J, Spangler C. (2011) Listening Inventory for Education-Revised (LIFE-R). Available online at: http://successforkidswithhearingloss.com.

Antia SD, Sabers DL, Stinson MS. (2007) Validity and reliability of the classroom participation questionnaire with deaf and hard of hearing students in public schools. *J Deaf Stud Deaf Educ.* 12, 158–171.

Bauman S, Pero H. (2011) Bullying and cyberbullying among deaf students and their hearing peers: An exploratory study. *J Deaf Stud Deaf Educ.* 16 (2), 236–253.

Educational Audiology Association. (2009) Recommended professional practices for educational audiologists. Available online at: http://www.edaud.org.

Elkayam J, English K. (2011a) Self-Assessment of Communication – Adolescent (SAC-A). Available online at: http://gozips.uakron.edu/~ke3/SAC-A.pdf.

Elkayam J, English K. (2011b) Significant Other Assessment of Communication – Adolescent (SOAC-A). Available online at: http://gozips.uakron.edu/~ke3/SOAC-A.pdf.

English K. (2002) The Children's Peer Relationship (CPR) Scale. Available online at: http://successforkidswithhearingloss.com.

English K. (2012) *Self-ADVOCACY for Students Who Are Deaf or Hard of Hearing.* 2nd ed. Available online at: http://gozips.uakron.edu/~ke3/Self-Advocacy.pdf.

Fleming M, Towey K. (eds.) (2002) Educational Forum on Adolescent Health: Youth bullying. *Paper presented at the American Medical Association,* Chicago, IL.

Gallaudet Research Institute. (2010) *Regional and National Summary Report of Data from the 2010 Annual Survey of Deaf and Hard of Hearing Children and Youth.* Washington, DC: Author.

Guide to Access Planning (GAP). Available online at: http://www.phonakpro.com/us/b2b/en/pediatric/GAP.html.

Johnson CD. (2013a) Functional Listening Evaluation (FLE). Available online at: www.adevantage.org.

Johnson CD. (2013b) Functional Skills Screening for Children with Hearing Loss. Available online at: www.adevantage.org.

Johnson CD, Meinke D. (2008) Noise-induced hearing loss: Implications for schools. *Semin Hear.* 29 (1), 58–65.

Johnson CD, Seaton J. (2012) *Educational Audiology Handbook.* 2nd ed. Clifton Park, NY: Delmar Cengage Learning.

Langford J, West D. (1993) A study of noise exposure and hearing sensitivity in a high school woodworking class. *Lang Speech Hear Serv Schools.* 24, 167–173.

Niskar S, Kieszak S, Holmes A, Esteban E, Rubin C, Brody D. (2001) Estimated prevalence of noise-induced hearing threshold shifts among children 6 to 19 years of age: The Third National Health and Nutrition Examination Survey, 1988–1994, United States. *Pediatrics.* 108, 40–43.

Pacer National Bullying Prevention Center. (2012) Bullying and harassment of students with disabilities. Available online at: http://www.pacer.org/bullying/resources/students-with-disabilities/.

Shargorodsky J, Curhan SG, Curhan GC, Eavey R. (2010) Change in prevalence of hearing loss in US adolescents. *J Am Med Assoc.* 304 (7), 772–778.

Smoski W, Brunt M, Tannahill C. (1998) Children's Auditory Performance Scale (CHAPS). Available online at: http://www.edaud.org.

Squires M, Spangler C, Johnson CD, English K. (2013) Bullying is a safety and health issues: How pediatric audiologists can help. *Audiol Today.* 25 (5), 18–26.

Stredler-Brown A, Johnson CD. (2004) Functional Auditory Performance Indicators (FAPI). Available online at: http://arlenestredlerbrown.org.

US Department of Education. (2006) Assistance to states for the education of children with disabilities and preschool grants for children with disabilities; Final Rule, 34 CFR Parts 300 and 301. Available online at: http://idea.ed.gov/download/final-regulations.pdf.

US Department of Health and Human Services. (2010) *Healthy People 2020.* Washington, DC: Author. Available online at: http://www.healthypeople.gov/2020/default.aspx.

Whitney I, Smith P, Thompson D. (1994) Bullying and children with special educational needs. In: Smith PK, Sharp S, eds. *School Bullying: Insights and Perspectives.* New York, NY: Routledge; pp 213–240.

CHAPTER 27

Central Auditory Processing: A Functional Perspective from Neuroscience

Dennis P. Phillips and Rachel N. Dingle

INTRODUCTION

Generally, reviews of central auditory processing (CAP) take either of two approaches. One details the anatomy and physiology of the central auditory pathway, including such issues as synaptic organization, neurotransmitters, and neural circuits. The second approach is much more functional because it provides descriptions of the perceptual and cognitive processes involved in hearing and what goes wrong in various central disorders. In this chapter, we offer something of a "middle path": What we are interested in communicating is how the general neural architecture of CAP, as seen in neurophysiological studies, is related to the perceptual architecture that it supports. Those of us interested in functional hearing seek an understanding of CAP that links the properties of neurons (or of neural populations) responding to sounds with our private, mental experiences of those sounds. To be sure, we are a long way from a comprehensive understanding of this relationship. On the other hand, many exciting clues about this relationship are being reported.

The purpose of this chapter is to discuss specific topics that will provide a conceptual framework with which the reader can explore further. This chapter begins with the general anatomy and physiology of the central auditory system, starting with the auditory nerve and then ascending to the auditory cortex. We also provide a perspective on why the descending auditory pathways are so important. Lastly, because sounds inescapably unfold in both time and space, we discuss topics in central processing that emphasize temporal and spatial processing; great advances have been made in relating these neurophysiological and perceptual functions.

THE AUDITORY NERVE

The cochlea communicates with the auditory brainstem via spiral ganglion cells (the axons of which are sometimes called "auditory nerve fibers"). About 90% to 95% of spiral ganglion neurons form synapses with the cochlea's inner hair cells (IHCs). The form of this afferent organization is to connect one-to-many, that is, each spiral ganglion cell contacts a single IHC, but each IHC provides input to 20 to 25 auditory nerve fibers. There are no synaptic connections between spiral ganglion cells that serve any given IHC, nor is there significant information transmission between IHCs, with the result that the information transmitted by a given cochlear neuron is largely slave to the activity of the one IHC it innervates.

The cochlea performs a spectral decomposition of the incoming vibrations, such that different basilar membrane loci are forced into oscillation by sounds of different frequencies. This decomposition has the consequence that different IHCs, and thus different auditory nerve fibers, are responsible for transducing sounds of different frequencies and transmitting that information to the central nervous system. In practice, the motion of the basilar membrane at any given site is driven by two factors (Carney, 2012; Harrison, 2012). One is the passive response of the membrane to pressure waves in the cochlear fluids produced by motion of the stapes at the oval window. The second factor is the cochlea's "active process" or "cochlear amplifier." The active process can be thought of as an amplification of basilar membrane motion resulting from outer hair cell contractility (hair cell shortening as the membrane moves upward toward scala vestibuli, and hair cell relaxation as the basilar membrane moves downward toward scala tympani). The active process both enhances the sensitivity of the mechanical response and markedly improves its selectivity. The IHCs then passively receive the finely tuned basilar membrane motion and transduce it.

At low frequencies, the IHC membrane response is dominated by an alternating current component; the IHC depolarizes with upward motions of the basilar membrane and hyperpolarizes with downward ones. Because neurotransmitter release by the IHC is tied to membrane depolarization, the oscillating membrane potential affords neurotransmitter release and thus excitation of spiral ganglion cells selectively during upward motion of the basilar membrane. This means that the cochlear nerve spike trains evoked by low-frequency sounds carry information about

the phase of basilar membrane motion, and by extension, the phase of eardrum motion. This synchrony between neural spiking and ear drum motion or sound frequency is termed "phase-locking." At higher frequencies, the IHC membrane response is increasingly dominated by a direct current ("pedestal") depolarization response, which results in neurotransmitter release that is more continuous during the stimulus and impedes phase-locking of cochlear neuron action potentials to stimulus phase. To be sure, phase-locking of auditory nerve responses can occur for high-frequency sounds, but it is usually tied to the amplitude envelope of modulated signals and not to individual cycles of the carrier stimulus (see descriptions in Carney, 2012; Phillips et al., 2012b).

The spectral decomposition function of the cochlea is perhaps most clearly revealed in the frequency tuning curves of cochlear neurons (Figure 27.1A). A frequency tuning curve is a plot of the minimum tone amplitude in decibels required to evoke an increase in neural spike rate (above spontaneous levels) as a function of tone frequency. These tuning curves are deep, narrow, V-shaped functions. The tuning curves of high-frequency cells additionally have a high-threshold low-frequency tail. The frequency to which the neuron is most sensitive is called the characteristic frequency (CF) for that neuron. Without the cochlear amplifier, tuning curves are insensitive and broad (Harrison, 2012). Tuning curves for the basilar membrane motion at any given site are strikingly similar to those of cochlear neurons innervating that site. Thus, despite the change in response from up and down motion (basilar membrane) to discharge of action potentials (auditory nerve fiber), there is little change in the frequency tuning information being transmitted.

Another way to examine the frequency selectivity of cochlear neurons is by studying the response area, which is a plot of the spike firing rate as a function of frequency with tone amplitude as the parameter (Figure 27.1B; see also Carney, 2012). At low sound amplitudes, these functions are narrow and peaked. At high amplitudes, the functions are broader and rounder. The difference reflects the influence of the cochlear amplifier (at low sound pressure levels) giving way to passive mechanics (at high sound pressures; see also Plack, 2005). Consider Figure 27.1B. At this neuron's CF (4.5 kHz), the response is both sensitive (threshold near 20 dB) and grows over a dynamic range of about 40 dB before saturating. In contrast, at low frequencies (e.g., 2 kHz), the threshold is higher (approximately 55 dB) and the dynamic range is only about 25 dB wide. The responses near CF are shaped by the cochlea's active process, whereas those at high intensities are shaped by passive cochlear mechanics. As we step from responses to low-amplitude sounds to responses to high-amplitude ones, we also step from the active cochlea to the passive one. In the absence of the active process, cochlear neurons are both insensitive and have broad frequency responsiveness (see also Harrison, 2012). The behavioral correlates of this are loss of absolute sensitivity to sound ("hearing loss"), impaired frequency selectivity (because the fine frequency tuning is lost and so the spectral decomposition process is coarser), and possibly loudness recruitment (because neural firing rates go from minimum to maximum over a small amplitude dynamic range).

Figure 27.1 contrasts these two depictions of the frequency selectivity of cochlear nerve fibers. Figure 27.1A presents a tuning curve for an idealized cochlear neuron with a CF near 4.5 kHz. Figure 27.1B presents response area

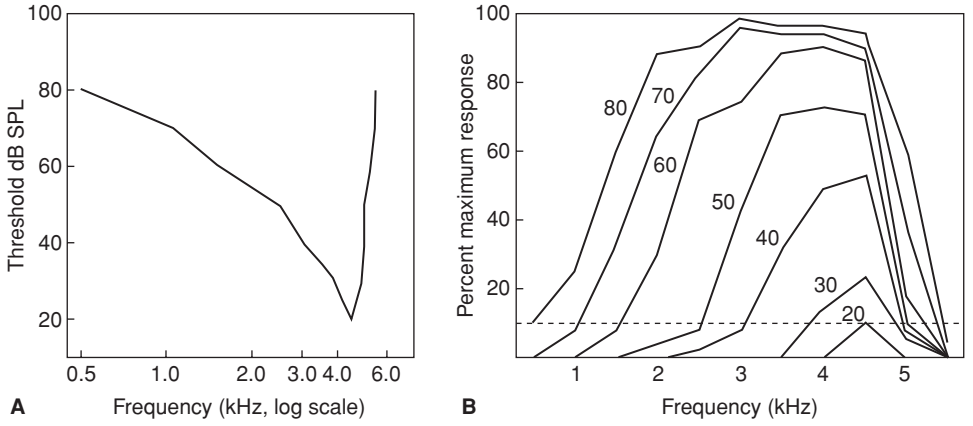


FIGURE 27.1 The frequency selectivity of an idealized cochlear neuron depicted in two ways. **A:** The frequency tuning curve, which is a plot of the minimum tone level required for and excitatory response plotted as a function of tone frequency. These are typically narrow and V-shaped, with a clearly defined characteristic frequency to which the neuron is most sensitive. **B:** Response area data for the same idealized neuron. Here, firing rate is plotted as a function of tone frequency with tone level in dB sound pressure level as the parameter. The tuning curve can be derived from the response area data by defining threshold as 10% of maximum firing rate (*dotted line* in **B**) and reading off the frequency at which each isointensity contour crosses it.

data for the same idealized neuron. The lines are isointensity contours, with intensity specified in dB sound pressure level. Note that at low tone amplitudes the effective frequency range is narrow and that as tone level is increased there is a negatively accelerating growth in response rate, and an expansion of the range of effective frequencies, especially toward the low-frequency side. The tuning curve in Figure 27.1A was derived from the response area (Figure 27.1B) by reading off the stimulus frequency at which each isointensity contour crossed the 10% maximum response rate (dotted horizontal line in Figure 27.1B).

Cochlear neuron physiology is thus the endpoint of the peripheral spectral decomposition process. It is the job of cochlear neurons to transmit to the brain the presence of stimulus energy within their tuning curves, the amplitude of that energy, and the timing of the stimulus event(s). It is the job of the central auditory system to represent this information (i.e., establish a "neurological picture" of it) and to group together the activity in different frequency channels to define separate auditory sources, their spatial locations, and their timings.

THE CENTRAL AUDITORY PATHWAYS

The Auditory Brainstem

Cochlear neurons send their axons to the cochlear nucleus (Figure 27.2); the axons bifurcate, with one branch going to the anteroventral cochlear nucleus (AVCN) and the other one going to the cells of the posteroventral cochlear nucleus en route to the dorsal cochlear nucleus (DCN). The cochlear nucleus is strictly organized tonotopically. That is to say, the projections from the cochlea to each division of the cochlear nucleus are topographically arrayed, so that information from each cochlear locus is sent to three sheets of neurons in the cochlear nucleus, and within each division, cells in sheets that are spatially next to each other have CFs that are spectrally next to each other. The fact that each division of the cochlear nucleus thus contains a complete representation of the cochlea's frequency organization permits a parallel processing of different streams of auditory information. As a first example of this, neurons of the AVCN, deriving input from the apical (low-frequency) cochlea, often have a low input resistance; they are, however, innervated by auditory nerve axons via end-bulbs of Held, special synapses that provide a large synaptic current. This matching between input resistance and synaptic current imparts a strong spike-in/spike-out relationship that preserves the time structure of the primary afferent spike train in the AVCN output. This preservation of temporal information becomes important in the medial superior olive (MSO) (a major target of AVCN output), because MSO neurons compare spike times from the two ears to compute sound source location from the relative phases of the stimuli at the two ears (see below). In

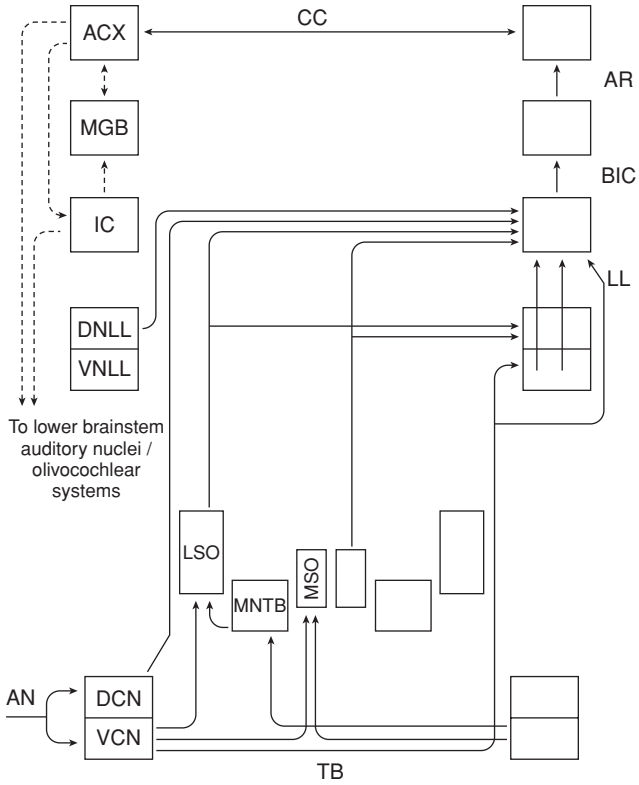


FIGURE 27.2 Schematic diagram illustrating the central auditory pathways. For simplicity, only major structures and pathways directly relevant to this chapter are shown. Ascending (body of illustration) and descending connectivities (left side) are for clarity shown for one side of the auditory forebrain only. Abbreviations: ACX, auditory cortex; AN, auditory nerve; AR, auditory radiations; BIC, brachium of inferior colliculus; CC, corpus callosum; DCN, dorsal cochlear nucleus; DNLL, dorsal nucleus of lateral lemniscus; IC, inferior colliculus; LL, lateral lemniscus; LSO, lateral superior olive; MGB, medial geniculate body; MNTB, medial nucleus of trapezoid body; MSO, medial superior olive; TB, trapezoid body; VCN, ventral cochlear nucleus; VNLL, ventral nucleus of lateral lemniscus.

contrast, the DCN contains circuitry that supports elaborate inhibitory domains in its cells' frequency-intensity response areas. Many of these cells develop nonmonotonic spike rate-versus-intensity functions and become sensitive to stimulus bandwidth because of inhibitory response areas flanking the excitatory one centered on the CF. The excitatory response areas of these cells can be very small, so that small changes in stimulus spectrum could in principle result in large changes in the populations of neurons activated. These specializations of the DCN likely serve to enhance the central representation of fine spectral detail.

A major target of projections from the ventral cochlear nucleus (VCN) is the superior olivary complex (SOC; Figure 27.2). The SOC contains a number of nuclei and these are also tonotopically organized. The MSO receives bilateral

inputs from the VCN, specifically from neurons that preserve the phase-locked spike timing that was generated in the auditory nerve. This means that MSO neurons can serve as "coincidence detectors" for the timing of spikes from the two sides and in this way compare the phase of low-frequency stimuli at the two ears. Early studies used tonal stimuli (usually CF ones) to examine sensitivity to interaural phase difference (IPD) of neurons rostral to MSO, with the inference being that what was observed in the more rostral nuclei was in part a reflection of processing at the MSO. They showed that the inputs from the two ears are ones phase-locked to basilar membrane motion on each side. In turn, basilar membrane motion is phase-locked to eardrum motion, and the phase relation of sound as it reaches each eardrum is dependent on the azimuthal location of the sound source. The firing rate of MSO cells depends on the temporal coincidence with which spikes from the two ears arrive. This behavior is to be expected of a mechanism that executes a cycle-by-cycle comparison of the phase of the stimulus at the two ears.

More recently, information about neural sensitivity to IPDs has been acquired using noise stimuli (Hancock and Delgutte, 2004; McAlpine et al., 2001). The basic ideas involved in this approach are (a) that central neurons respond to all of the frequencies within their tuning curves and (b) that by using noise stimuli, one obtains a weighted average of responses to IPDs at all of the frequencies to which the neuron is responsive. Plots of firing rate versus IPD obtained in this way are generally called "composite delay functions." These composite delay functions tend to have a single peak at a "best IPD" and may or may not have lower side-lobes. In guinea pigs (McAlpine et al., 2001), gerbils (Brand et al., 2002), and cats (Hancock and Delgutte, 2004) composite delay functions have at least two features in common. One feature is that the peak of the delay function, when the IPD is expressed in units of phase (as opposed to time), is nearly constant at about 45 degrees (i.e., about 1/8th of a cycle of interaural phase) favoring the contralateral ear, irrespective of a cell's CF. For animals with small heads, this means that the peak firing rate is evoked by IPDs outside the behaviorally relevant range. The second feature is a correlate of the first, namely that the steep part of the delay function, that is, the portion of the stimulus–response relationship that is most informative about the IPD of the sound, is centered very close to zero delay. That is, small changes in IPD near 0 μs (or degrees of phase angle) bring about large changes in neural firing rate. The fact that this is true in species with head sizes as different as those of gerbils, guinea pigs, and cats has important implications for the evolution of sound localization mechanisms in mammals (Phillips et al., 2012b).

In principle, the coincidence detection seen in MSO and higher neurons could be based solely on excitatory inputs from the two sides (after Jeffress, 1948). In practice, neural sensitivity to IPDs (and thus, sound source location) at the MSO and other nuclei typically involves inhibitory inputs—likely mediated through other SOC nuclei. These inhibitory inputs serve at least two functions. One is to increase the extent to which firing rates are modulated by variations in interaural phase. The second is to ensure that the maximum sensitivity to change in IPD is centered over IPDs very close to 0 μs (Brand et al., 2002). We shall return to this point below in the section specifically on sound localization mechanisms. For now, what is important is that the spike rate-versus-IPD function is most informative for IPDs relatively close to zero, with spike rates commonly at a maximum for IPDs favoring the contralateral ear and at a minimum for those favoring the ipsilateral ear. Behavioral sensitivity to the location of low-frequency sounds is also greatest for source locations near the midline, and so we have a clear neural correlate of behavioral performance (Hancock and Delgutte, 2004; Phillips and Brugge, 1985; Phillips et al., 2012b).

The lateral superior olive (LSO; Figure 27.2) also receives bilateral inputs from the VCN. The ipsilateral input to LSO cells is direct and excitatory. The contralateral input is via the medial nucleus of the trapezoid body and is inhibitory. The strength of each input is intensity dependent. LSO cells thus have firing rates that are sensitive functions of the relative amplitudes of the sound at the two ears. That is, they encode the interaural level difference (ILD) of a sound, and since the ILD is azimuth dependent, the sensitivity to ILD is a code for sound source azimuth (location). In practice, the spike rate-versus-ILD functions are sigmoidal in shape, with the strongest responses for stimuli in which the ILD favors the ipsilateral ear and with the steep portion of the function centered close to zero ILD.

The axonal outputs of the MSO are predominantly uncrossed and projected on the inferior colliculus (IC) directly, or indirectly via the dorsal nucleus of the lateral lemniscus (DNLL). The outputs of the LSO are predominantly crossed and again are projected directly on the IC, or indirectly on the IC via the DNLL. A consequence of this pattern of uncrossed and crossed connectivity is that above the level of the SOC, binaural neurons sensitive to IPDs and ILDs most often tend to be excited maximally by IPDs and ILDs that favor the contralateral ear. By extension, this means that the majority of forebrain auditory neurons sensitive to sound location cues should respond maximally to free-field sound sources in the contralateral auditory hemifield and minimally to sources in the ipsilateral hemifield and have spatial receptive field borders straddling the midline. Because the MSO and LSO outputs have minor crossed and uncrossed outputs, respectively, there should also be exceptions to this generality. As will be seen below, these hypotheses are confirmed empirically.

The convergence of input on the IC has some striking features. An excellent example comes from Semple and Aitkin (1981). They showed that some high-CF cells of the IC had physiologies and afferent connectivities indicating a

convergence of input from the DCN (e.g., nonmonotonic rate-intensity functions for CF tones) and binaural interactions reflecting LSO input. In a different example of hierarchical processing, whereas the responses of MSO cells to IPD stimuli are dominated by the instantaneous IPD of the stimulus, at the IC, neurons become sensitive to the recent history of stimulus IPD (i.e., they become sensitive to change in IPD, and thus simulated auditory motion: Spitzer and Semple, 1998). In yet another example, crossed inhibitory projections from the DNLL to the IC clearly "sharpen" the sensitivity of IC neurons to binaural sound localization cues (see Kidd and Kelly, 1996). What is less clear is whether the resulting sensitivity to localization cues is any better than that seen in the input sources (i.e., whether the "sharpening" is a sharpening per se or simply restores the sensitivity lost when cue-sensitive and cue-insensitive inputs converge.)

The Auditory Forebrain

The ICs project on the ipsilateral thalamic medial geniculate bodies (MGBs; Figure 27.2), which in turn project on the auditory cortex of the same side. The auditory cortex has a complex structural and functional organization (see Rauschecker and Romanski, 2011 for recent review). Particularly, in human beings and primates (probably also in cats), the auditory cortex is divided into "core" and "belt" regions, each of which is made up of a number of separable fields distinguished by their physiology and their afferent and efferent connectivities. Some of these fields are tonotopically organized, typically manifested as strips of cortical tissue containing neurons of comparable CF, and spatially arrayed to span the audible frequency range. Other fields have less obvious tonotopy and contain neurons with broad or irregular frequency tuning. In the tonotopic primary auditory cortex (AI), neurons are usually sharply tuned to frequency, have short response latencies, and have diverse binaural interactions that have been inherited and modified from those initially generated in the auditory brainstem. Studied with complex stimulus paradigms, it is clear that AI neuronal response areas display both a convergence of input within the excitatory response ranges and a development of inhibitory inputs outside those (Phillips and Hall, 1992). Within AI, neurons tend to be segregated into patches according to both their binaural interactions and their intensity coding properties.

Especially in anesthetized animals, responses to tonal or noise stimuli are usually dominated by a frequency-tuned and precisely timed onset component, suggesting that the cortex is especially concerned with the identity and timing of auditory events. Studied with complex sounds, for example, vocalizations, these onset responses can be "mapped" across the cortex, and the spectral content and timing of stimulus events can be quite faithfully represented in the (tonotopic) identities of the neurons contributing to the response and in the timing of those responses (Wang et al., 1995). In this regard, the precision with which transient responses of AI neurons are timed matches that seen in the cochlear nerve (Phillips and Hall, 1990), indicating that central auditory pathways have preserved this aspect of stimulus timing. In contrast, the temporal coding of stimulus periodicities (e.g., phase-locking to simple tones or to periodic amplitude modulation envelopes) is massively poorer (less than about 100 Hz) than that seen in cochlear neurons (c.f. Eggermont, 1991; Joris and Yin, 1992). This raises interesting questions about the neural coding underlying pitch percepts that typically rely on the existence of periodicities in the stimulus waveform (after Cariani and Delgutte, 1996) if the cortex is unable to support temporal representations of periodicities over the complete pitch range. There have, however, been recent advances that suggest that the neural codes underlying perceived pitch are transformed at the cortex, from temporal ones to rate ones and that the cortex may have dedicated circuits for pitch processing (Patterson et al., 2002; Wang and Walker, 2012).

Still more intriguing is the possibility that parts of the core and belt regions of the auditory cortex differentially participate in "what" and "where" streams of auditory processing (Lomber and Malhotra, 2008; Rauschecker and Romanski, 2011). By this, we mean that one stream of processing is composed of interconnected cortical territories containing a high proportion of neurons sensitive to sound source location, whereas another stream is characterized by a high proportion of neurons sensitive to sound source spectrum. This streaming appears to occur in cats (Lomber and Malhotra, 2008), nonhuman primates (Rauschecker and Romanski, 2011), and humans (Arnott et al., 2004). Most likely, the spatial stream may be involved not only in sound localization per se, but in providing input to multimodal cortical regions involved in spatially directed attention. The other stream may be the auditory corollary of the visual system's pathways for object identification or recognition.

The auditory cortex is also the source of a highly organized system of descending connections (for review, see Malmierca and Ryugo, 2012). The auditory cortex itself is under modulatory control from "higher" regions, with the result that attentional processes influence the responsivity of cortical regions in imaging studies, and the responsivity and selectivity of individual neurons in the auditory cortex in animal neurophysiological ones (Krumbholz et al., 2007; Lee and Middlebrooks, 2010). Some of these efferent pathways are ultimately involved in the control of middle ear muscle responses and modulation of otoacoustic emissions familiar to audiologists. These descending projections extend as far as the cochlea.

Scharf et al. (1994) studied attention effects in a patient before and after vestibular neurectomy (which necessarily severs descending axons to the cochlea). The listener's task was to detect low-amplitude tones of a target frequency against a background of noise. Occasionally, tones of nontarget

frequency were presented. Without surgery, correct detection of target tones was high, whereas that of the nontarget tones was low. This is evidence of selective attention in the frequency domain. Following surgery, the listener detected target and nontarget tones at comparable (high) rates, indicating a loss of that attentional control. Especially interesting is the implication that the effect of attention in this instance was a suppression of responses to nontarget tones, rather than any enhancement of the ability to detect tones of the target frequency. Likely, the absolute sensitivity of the cochlea is already as great as it can get, so attentional processes were expressed as an apparent suppression of cochlear output at the nonattended frequencies.

Feedback loops between the cortex and the midbrain and thalamic auditory nuclei have recently taken on special interest (Figure 27.2, left side). One of these loops is the reciprocal one between the cortex and the thalamic nuclei that provide the ascending input. The second is a loop formed by corticofugal projections from the cortex to the IC and the two-step ascending projection from the colliculus through the MGB to the cortex (Malmierca and Ryugo, 2012). These circuits enable the cortex to modulate or refine its own inputs so that inputs of the greatest behavioral significance receive an elaborated cortical representation (Suga, 2012). In what may be a prescient case of this phenomenon, Suga et al. (1987) reported that the "resting" frequency of mustached bat echo-location calls varied between individuals and that the tonotopic cortical maps of frequency were "personalized" to the individuals' resting frequencies. This is important in bats that often hunt in groups, because the bats need to be able to differentiate their own calls (and echoes driven by them) from those of other members of the group.

There is an arguably far more general importance of these circuits. As might be inferred from the preceding paragraph, these circuits may mediate forebrain auditory plasticity. That is, they may be a mechanism for optimizing the cortical representation of behaviorally relevant signals. This is one mechanism that may contribute to auditory learning. In Suga's terms, the cortex is able to self-select the afferent inputs of interest for cortical elaboration. In primates, temporally correlated activation of peripheral inputs drives cortical receptive field organization in the somatosensory system, and there is some evidence that in the auditory system, too, the behavioral relevance of stimuli enhances their cortical representation (Recanzone et al., 1993; see also Suga, 2012). Behavioral relevance might be construed as a plasticizing agent effected by the nucleus basalis of the basal forebrain on cortical synaptic connectivity (Weinberger, 2003). That is, the cholinergic (and possibly other) inputs serve as modulators or enablers of synaptic development at the affected neural loci. The following section details temporal synchrony and/or temporal coordination of the relevant inputs are critical to the development of the new synaptic connectivity.

THE IMPORTANCE OF NEURAL SYNCHRONY

Stimulus Representation

All sounds unfold over time. The fashion in which they do so is described by their time waveforms that specify the spectral and temporal content of the sounds. For simple tones of relatively low frequency, cochlear neurons and some of their direct and indirect central connections are able to synchronize ("phase-lock") action potentials to individual cycles of the stimulus (see above), so that the time structure of the stimulus is largely preserved in the temporal cadence of spike trains emerging from the cochlea. This property extends to more complex sounds, including speech, such that both glottal pulse rates and spectral elements of vowels have clear temporal representations in the central auditory system (Aiken and Picton, 2008). A case can be made that all sounds evoking clear pitch percepts have periodicities in the phase-locking range, whether they are at the level of the fine time structure or the amplitude envelope of the signal (Cariani and Delgutte, 1996).

Precisely how the nervous system extracts the pitch from the spike trains is unclear, but may involve an autocorrelation process in which the spike train is delayed and then compared to the original train (see Plack, 2005). The peak in the autocorrelation function will occur when the delay matches the periodicity in the spike trains, and thus the periodicity in the stimulus. Consider the case of rippled noise: A wideband noise is delayed and added to itself; this process is iterated repetitively and results in a noisy stimulus that evokes a pitch inversely related to the iterated delay (Patterson et al., 2002). If one were to perform an autocorrelation of the instantaneous amplitudes of the two waveforms themselves, then the peak of the function would occur at the iterated delay.

If the stimulus regularity is long enough in period, then the sound fails to evoke a clear pitch percept (Phillips et al., 2012a). This occurs for intervals longer than about 30 to 40 ms. Under these circumstances, discrimination of differences in periodicity still likely relies on synchrony of neural responses to stimulus event times, but the perceptual operation becomes one of the discrimination of the rhythm or relative timing of individuated stimulus events (Phillips et al., 2012a).

Somewhat analogous cases can be made for transient stimulus events. Auditory temporal gap detection will serve as a useful illustration (see Phillips, 2012 for review). The classical stimulus design in gap detection studies presents the listener with two streams of otherwise identical sound in which one stream has a silent period ("gap") inserted at some point in its duration. The task of the listener is to identify which stream of sound ("standard," "target") contains the gap. This two-interval, two-alternative forced choice is embedded in an adaptive, threshold-tracking staircase

designed to measure the shortest detectible gap. This general stimulus paradigm has been termed "within-channel" gap detection, because the operation required to detect the silent period ultimately reduces to the detection of a discontinuity of activity within the frequency channels carrying information about the presence of the gap. Gap thresholds decrease with increases in stimulus bandwidth. Gap thresholds for pairs of noise bands are lower than those for single ones, largely irrespective of the bands' frequency separation. Both of these findings might be explained by the fact that the more frequency channels that carry information about the presence of the gap, the greater is the efficiency with which the perceptual recovery of the gap is executed because more information is available. In turn, however, this process requires that the cochlear nerve (and central auditory system) time the stimulus continuity with great precision, because it is the temporal correlation/coherence of those event detections that is the neural database for the perceptual recovery of the gap.

A different form of the gap detection measurement is the one in which the sound following the silent period is different from the sound that precedes it (between-channel gap detection: See Phillips, 2012). Gap detection thresholds in this paradigm may be up to an order of magnitude (or more) greater than those seen in within-channel paradigms and may asymptote if the frequency disparity between leading and trailing sounds is sufficiently large. For widely frequency-spaced gap markers, the operation required to detect the silent period presumably involves a relative timing of the offset of activity in the neural/perceptual channel representing the leading marker and the onset of activity in the channel representing the trailing one (Phillips, 2012); in conditions of frequency overlap/proximity between the gap markers, other operations may contribute (again, see Phillips, 2012 for a detailed review). We shall return to the relative timing point later. For now, what is important is that efficient performance of the between-channel gap detection task requires a precise neural timing of the offset of the leading marker and the onset of the trailing one. The salience of that neural code depends on the synchrony of neural discharges to those stimulus events.

Neural Synchrony at the Synaptic Level

The preceding section emphasized the need of the auditory system to be able to synchronize neural action potentials with stimulus event times. A second expression of neural synchrony is in the ability of central neurons to synchronize their activity with each other. To explore this issue in principle, let us use the example of the role of glutamate receptors in the development of new synaptic strengths. The point here is to understand how it is that neurons alter the strengths of their synaptic connections (or for that matter, establish new connections), because alteration of connectivity strengths is a major expression of neural plasticity, quite likely including that involved in auditory learning (recalling the seminal work of Hebb, 1949). For a more detailed coverage of this topic, the reader is referred to almost any modern textbook of cellular or systems neuroscience (Bear et al., 2007).

Glutamate neurotransmitter receptors come in a number of forms, two of which are the α-amino-3-hydroxy-5-methyl-4-isoxazolepropionic acid (AMPA) receptor and the *N*-methyl-D-aspartate (NMDA) receptor. These frequently coexist in the postsynaptic membranes in receipt of glutamatergic input. AMPA receptors are ionotropic, meaning that binding of arriving (presynaptically released) glutamate opens the channel protein pore to admit sodium ions into the postsynaptic cell, and thus induce a modest depolarization (because sodium ions are positively charged and bring that charge with them). NMDA receptors in their resting state have their ion channel pore occluded by a magnesium ion. Binding of arriving glutamate is relatively ineffective in opening the pore, but postsynaptic depolarization (mediated by AMPA receptors) displaces the magnesium plug and permits major inflow of both sodium and calcium ions-–each of which is positively charged and thus effect significant postsynaptic depolarization. We therefore say that NMDA receptors are "voltage gated." The important feature here is that effective function of the synapse requires a coordination of presynaptic activity (release of glutamate) and postsynaptic activity (depolarization mediated by AMPA receptors) for the synapse to "work." The further important consequence of this is that the inflow of calcium ions through the NMDA receptors initiates a sequence of intracellular events that can result in the recruitment of new, preassembled AMPA receptors to the postsynaptic site. This has the consequence that presynaptic release of glutamate becomes more efficient at inducing postsynaptic depolarization, voltage-gating of the NMDA receptors, further depolarization, and potentially recruitment of still further AMPA receptors and stabilization of the synapse. This is likely a mechanism contributing to the so-called "long-term synaptic potentiation." In contrast, failure of this mechanism, perhaps because of poor presynaptic input or its coordination, can result in failure to maintain the synapse ("long-term synaptic depression").

Now consider the case of competing glutamatergic inputs to the same postsynaptic neuron. If one of the inputs is strong and has a strict temporal organization, whereas the other is weak and/or temporally sporadic, then the former input is more likely to stabilize or strengthen its connection, whereas the latter is likely to weaken its connectivity. Consider now the situation of a peripheral noise- (or other-) induced peripheral hearing loss at one or both ears. The effective inputs retain or expand their connectivity, whereas the impaired inputs lose theirs. This is neural plasticity, and it is no surprise that cochlear regions adjacent to the damage

expand their cortical representation at the cost of representation of the cochlear regions deprived of significant output. By the same token, if the behavioral importance of one input supersedes that of another (likely mediated by a modulation of synaptic plasticity exerted by the cholinergic nucleus basalis), then the behaviorally relevant input is selectively strengthened. This may be a neurophysiological underpinning of auditory learning. Again, the important points are that pre- and postsynaptic responses must be coordinated and that the rate and temporal properties of competitive inputs determine their relative developments of strength of connectivity.

These and other mechanisms (e.g., feed-forward inhibition: Eggermont and Roberts, 2004) may be at work in any reorganization of neural frequency-intensity response areas and in the reorganization of topographic cortical maps when there is a selective activation or a selective deactivation of afferent inputs. As mentioned above, this kind of selective modulation of inputs might occur after restricted cochlear hearing loss (Robertson and Irvine, 1989) and in auditory learning (Weinberger, 1997). It is also seen in studies using rearing in experimental acoustic environments that themselves produce no peripheral hearing loss (Norena et al., 2006; Pienkowski and Eggermont, 2010).

We can construe the thalamic input to the cortex as having a direct component that excites the target neurons. But it may also have a direct or indirect feed-forward ("lateral") inhibitory component (Eggermont and Roberts, 2004) that serves to suppress activity in adjacent neurons representing off-stimulus frequencies (e.g., through contribution to the development of so-called lateral inhibitory inputs to their response areas: Phillips and Hall, 1992). The frequency-restricted loss of afferent input not only deprives a focal cortical region of stimulus signal, but releases adjacent cortical regions from feed-forward inhibition. These are conditions that may permit the expansion of the (adjacent) functional inputs to deafferented recipient zones, resulting in changed neural response areas and distortions to tonotopic organization. A focal enhancement of afferent input, whether deriving from a cortical or thalamic source, is capable of expanding the cortical representation of the inputs that are selectively stimulated, and perhaps of suppressing the representation of adjacent inputs. In both cases, there will be changes in neural synchrony deriving from the changes in the efficiency of shared afferent inputs. As Eggermont (2007) remarks, temporally correlated neural activity may be the driving force for changes in the organization of both the properties of individual cortical neurons and the topographic organization of cortical fields. Precisely what functional deficits or advantages accrue from the neural changes described above is unclear, but this issue should be a major focus of future research. From the clinical standpoint, these data make clear that rearing conditions that do not induce peripheral hearing loss can nevertheless induce changes in central functional connectivity. This raises important issues for how we conceptualize central auditory processing disorder (CAPD).

Synchrony of Activity between Geographically Remote Brain Regions

There are good grounds to support the view that there is no (conscious) perception without attention. Thus, it becomes inescapable that forebrain neural circuits involved in attentional/cognitive processing need to be temporally coordinated with those involved in strictly sensory/perceptual processing to generate fully elaborated, conscious percepts of the relevant stimulus. For the audiologist, this point penetrates even the most basic, elemental levels of auditory examination. We have already seen this phenomenology in the work of Scharf and his colleagues (above). Clinically, the issue is important because to interpret poor hearing performance, one often needs to separate out sensory processing issues and attentional–cognitive ones. This may be particularly relevant in cases of CAPD (cf. Bellis, 2007; Moore et al., 2010) and in aging in which one might anticipate some attentional or cognitive deficits.

To be sure, one can assess auditory performance and attentional/cognitive state independently and try to infer from those measurements the source of poor hearing performance. Another approach is to employ preattentive electrophysiological measures, for example, the mismatch negativity response (MMN), to isolate strictly sensory function. A new question is whether one can objectively quantify the temporal coordination ("synchrony") between geographically remote brain regions that are required to execute the task at hand.

This brings us to advanced brain imaging methods. Magnetoencephalography provides a high spatial resolution and high temporal resolution of brain activity in which the temporal relations of activity in geographically remote brain regions can be assessed (Ross et al., 2010). Diffusion tensor imaging provides measures of the structural status of white matter pathways, and this may change with as little as 2 hours of task training (Sagi et al., 2012). Most recently, methods have been developed to measure white matter functional activation during task performance (Mazerolle et al., 2010). Certainly, all of these methods are still being refined and some of them can be expensive to employ because of scan-time fees. The point, however, is that these methods may prove to be immensely valuable in measuring the degree to which impairments in hearing performance reflect, or are associated with, impairments in the coordination of geographically separated brain regions mediating different (sensory, attentional, cognitive) contributions to task performance.

The importance of this point comes home if we consider the processes thought to contribute to performance in within- and between-channel gap detection tasks. In the

former case, the task of the listener is to detect the singular "glitch" or "hiccup" in the target stimulus. In the latter, it is currently assumed that a relative timing process is involved, that is, the listener may have to consciously execute a temporal ordering process (offset of the leading marker, onset of the trailing one, detection of a silent period between them). In this regard, differentially poor performance on the between-channel task may be helpful diagnostically (Phillips et al., 2010), but it fails to specify the level at which the deficit occurs (sensory coding, attentional processing). This is a nontrivial point because the between-channel version of the gap detection task is the more cognitively/attentionally demanding.

Behavioral Evidence

There are many diagnoses or specifiable disease processes that are capable of disrupting neural synchrony at one or more of the levels described above. These include multiple sclerosis, CAPD, auditory neuropathy (Starr et al., 1996), and aging (Pichora-Fuller et al., 2007). Let us consider the latter two.

Auditory neuropathy is now a relatively well-understood condition (c.f. Starr et al., 1996; Zeng et al., 2005). It involves pathology of the IHC-afferent fiber synapse and/or pathology of the cochlear nerve (and possibly more central neurons). Its hallmark features are (a) intact otoacoustic emissions, (b) severely impaired auditory brainstem response, and (c) deficits in speech perception more severe than would be expected on the basis of absolute sensitivity to sound (e.g., puretone average). The intact otoacoustic emissions are a sign that the cochlea's "active process" (and therefore the spectral decomposition function of the cochlea) is likely normal. The poor brainstem response speaks to impaired neural synchrony—at the level of neurons synchronizing their spike times with stimulus event times and/or at the level of the spike times of neurons responding to the same stimuli not being synchronous enough to support a measurable response at the scalp. The speech perception deficit, which is often particularly severe in noisy settings, is what one might expect in the face of impaired neural timing available to encode both the fine time structure of the speech signal and the timing of the phonetically important elements.

The conceptualization of auditory neuropathy as a case of neural dys-synchrony is bolstered by recent studies that have explored the electrophysiological and perceptual responses that depend on precise neural timing. Zeng et al. (2005) showed that neuropathy patients had impaired perceptual discrimination of stimuli encoded temporally (e.g., interaural time differences, pitch of low-frequency tones), but not of stimuli neurally represented by a rate/place code (e.g., ILDs, pitch of high-frequency tones). Kraus et al. (2000) provided a particularly thorough evaluation of an auditory neuropathy patient with normal audiograms and speech reception thresholds in quiet. As expected, they found that the patient's speech discrimination in quiet was very good, but significantly impaired (compared to controls) if the stimuli were presented against a noise masker. Analytical studies with a /ba/-/wa/ stimulus continuum (in which the independent variable was the duration of the formant transitions) revealed a normal just noticeable difference for transition duration. In contrast, studies with a /da/-/ga/ continuum (in which the independent variable was the starting frequency of the third formant transition) revealed an abnormally high just noticeable difference. The authors concluded that the patient had "difficulty discriminating stimuli that differ spectrally at stimulus onset and are characterized by rapid spectro-temporal changes throughout the formant transition" (p. 36). Interestingly, when the patient was studied for MMN responses to /ba/-/wa/ and /da/-/ga/ contrasts, responses were normal for the former, but absent for the latter (Kraus et al., 2000). Because the MMN is a preattentive response, this result suggests that the patient's difficulty was at a sensory representation level rather than at an attentional or cognitive one (although those may also have existed).

The recent literature on auditory processing in aging offers another intriguing hypothesis on the role of neural synchrony. Miranda and Pichora-Fuller (2002) showed that temporally "jittering" the low-frequency (<1.2 kHz) content of speech stimuli produced a perceptual deficit of "rollover" (reduced speech discrimination at high stimulus levels) in young, normal-hearing listeners mimicking that seen in aged listeners. This effect cannot be due to a spectral distortion caused by the jitter, because spectral smearing in the absence of jitter was without effect on word identification (Pichora-Fuller et al., 2007). The authors make the assertion that temporal jitter introduced to the stimulus provides a model or "simulation" of aging (Pichora-Fuller et al., 2007), that is, that the aging brain is prone to jittered neural timing.

SPATIAL HEARING

Sound localization is computational. In the visual and somatic sensory systems, there is a direct mapping of stimulus location onto the nervous system. It takes the form of neurons with spatially adjacent receptive field locations being spatially adjacent in the brain. This forms a topographic "map" of the world (e.g., skin surface, visual field) in which there is a "place code" for stimulus location, that is, stimulus location is specified by which neurons in the map are active. There is no such mapping of source location in the auditory periphery. Instead, central processes compute source location from location cue information that is present at the ear(s). The cue information comes in two forms: Monaural and binaural. Monaural cues are most helpful in determining sound source elevation and for making front/back discriminations. Binaural cues are important for localization in the azimuthal plane.

Monaural cues are made up of directionally dependent spectral filtering of a sound wave by the outer ear; reflection and absorption by the pinnae and the conchae result in specific changes in the amplitudes and frequencies within a sound, which are described by the "head-related transfer function" (HRTF). The characteristic modifications in a sound's waveform resulting from the HRTF change with the sound source position and so can serve as a cue for location. However, the use of these cues requires a listener to be familiar with the sound's spectrum and able to associate a particular filter with the correct location. This ability to compare a sound against pre-existing neural templates is probably partially learned and partially a result of innately making certain assumptions about a sound (such as that natural sounds will not contain the prominent peaks or notches caused by outer ear filtering; Middlebrooks, 1992) or about how characteristic filtering will change as sound (or as your head) moves. This HRTF filtering is unique for each individual, and damage to the outer ear can disrupt these location cues for a time until internal templates are relearned. Experiments in which ear molds have been used to alter the shape of participants' pinnae have shown that their abilities to use monaural cues is dramatically diminished at first, but returns to normal over a course of about 6 weeks (Hofman et al., 1998). Critically, participants in this experiment lost their ability to localize sound in the vertical plane while maintaining their ability to discriminate sound position along the azimuth, confirming that binaural cues are insufficient for vertical localization. On the other hand, whereas binaural cues are normally dominant for horizontal sound localization, some unilaterally deaf listeners can learn, in the absence of binaural information, to quite effectively use monaural cues for horizontal sound localization (Slattery and Middlebrooks, 1994).

Binaural cues are associated with the azimuthal position of a sound source, especially for sources within about 45 degrees of the midline. ILDs arise for frequencies with wavelengths shorter than head diameter because of the sound-shadowing effect of the head on signal level at the ear further from the source; for humans, ILDs are useful above about 1,500 Hz. For low frequencies, the extra travel time of the sound to the further ear imposes an interaural time (phase) difference (IPD) that is best resolvable by the nervous system below about 750 Hz. The computation involved in both cases is, thus, a comparison of the signals at the two ears.

There are countless descriptions of the fashions in which neurons of the central auditory system encode ILDs and IPDs. The broad picture is twofold. Because cochlear output is frequency specific and intensity dependent, it is possible for the auditory nervous system (esp. LSO, see above) to compare the signal levels at the two ears. Second, because low-frequency cochlear output is phase-locked to basilar membrane (and thus eardrum) motion, it is possible for central neurons (esp. MSO, see above) to compare the relative phases of those signals at the two ears.

For many years, the Jeffress (1948) model of a place code for sound localization has been highly influential in conceptualizing the "architecture" of sound localization mechanisms. It was offered initially for the encoding of interaural time differences and was predicated on the assumption that by means of a set of neural "delay lines," binaural neurons would develop a preferred interaural delay and that in principle, neurons could be spatially arrayed according to their preferred delays. This model played out exquisitely in barn owls in which it was shown that midbrain neurons had narrow ranges of preferred IPDs (which determined the azimuthal range of free-field receptive fields) and ILDs (which in owls specify the elevations of receptive fields) and were spatially arrayed to form a neurophysiological "map" of contralateral auditory space (see Konishi, 1993).

The model does not, however, appear to hold up in mammals (McAlpine et al., 2001; Phillips, 2008). For central neurons sensitive to ILDs, most are broadly tuned to disparities favoring the contralateral ear, although there are smaller populations tuned to disparities close to zero ILD or broadly tuned to ILDs favoring the ipsilateral ear. These data are matched by free-field observations in animal neurophysiology: Most spatially sensitive high-frequency neurons have receptive fields that are broadly tuned to contralateral azimuths, with medial borders near the midline, although there are smaller populations of cells with ipsilaterally or centrally (midline) located receptive fields (Lee and Middlebrooks, 2010; Stecker et al., 2005). For IPDs of noise stimuli, firing rates are broadly tuned to disparities favoring the left or right auditory hemifields (McAlpine et al., 2001). There is less evidence for a distinct population of neurons tuned to zero IPD, and there is a startling absence of data on the free-field receptive fields of low-frequency neurons (Dingle et al., 2013).

The neurophysiological data from animals have a striking parallel in data from human psychophysics. Using selective adaptation paradigms, it has been revealed that human perceptual channels for ILD-based azimuths are broadly tuned to left or right auditory hemifields or to midline locations and at both low and high frequencies (Dingle et al., 2012; Phillips and Hall, 2005). For IPDs, there is strong evidence for left and right auditory hemifields at both low and high frequencies, and modest evidence for a midline channel at low frequencies (Dingle et al., 2010, 2013). The current account, then, is that the neural code for source azimuth resides in the relative activation of two or three neural-perceptual channels, because those relative rates of activity uniquely specify sound source azimuth (after Phillips and Hall, 2005; Stecker et al., 2005). This model is reminiscent of that for color vision in which the relative activation of blue, green, and red cone systems enables the discrimination of a million colors. The existence of left and right azimuthal channels was predicted in an early auditory temporal gap detection study and the midline channel had been suspected

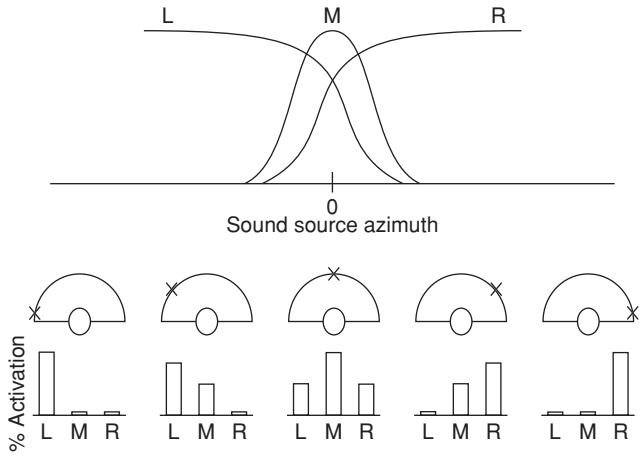


FIGURE 27.3 The three-channel model of sound localization mechanisms in mammals. **Upper diagram** shows the rate of activity aroused in each channel as a function of the azimuth of a sound source. L, M, and R refer to the left, midline, and right channels, respectively. **Middle panels** show the location of a sound source in relation to a listener's head. **Lower panels** show the relative activation of each channel evoked by the source locations depicted in the **middle row**. Note that each stimulus location is associated with a unique pattern of relative activation of the three channels.

to exist (see Phillips, 2008); for IPDs, the model has recently received independent support from human brain imaging studies (Salminen et al., 2010).

The functioning of the three-channel model is illustrated schematically in Figure 27.3. The upper part shows the activation of left (L), midline (M), and right (R) channels as a function of sound source location (in degrees of azimuth). The middle row of images depicts selected sound source locations relative to the listener's head, and the bottom row of histograms shows the relative activation of the three channels for each source location depicted in the middle row. The important point to be gleaned from this illustration is that each sound source azimuthal location is represented by a unique distribution of activity across the three channels. How far the "skirts" of the channels' tuning extend is not known with certainty. However, note that small changes in source azimuth will bring about the greatest changes in the distribution of channel activity for locations relatively close to the midline. This is where behavioral spatial acuity is at its greatest. It reflects two related factors. One is that disparity size versus azimuth functions are steepest near the midline, especially for ILDs. The second is that neural spike rate versus disparity size functions are steepest for disparities close to zero (Phillips and Brugge, 1985). Restated, the neural code for disparity size is most unambiguous over disparity sizes which themselves most precisely specify source azimuth. It is thus no surprise that spatial acuity is greatest around the midline.

CONCLUSION

Sounds unfold over time. We are used to the notion that the auditory system is able to encode the spectral identity of sounds, but it is becoming increasingly apparent that sound source identity and location can reside as much in the temporal properties of the stimuli at the ears as in the spectral ones. This is obvious in cases such as temporal ordering and sound localization, but it penetrates down to the generation of pitch percepts and temporal regularity, and neural timing clearly plays an important role in the coordination of responses involved in the generation of central representations of sounds, auditory plasticity, and sound detection and discrimination. Methods are available for the behavioral, electrophysiological, and brain imaging measurements of these temporal processes. With the understanding that "temporal processing" has become an umbrella term with a wide capture, it behooves us to dissect out these processes and to use the resultant data to define, or redefine, disorders of CAP.

FOOD FOR THOUGHT

1. Consider the following two assertions. (A), If you practice auditory temporal processing intensively and in a structured way, then you will improve your auditory temporal processing skills. (B), If you practice auditory temporal processing intensively and in a structured way, then neuroplasticity will kick in and you will improve your auditory temporal processing skills. Which of those two assertions carries more weight with you, and why? If you substituted "caber tossing" or "cake baking" for "auditory temporal processing," would your answer change?
2. It is argued that in mammals, the perceived location of a sound in the azimuthal plane depends on the relative outputs of two or three perceptual channels, each of which is rather broadly tuned to source location. In the case of two-channel systems, this is sometimes termed "opponent processing." How many instances of opponent processing can you find in other sensory or cognitive systems? That is, how common is the implementation of this strategy in the brain?
3. Suppose that an otherwise normal person was born with the following abnormality: their left cochlea projects upon the right cochlear nucleus, and their right cochlea projects upon the left cochlear nucleus. What would be the consequences of this abnormality for that person's hearing?

ACKNOWLEDGMENTS

The preparation of this chapter, and some of the research described herein, was supported by grants from NSERC of Canada and the Killam Trust to DPP. RND was supported by an NSERC postgraduate scholarship. Many thanks are due to Susan E. Hall for her input at all stages of this work.

KEY REFERENCES

thePoint A full list of references for this chapter can be found at http://thePoint.lww.com. Below are the key references for this chapter.

Aiken SJ, Picton TW. (2008) Envelope and frequency-following responses to vowel sounds. *Hear Res.* 245, 35–47.

Arnott SR, Binns MA, Grady CL, Alain C. (2004) Assessing the auditory dual-pathway model in humans. *NeuroImage.* 22, 401–408.

Bear MF, Connors BW, Paradiso MA. (2007) *Neuroscience. Exploring the Brain.* 3rd ed. Philadelphia, PA: Lippincott Williams and Wilkins.

Bellis TJ. (2007) Historical foundations and the nature of (central) auditory processing disorder. In: Musiek FE, Chermak GD, eds. *Handbook of (Central) Auditory Processing Disorder. Volume 1. Auditory Neuroscience and Diagnosis.* San Diego, CA: Plural; pp 119–136.

Brand A, Behrend O, Marquardt T, McAlpine D, Grothe B. (2002) Precise inhibition is essential for microsecond interaural time difference coding. *Nature.* 417, 543–547.

Cariani P, Delgutte B. (1996) Neural correlates of the pitch of complex tones. I. Pitch and pitch salience. *J Neurophysiol.* 76, 1698–1716.

Carney LH. (2012) Peripheral anatomy and physiology. In: Tremblay KL, Burkard RF, eds. *Translational Perspectives in Auditory Neuroscience. Normal Aspects of Hearing.* San Diego, CA: Plural; pp 91–112.

Dingle RN, Hall SE, Phillips DP. (2010) A midline azimuthal channel in human spatial hearing. *Hear Res.* 268, 67–74.

Dingle RN, Hall SE, Phillips DP. (2012) The three-channel model of sound localization mechanisms: Interaural level differences. *J Acoust Soc Am.* 131, 4023–4029.

Dingle RN, Hall SE, Phillips DP. (2013) The three-channel model of sound localization mechanisms: Interaural time differences. *J Acoust Soc Am.* 133, 417–424.

Eggermont JJ. (1991) Rate and synchronization measures of periodicity coding in cat primary auditory cortex. *Hear Res.* 56, 153–167.

Eggermont JJ. (2007) Correlated neural activity as the driving force for functional changes in auditory cortex. *Hear Res.* 229, 69–80.

Eggermont JJ, Roberts LE. (2004) The neuroscience of tinnitus. *Trends Neurosci.* 27, 676–682.

Hancock KE, Delgutte B. (2004) A physiologically based model of interaural time difference discrimination. *J Neurosci.* 24, 7110–7117.

Harrison RV. (2012) Anatomy and physiology of the cochlea. In: Tremblay KL, Burkard RF, eds. *Translational Perspectives in Auditory Neuroscience. Normal Aspects of Hearing.* San Diego, CA: Plural; pp 65–90.

Hebb DO. (1949) *The Organization of Behavior.* New York, NY: Wiley.

Hofman PM, Van Riswick JA, Van Opstal AJ. (1998) Relearning sound localization with new ears. *Nat Neurosci.* 1, 417–421.

Jeffress LA. (1948) A place theory of sound localization. *J Comp Physiol Psychol.* 41, 35–39.

Joris PX, Yin TCT. (1992) Responses to amplitude-modulated tones in the auditory nerve of the cat. *J Acoust Soc Am.* 91, 215–232.

Kidd SA, Kelly JB. (1996) Contribution of the dorsal nucleus of the lateral lemniscus on binaural responses in the inferior colliculus of the rat: Interaural time delays. *J Neurosci.* 16, 7390–7397.

Konishi M. (1993) Listening with two ears. *Sci Am.* 268, 66–73.

Kraus N, Bradlow AR, Cheatham MA, Cunningham J, King CD, Koch DB, et al. (2000) Consequences of neural asynchrony: A case of auditory neuropathy. *J Assoc Res Otolaryngol.* 1, 33–45.

Krumbholz K, Eickhoff SB, Fink GR. (2007) Feature- and object-based attentional modulation in the human auditory "where" pathway. *J Cogn Neurosci.* 19, 1721–1733.

Lee CC, Middlebrooks JC. (2010) Auditory cortex spatial sensitivity sharpens during task performance. *Nat Neurosci.* 14, 108–114.

Lomber SG, Malhotra S. (2008) Double dissociation of 'what' and 'where' processing in auditory cortex. *Nat Neurosci.* 11, 609–616.

Malmierca MS, Ryugo DK. (2012) Descending connections of auditory cortex to the midbrain and brain stem. In: Winer JA, Schreiner CE, eds. *The Auditory Cortex.* New York, NY: Springer; pp 189–208.

Mazerolle EL, Beyea SD, Gawryluk JR, Brewer KD, Bowen CV, D'Arcy RCN. (2010) Confirming white matter fMRI activation in the corpus callosum: Co-localization with DTI tractography. *NeuroImage.* 50, 616–621.

McAlpine D, Jiang D, Palmer AR. (2001) A neural code for low-frequency sound localization in mammals. *Nat Neurosci.* 4, 396–401.

Middlebrooks JC. (1992) Narrow-band sound localization related to external ear acoustics. *J Acoust Soc Am.* 61, 2607–2624.

Miranda TT, Pichora-Fuller MK. (2002) Temporally jittered speech produces performance intensity, phonetically balanced rollover in young normal-hearing listeners. *J Am Acad Audiol.* 13, 50–58.

Moore DR, Ferguson MA, Edmondson-Jones M, Ratib S, Riley A. (2010) Nature of auditory processing disorder in children. *Pediatrics.* 126, e382–e390.

Norena AJ, Gourévitch B, Aizawa N, Eggermont JJ. (2006) Spectrally enhanced acoustic environment disrupts frequency representation in cat auditory cortex. *Nat Neurosci.* 9, 932–939.

Patterson RD, Uppenkamp S, Johnsrude IS, Griffiths TD. (2002) The processing of pitch and melody information in auditory cortex. *Neuron.* 36, 767–776.

Phillips DP. (2008) A perceptual architecture for sound lateralization in man. *Hear Res.* 238, 124–132.

Phillips DP. (2012) Time and timing in audition: Some current issues in auditory temporal processing. In: Tremblay KL, Burkard RF, eds. *Translational Perspectives in Auditory Neuroscience. Special Topics.* San Diego, CA: Plural; pp 69–102.

Phillips DP, Brugge JF. (1985) Progress in neurophysiology of sound localization. *Ann Rev Psychol.* 36, 245–274.

Phillips DP, Comeau M, Andrus JN. (2010) Auditory temporal gap detection in children with and without auditory processing disorder. *J Am Acad Audiol.* 21, 404–408.

Phillips DP, Dingle RN, Hall SE, Jang M. (2012a) Dual mechanisms in the perceptual processing of click train temporal regularity. *J Acoust Soc Am.* 132, EL22–EL28.

Phillips DP, Hall SE. (1990) Response timing constraints on the cortical representation of sound time structure. *J Acoust Soc Am.* 88, 1403–1411.

Phillips DP, Hall SE. (1992) Multiplicity of inputs in the afferent path to cat primary auditory cortex neurons revealed by tone-on-tone masking. *Cereb Cortex.* 2, 425–433.

Phillips DP, Hall SE. (2005) Psychophysical evidence for adaptation of central auditory processors for interaural differences in time and level. *Hear Res.* 202, 188–199.

Phillips DP, Quinlan CK, Dingle RN. (2012b) Stability of central binaural sound localization mechanisms in mammals, and the Heffner hypothesis. *Neurosci Biobehav Rev.* 36, 889–900.

Pichora-Fuller MK, Schneider BA, MacDonald E, Pass HE, Brown S. (2007) Temporal jitter disrupts speech intelligibility: A simulation of auditory aging. *Hear Res.* 223, 114–121.

Pienkowski M, Eggermont JJ. (2010) Intermittent exposure with moderate-level sound impairs central auditory function of mature animals without concomitant hearing loss. *Hear Res.* 261, 30–35.

Plack CJ. (2005) *The Sense of Hearing.* Mahwah, NJ: Lawrence Erlbaum.

Rauschecker JP, Romanski LM. (2011) Auditory cortical organization: Evidence for functional systems. In: Winer JA, Schreiner CE, eds. *The Auditory Cortex.* New York, NY: Springer; pp 99–116.

Recanzone GH, Schreiner CE, Merzenich MM. (1993) Plasticity in the frequency representation of primary auditory cortex following discrimination training in adult owl monkeys. *J Neurosci.* 13, 87–103.

Robertson D, Irvine DRF. (1989) Plasticity of frequency organization in auditory cortex of guinea pigs with partial unilateral deafness. *J Comp Neurol.* 282, 456–471.

Ross B, Hillyard SA, Picton TW. (2010) Temporal dynamics of selective attention during dichotic listening. *Cereb Cortex.* 20, 1360–1371.

Sagi Y, Tavor I, Hofstetter S, Tzur-Moryosef S, Blumenfeld-Katzir T, Assaf Y. (2012) Learning in the fast lane: New insights into neuroplasticity. *Neuron.* 73, 1195–1203.

Salminen NH, Tiitinen H, Yrttiaho S, May PJC. (2010) The neural code for interaural time difference in human auditory cortex. *J Acoust Soc Am.* 127, EL60–EL65.

Scharf B, Magnan J, Collet L, Ulmer E, Chays A. (1994) On the role of the olivocochlear bundle in hearing: A case study. *Hear Res.* 75, 11–26.

Semple MN, Aitkin LM. (1981) Integration and segregation of input to the cat inferior colliculus. In: Syka J, Aitkin LM, eds. *Neuronal Mechanisms of Hearing.* New York, NY: Plenum Press; pp 155–161.

Slattery WH III, Middlebrooks JC. (1994) Monaural sound localization: Acute versus chronic unilateral impairment. *Hear Res.* 75, 38–46.

Spitzer MW, Semple MN. (1998) Transformation of binaural response properties in the ascending auditory pathway: Influence of time-varying interaural phase disparity. *J Neurophysiol.* 80, 3062–3076.

Starr A, Picton TW, Sininger Y, Hood LJ, Berlin CI. (1996) Auditory neuropathy. *Brain.* 119, 741–753.

Stecker GC, Harrington IA, Middlebrooks JC. (2005) Location coding by opponent neural populations in the auditory cortex. *PLoS Biol.* 3, 520–528, e78.

Suga N. (2012) Tuning shifts of the auditory system by corticocortical and corticofugal projections and conditioning. *Neurosci Biobehav Rev.* 36, 969–988.

Suga N, Niwa H, Taniguchi I, Margoliash D. (1987) The personalized auditory system of the mustached bat: Adaptation for echolocation. *J Neurophysiol.* 58, 643–654.

Wang X, Merzenich MM, Beitel R, Schreiner CE. (1995) Representation of a species-specific vocalization in the primary auditory cortex of the common marmoset: Temporal and spectral characteristics. *J Neurophysiol.* 74, 2685–2706.

Wang X, Walker KMM. (2012) Neural mechanisms for the abstraction and use of pitch information in auditory cortex. *J Neurosci.* 32, 13339–13342.

Weinberger NM. (1997) Learning-induced receptive field plasticity in the primary auditory cortex. *Semin Hear.* 9, 59–67.

Weinberger NM. (2003) The nucleus basalis and memory codes: Auditory cortical plasticity and the induction of specific, associative behavioral memory. *Neurobiol Learn Mem.* 80, 268–284.

Zeng FG, Kong YY, Michalewski HJ, Starr A. (2005) Perceptual consequences of disrupted auditory nerve activity. *J. Neurophysiol.* 93, 3050–3063.

CHAPTER 28

Auditory Pathway Representations of Speech Sounds in Humans

Daniel A. Abrams and Nina Kraus

INTRODUCTION

An essential function of the human auditory system is the neural encoding of speech sounds. The ability of the brain to translate the acoustic events in the speech signal into meaningful linguistic constructs relies in part on the way the central nervous system represents the acoustic structure of speech. Consequently, an understanding of how the nervous system accomplishes this task would provide important insights into the basis of language function and auditory-based cognition.

One of the challenges faced by researchers is that speech is a complex acoustic signal that is rich in both spectral and temporal features. In everyday listening situations, the abundance of acoustical cues in the speech signal provides enormous perceptual benefits to listeners. For example, listeners are able to shift their attention between different acoustical cues when perceiving speech from different talkers to compensate for the built-in variations in the acoustical properties (Nusbaum and Morin, 1992). This form of "listener flexibility" reflects a critical aspect of speech perception: The listener makes use of whatever spectral or temporal cues are available to help decode the incoming speech signal.

There are two basic approaches that researchers have adopted for conducting experiments on speech perception and the underlying physiology. One approach uses "simple" acoustic stimuli, such as tones and clicks, as a means to control for the complexity of the speech signal. Whereas simple stimuli enable researchers to reduce the acoustics of speech to its most basic elements, the auditory system is nonlinear (Sachs and Young, 1979) and, therefore, responses to simple stimuli generally do not accurately predict responses to actual speech sounds. A second approach uses speech and speech-like stimuli (Song et al., 2006; Cunningham et al., 2002; Skoe and Kraus, 2010). There are many advantages to this approach. First, these stimuli have greater face validity for understanding speech processing. Second, a complete description of how the nonlinear auditory system responds to speech can only be obtained by using speech stimuli. Third, long-term exposure to speech sounds and their use linguistically produces plastic changes in the auditory pathways that may alter neural representation of speech in a manner that cannot be predicted by simple stimuli. Fourth, when speech stimuli are chosen carefully, the acoustic properties of the signal can still be well controlled.

This chapter is organized into five sections, with each section describing what is currently known about how the brain represents a particular acoustic feature present in speech (see Table 28.1). These acoustic features of speech were chosen because they have essential roles in normal speech perception. Each section contains a description of the acoustical feature, an explanation of its importance in speech perception, followed by a review and assessment of the data for that acoustic feature.

An exciting aspect of brain function is the remarkable capacity of the brain to modify its functional properties following training. In the auditory domain, a growing body of research has shown that targeted training and remediation programs can provide substantial speech perception benefit to a number of populations, including both normal listeners and clinical populations with impaired auditory function. Given the prevalence of hearing deficits in industrialized societies and an aging population in most Western countries, targeted auditory training to maintain and improve speech perception, particularly in the presence of background noise, represents an important strategy for sustaining speech-based communication and cognitive skills (Lin et al., 2013). Importantly, behavioral improvements that result from training originate in changes in brain function, and it is of great interest to the field of auditory research to understand what aspects of brain function change in response to auditory-based training. These findings are of theoretical interest: Many auditory training paradigms constitute relatively complex tasks, exposing the listener to a host of acoustical features and tapping into a range of sensory and cognitive skills; therefore, an understanding of the specific brain changes that accompany training-based improvement provides a window on the particular acoustical features that are most important for improvement on the trained tasks. Thus, a final goal of this chapter is to highlight exciting recent research describing

TABLE 28.1

Acoustic Features of Speech and their Representations in the Central Auditory System

Major Sections: Acoustic Features in Speech	Feature's Role in the Speech Signal	Brainstem Measure	Cortical Measure
1. Formant structure	Ubiquitous in vowels, approximants, and nasals; essential for vowel perception	Frequency-following response	N100m source location; STS activity (fMRI)
2. Periodicity	Temporal cue for the fundamental frequency and low formant frequencies (50–500 Hz)	Frequency-following response	N100m source location and amplitude; nonprimary auditory cortex activity patterns (fMRI)
3. Frequency transitions	Consonant identification; signal the presence of diphthongs and glides; linguistic pitch	Frequency-following response	Left versus right STG activity (fMRI)
4. Acoustic onsets	Phoneme identification	ABR onset complex	N100m source location; N100 latency
5. Speech envelope	Syllable and low-frequency (<50 Hz) patterns in speech	N/A	N100m phase-locking

changes in auditory brain function following speech and auditory training, with a focus on therapeutic training paradigms designed to improve speech perception in both clinical populations and normal hearing listeners.

An important consideration is that the acoustical features described in this chapter are not mutually exclusive. For example, one section of this chapter describes the neural encoding of "periodicity," which refers to acoustical events that occur at regular time intervals. Many features in the speech signal are periodic; however, describing all of these simultaneously occurring periodic features would be experimentally unwieldy. For simplicity, and to show how these features were investigated, some related acoustical features will be discussed in separate sections. Throughout the chapter we have tried to identify when there is overlap among the acoustical features.

THE SIGNAL: BASIC SPEECH ACOUSTICS

The speech signal can be described according to a number of basic physical attributes (Johnson, 1997). An understanding of these characteristics is essential to any discussion of how the auditory system encodes speech. The linguistic roles of these acoustic features are described separately within each section of the chapter.

Fundamental Frequency

The fundamental frequency component of speech results from the periodic beating of the vocal folds. In Figure 28.1A, the frequency content of the naturally produced speech sentence "The Young Boy Left Home" is plotted as a function of time: Greater amounts of energy at a given frequency are represented with dark lines whereas smaller amounts of energy are depicted in white. The fundamental frequency can be seen as the horizontal band of energy in Figure 28.1A that is closest to the *x*-axis (i.e., lowest in frequency). The fundamental frequency is labeled F0 and provides the perceived pitch of an individual's voice.

Harmonic Structure

An acoustical feature that is related to the fundamental frequency of speech is known as the harmonic structure. Speech harmonics, which are integer multiples of the fundamental frequency, are present in ongoing speech. The harmonic structure of speech is displayed in Figure 28.1A, as the regularly spaced horizontal bands of energy that are seen throughout the sentence.

Formant Structure

Another essential acoustical feature of speech is the formant structure which describes a series of discrete peaks in the frequency spectrum of speech that are the result of an interaction between the frequency of the vocal-fold vibrations and the speaker's vocal tract resonance. The frequency of these peaks, as well as the relative frequency between peaks, varies for different speech sounds. The formant structure of speech depends on the harmonic structure of speech. Harmonic structure is represented by integer multiples of the fundamental frequency, and formants are harmonics that are close to a resonant frequency of the vocal tract. In Figure 28.1, the formant structure of speech is represented by the series of horizontal, and occasionally diagonal, lines that

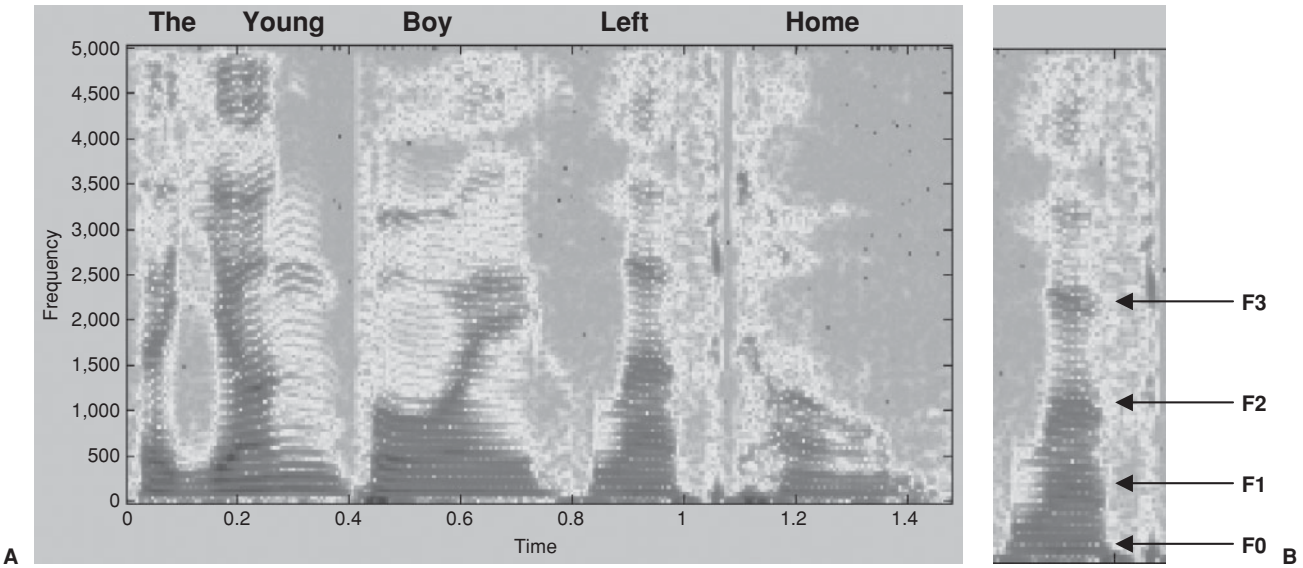


FIGURE 28.1 Spectrogram for the naturally produced speech sentence "The young boy left home." [**A**] The complete sentence; [**B**] the word "left" is enlarged to illustrate the frequency structure: The fundamental frequency (F0) and formants (F1–F3) are represented in the spectrogram by *broad dark lines* of energy.

are darker than their neighbors that run through most of the speech utterance. The word "left" has been enlarged in Figure 28.1B to better illustrate this phenomenon. The broad and dark patches seen in this figure represent the peaks in the frequency spectrum of speech that are the result of an interaction between the frequency of vibration of the vocal folds and the resonances of a speaker's vocal tract. The frequency of these peaks, as well as the relative frequency between peaks, varies for different speech sounds within the sentence. The lowest frequency formant is known as the first formant and is notated F1, whereas subsequent formants are notated F2, F3, and so on. The frequencies of F1 and F2 in particular are important for vowel identity.

THE MEASURES OF BRAIN ACTIVITY

We begin by describing the neurophysiological measures that have been used to probe auditory responses to speech and speech-like stimuli (comprehensive descriptions of these measures can be found elsewhere: Hall, 1992 as well as in chapters in this book). Historically, the basic research on the neurophysiology of speech perception has borrowed a number of clinical tools to assess auditory system function.

Brainstem Responses

The auditory brainstem response (ABR) consists of small voltages originating from neural activity in auditory structures in the brainstem in response to sound. Although these responses do not pinpoint the specific origin of auditory activity among the auditory brainstem nuclei, the great strength of ABRs (and auditory potentials in general) is that they precisely reflect the time-course of neural activity at the microsecond level. The ABR is typically measured with a single active electrode referenced to the earlobe or nose. Clinical evaluations using the ABR typically use brief acoustic stimuli, such as clicks and tones, to elicit brainstem activity. The ABR is unique among the AEPs because of the remarkable reliability of this response, both within and across subjects. In the clinic, the ABR is used to assess the integrity of the auditory periphery and lower brainstem (Hall, 1992). The response consists of a number of peaks, with wave V being the most clinically reliable. Deviations on the order of microseconds are deemed "abnormal" in the clinic and are associated with some form of peripheral hearing damage or with retrocochlear pathologies. Research using the ABR to probe acoustic processing of speech utilizes similar recording procedures, but different acoustic stimuli.

Cortical Responses

Cortical-Evoked Potentials and Fields

Cortical-evoked responses are used as a research tool to probe auditory function in normal and clinical populations. Cortical-evoked potentials are small voltages originating from neural activity auditory cortical structures in response to sound. These potentials are typically measured with multiple electrodes, often referenced to a "common reference," which is the average response measured across all electrodes. Cortical-evoked "fields" are the magnetic counterpart to cortical-evoked potentials; however, instead of measuring voltage across the scalp, magnetic fields produced by brain activity are measured.

Electroencephalography (EEG) is the technique by which evoked potentials are measured and magnetoencephalography (MEG) is the technique by which evoked fields are measured. Similar to the ABR, the strength of assessing cortical-evoked potentials and fields is that they provide detailed information about the time-course of activation and how sound is encoded by temporal response properties of large populations of auditory neurons, though this technique is limited in its spatial resolution. Because of large inter- and intrasubject variability in cortical responses, these measures are not generally used clinically. Results from these two cortical methodologies are generally compatible, despite some differences in the neural generators that contribute to each of these responses. Studies using both EEG and MEG are described interchangeably throughout this chapter despite the subtle differences between the measures. The nomenclature of waveform peaks is similar for EEG and MEG: Typically, an N or P, depicting a negative or positive deflection, followed by a number indicating the approximate latency of the peak. Finally, the letter "m" follows the latency for MEG results. For example, N100 and N100m are the labels for a negative deflection at 100 ms as measured by EEG and MEG, respectively.

Functional Imaging

Functional imaging of the auditory system is another often-used technique to quantify auditory activity in the brain. The technology that is used to measure these responses, as well as the results they yield, is considerably different from the previously described techniques. The primary difference is that functional imaging is an indirect measure of neural activity, that is, instead of measuring voltages or fields resulting from activity in auditory neurons, functional imaging measures hemodynamics, a term used to describe changes in metabolism as a result of changes in brain activity. The data produced by these measures is a three-dimensional map of activity within the brain as a result of a given stimulus. The strong correlation between actual neural activity and blood flow to the same areas of the brain (Smith et al., 2002) has made functional imaging a valuable investigative tool to measure auditory activity in the brain. The two methods of functional imaging described here are functional magnetic resonance imaging (fMRI) and positron emission tomography (PET). The difference between these two techniques is that fMRI measures natural levels of oxygen in the brain, as oxygen is consumed by neurons when they become active. PET, however, requires the injection of a radioactive isotope into a subject. The isotope emits positrons, which can be detected by a scanner, as it circulates in the subject's bloodstream. Increases in neural activity draw more blood, and consequently more of the radioactive isotope, to a given region of the brain. The main advantage that functional imaging offers relative to evoked potentials and evoked fields is that it provides extremely accurate and precise spatial information regarding the origin of neural activity in the brain. A disadvantage is the poor resolution in the temporal domain: Neural activity is often integrated over the course of seconds, which is considered extremely slow given that speech tokens are as brief as 30 ms. Although recent work using functional imaging has begun describing activity in subcortical regions, the work described here will cover only studies of temporal cortex.

ACOUSTIC FEATURES OF SPEECH

Periodicity

Definition and Role in the Perception of Speech

Periodicity refers to regular temporal fluctuations in the speech signal between 50 to 500 Hz (Rosen, 1992). Important aspects of the speech signal that contain periodic acoustic information include the fundamental frequency and all components of the formant structure (note that encoding of the formant structure of speech is covered in a later section). The acoustic information provided by periodicity conveys both phonetic information as well as prosodic cues, such as intonation and stress, in the speech signal. As stated in Rosen's paper, this category of temporal information represents both the periodic features in speech and the distinction between periodic and aperiodic portions of the signal, which fluctuate at much faster rates.

This section will review studies describing the neural representation of relatively stationary periodic components in the speech signal, most notably the fundamental frequency. An understanding of the mechanism for encoding a simple periodic feature of the speech signal, the F0, will facilitate descriptions of complex periodic features of the speech signal, such as the formant structure and frequency modulations.

Physiological Representation of Periodicity in the Human Brain

Auditory Brainstem

The short-latency frequency-following response (FFR) is an electrophysiological measure of phase-locked neural activity originating from brainstem nuclei that represents responses to periodic acoustic stimuli up to approximately 1,000 Hz (Smith et al., 1975; Stillman et al., 1978). Based on the frequency range that can be measured with the FFR, a representation of the fundamental frequency can be measured using this methodology (Krishnan et al., 2004; Russo et al., 2004; Skoe and Kraus, 2010), as well as the F1 in some instances (encoding of F1 is discussed in detail in the Formant Structure section).

A number of studies have shown that F0 is represented within the brainstem response (i.e., FFR) according to a

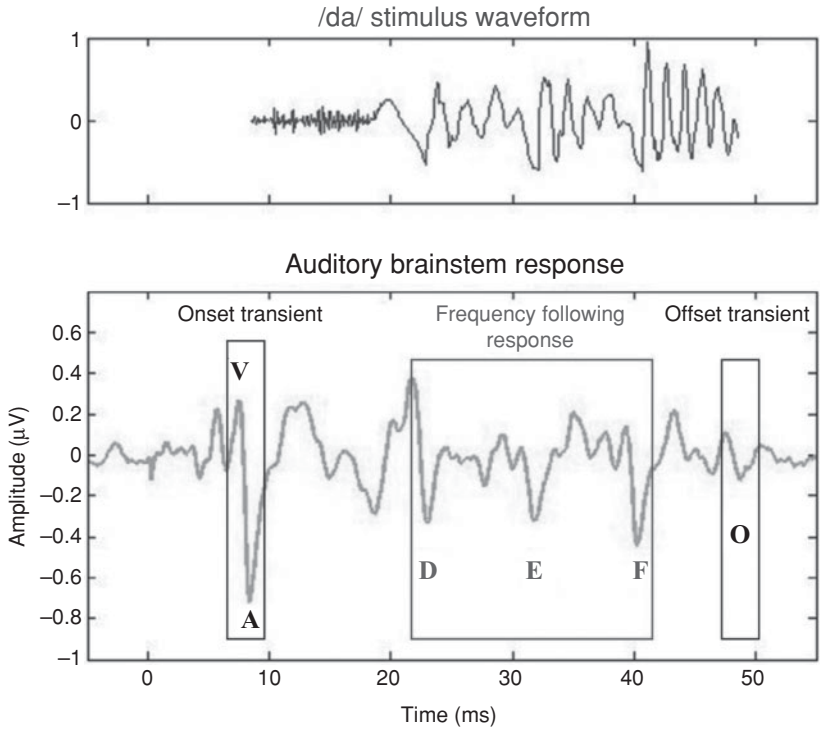


FIGURE 28.2 Acoustic waveform of the synthesized speech stimulus /da/ [*above*] and grand average auditory brainstem responses to /da/ [*below*]. The stimulus has been moved forward in time to the latency of onset responses [peak V] to enable direct comparisons with brainstem responses. Peaks V and A reflect the onset of the speech sound and peak O reflects stimulus offset. Peaks D, E, and F represent a phase-locked representation to the fundamental frequency of the speech stimulus, and the peaks between D, E, and F occur at the F1 frequency.

series of peaks that are temporally spaced corresponding to the wavelength of the fundamental frequency. An example of F0 representation in the FFR can be seen in Figure 28.2, which shows the waveform of the speech stimulus /da/ (top), an experimental stimulus that has been studied in great detail, as well as the brainstem response to this speech sound (bottom). A cursory inspection of this figure shows that the primary periodic features of the speech waveform provided by the F0 are clearly represented in negative-going peaks D, E, and F of the FFR brainstem response. Importantly, it has been shown that the FFR is highly sensitive to F0 frequency; this aspect of the brainstem response accurately "tracks" modulations in frequency (Krishnan et al., 2004), a topic which is discussed in depth in the Frequency Transitions section of this chapter.

A hypothesis regarding the brainstem's encoding of different aspects of the speech signal has been proposed (Kraus and Nicol, 2005). Specifically, it is proposed that the source (referring to vocal-fold vibration) and filter aspects (vocal musculature in the production of speech) of a speech signal show dissociation in their acoustical representation in the auditory brainstem. The source portion of the brainstem's response to speech is the representation of the F0, whereas the filter refers to all other features, including speech onset, offset, and the representation of formant frequencies. For example, it has been demonstrated that brainstem responses are correlated within source and filter classes but are not correlated between classes (Russo et al., 2004). Moreover, in a study of children with language-learning disabilities, whose behavioral deficits may be attributable to central auditory processing disorders, it has been shown that source representation in the auditory brainstem is normal whereas filter class representation is impaired (Banai et al., 2009; Hornickel et al., 2012a; King et al., 2002). The converse, impairments in brainstem encoding of source (F0) but not filter components, is a characteristic of individuals with poor hearing in noise (Anderson et al., 2011). These data suggest that the acoustical representations of source and filter aspects of a given speech signal are differentially processed and provide evidence for neural specialization at the level of the brainstem.

Cortex

Neurons in the auditory cortex respond robustly with time-locked responses to slow rates of stimulation (<~25 Hz) and generally do not phase-lock to frequencies greater than approximately 100 Hz (Creutzfeldt et al., 1980). Therefore, cortical phase-locking to the fundamental frequency of speech, which is near or greater than 100 Hz, is poor, and it is generally thought that the brainstem's phase-locked representation of F0 is transformed at the level of cortex to a more abstract representation. For example, it has been shown that cortical neurons produce sustained, nonsynchronized discharges throughout a high-frequency (>50 Hz) stimulus (Lu et al., 2001), which is a more abstract representation of the stimulus frequency compared to time-locked neural activation.

An important aspect of F0 perception is that listeners native to a particular language are able to perceive a given speech sound as invariant regardless of the speaker's F0, which varies considerably among men (F0 ~ 100 Hz), women (F0 ~ 200 Hz), and children (F0 up to 400 Hz). For

example, the speech sound "dog" is categorized by a listener to mean the exact same thing regardless of whether an adult or a child produces the vocalization, even though there is a considerable difference in the F0 of the adult's and child's vocalizations. To address how auditory cortical responses reflect relatively large variations in F0 between listeners, N100m cortical responses were measured with MEG for a set of Finnish vowel and vowel-like stimuli that varied in F0 while keeping all other formant information (F1–F4) constant (Makela et al., 2002). Results indicated that N100m responses were extremely similar in spatial activation pattern and amplitude for all vowel and vowel-like stimuli, irrespective of the F0. This is a particularly intriguing finding given that N100m responses differed when 100-, 200-, and 400-Hz puretone stimuli were presented to the same subjects in a control condition. The similarity of the speech-evoked brain responses, which were independent of the F0 frequency, suggests that variances in F0 may be filtered out of the neural representation by the time it reaches the cortex. The authors suggest that the insensitivity of cortical responses to variations in the F0 may facilitate the semantic categorization of the speech sound. In other words, since F0 does not provide essential acoustic information relevant to the semantic meaning of the speech sound, it may be that the cortex does not respond to this aspect of the stimulus in favor of other acoustic features that are essential for decoding word meaning.

Electrophysiological Changes due to Training

The brain's representation of periodicity has been shown to be malleable following auditory-based training. The goal of one study was to train the perception of speech in the presence of background noise, an environmental sound source which negatively impacts speech perception in normal individuals and has even more severe perceptual consequences in individuals with hearing impairments. In this study, a group of 28 normal hearing young adults were trained on a commercially available computer program entitled "Listening and Communication Enhancement" (LACE) (Sweetow and Sabes, 2006), which trains listeners on a number of auditory tasks including comprehension of degraded speech, auditory mnemonic and cognitive skills, and communication strategies (Song et al., 2012). After 4 weeks of training, participants showed improvements in measures of speech perception in noise as measured by LACE as well as independent measures of speech perception in noise, including the Hearing in Noise Test (Nilsson et al., 1994) and the Quick Speech in Noise Test (Killion et al., 2004). An age-matched group of normal hearing, untrained listeners showed no improvements in speech in noise perception.

Neural correlates of these behavioral improvements were explored by measuring ABRs to a synthetic /da/ stimulus in both quiet and in the presence of background noise. Results showed that behavioral improvements in trained listeners were accompanied by enhanced brainstem representation of periodicity, as measured by the spectral magnitude of the F0 and the second harmonic (H2), in responses measured in the presence of background noise. An important consideration is the breadth of auditory and cognitive skills trained by LACE and the specificity of these brainstem results. The LACE program broadly trains speech perception in noise, and consequently the brainstem representation of any number of acoustical features in speech could have shown training-related effects. Nevertheless, only the F0 and H2 features of the brainstem response were enhanced following LACE training. The interpretation of this result is that the brain's coding of periodicity is a particularly critical element for the perception of speech in noise. On the surface, this may be surprising: The fundamental frequency is not always necessary for speech comprehension. For example, the fundamental frequency is systematically filtered out of all telephone signals. Nevertheless, these results strongly suggest that in challenging listening conditions, including the perception of speech in noise, periodic features may provide important acoustical benefit to the listener as reflected by the sharpening of this feature in the brainstem response to speech in noise.

In summary, periodicity of the fundamental frequency is robustly represented in the FFR of the ABR. Moreover, the representation of the fundamental frequency is normal in children with learning disabilities (LDs) despite the abnormal representations of speech-sound onset and first formant frequency. Yet, its role appears to be essential in hearing speech in noise. This disparity in the learning disabled auditory system provides evidence that different features of speech sounds may be served by different neural mechanisms and/or populations. In the cortex, MEG results show that cortical responses are relatively insensitive to changes in the fundamental frequency of speech sounds, suggesting that differing F0s between speakers are filtered out by the time the signal reaches the level of auditory cortex. Results from speech in noise training indicate that improvements in speech perception in noise result in systematic enhancement of periodic aspects of the speech signal, including the F0 and H2 components.

Formant Structure

Role in the Perception of Speech

Formant structure describes a series of discrete peaks in the frequency spectrum of speech that are the result of an interaction between the frequency of vibration of the vocal folds and the resonances within a speaker's vocal tract (see Introduction for a more complete acoustical description of the formant structure). The formant structure is a dominant acoustic feature of sonorants, a class of speech sounds that includes vowels, approximants (e.g., /l/ and /ɹ/), and nasals. The formant structure has a special role in the perception of vowels in that formant frequencies, particularly

the relationship between F1 and F2 are the primary phonetic determinants of vowels. For example, the essential acoustic difference between /u/ and /i/ is a positive shift in F2 frequency (Peterson and Barney, 1952). Because of the special role of formants for vowel perception, much of the research regarding the formant structure of speech uses vowel stimuli.

Physiological Representation of Formant Structure in the Human Brain

Auditory Brainstem

The question of how the human auditory brainstem represents important components of the formant structure was addressed in a study by Krishnan (2002). In this study, brainstem responses (FFRs) to three steady-state vowels were measured and the spectral content of the responses was compared to that of the vowel stimuli. All three of the stimuli had approximately the same fundamental frequency; however, the first two formant frequencies were different in each of the vowel stimuli. Results indicate that at higher stimulus intensities the brainstem FFR accurately represents F1 and F2; however, the representation of F1 was greater than for F2. The author indicates the similarity between this finding and a similar result in a classic study of vowel representation in the auditory nerve of anesthetized cats (Sachs and Young, 1979) which also demonstrated a predominant representation of F1. These data provide evidence that phase-locking serves as a mechanism for encoding critical components of the formant structure not only in the auditory nerve, but also in the auditory brainstem.

Auditory Cortex

A number of studies have described the representation of formant structure in the human cortex as a means of investigating whether a cortical map of phonemes, termed the "phonemotopic" map, exists in the human brain. Specifically, researchers want to know if the phonemotopic map is independent of the tonotopic map, or alternatively whether phonemes are more simply represented according to their frequency content along the tonotopic gradient in auditory cortex. To this end, investigators have measured cortical responses to vowel stimuli, a class of speech sounds that differ acoustically from one another according to the distribution of F1–F2 formant frequencies. Vowel stimuli also offer the advantage of exhibiting no temporal structure beyond the periodicity of the formants.

The method that has been used to investigate the relationship between the tonotopic map in human auditory cortex and the representation of formant structure has been to compare cortical source locations for tones and specific speech sounds with similar frequency components. For example, in one study (Diesch and Luce, 1997) N100m source location was measured in response to separately presented 600- and 2,100-Hz puretones as well as a two-tone composite signal comprising the component puretones (i.e., simultaneous presentation of the 600- and 2,100-Hz puretones). These responses were compared to isolated formants, defined as the first and second formant frequencies of a vowel stimulus, complete with their harmonic structure, separated from the rest of the frequency components of the stimulus (i.e., F0, higher formant frequencies). These isolated formants had the same frequency as the tonal stimuli (i.e., 600 and 2,100 Hz). Finally, a two-formant composite signal, which constituted a vowel, was also presented. Results indicated that the N100m source in response to the vowel stimulus was different in location from that predicted by both the puretone responses and the superposition of responses to the component single formant stimuli. These data indicate that formant structure is spatially represented in human cortex differently than the linear sum of responses to the component formant stimuli and suggest that formant structure has a different representation relative to the tonotopic map. The authors of this work hypothesize that the different spatial representation of the vowel stimuli reflects the additional acoustic components of the vowel stimuli, including the harmonic and formant structures. The authors of this work refrain from a potentially more intriguing conclusion, that is, does the spatial representation of the vowel stimuli in some way reflect the behavioral experience of the subjects with these speech sounds? For example, it is possible that a larger, or different, population of cortical neurons is recruited for sounds that are familiar, or have significant ecologic importance, relative to the population recruited for puretones or single formant frequencies and that the source location for the vowels reflects this phenomenon.

Additional studies have attempted to better describe the acoustic representation of vowels in the human brain. In one study, Obleser et al. (2003) addressed the neurophysiology underlying a classic study of speech acoustics in which it was shown that the distinction of vowels is largely carried by the frequency relationship of F1 and F2 (Peterson and Barney, 1952). To this end, cortical source locations were measured in response to German vowels that naturally differ in F1–F2 relationships. Results indicated that the location of the N100m source reflects the relationship of the F1–F2 formant frequencies. This finding was replicated in a second study using 450 natural speech exemplars of three Russian vowels; again, the spectral distance between F1 and F2 was reflected in the dipole location of N100m responses (Shestakova et al., 2004).

Although these studies provide evidence that the cortex represents the formant structure of vowels in a manner that is (a) unrelated to the tonotopic map and (b) organized according to the perceptually essential formant frequencies, these findings require a number of caveats. First, the source locations described in these studies represent the center of gravity, as a single point in three-dimensional space in the cortex, of the neural contributors to a given N100m response (Naatanen and Picton, 1987). Second, approximately six

neural regions contribute to the N100 and therefore it represents a highly complex neural response. Consequently, the N100 described in these studies of phonemotopic maps should not be viewed as an exact representation of well-described, and highly localized, auditory maps in animal models (Schreiner, 1998). This is particularly relevant given that the clear tonotopic gradient in auditory cortex is no longer apparent when puretone stimuli are presented above 50 dB SPL (Schreiner, 1998), such as the levels used in the MEG experiments described in this section. In addition, it has not yet been definitively shown that the neural representations of phonemes described in these studies truly constitute a phonemotopic map. The presence of a phonemotopic map suggests behavioral relevance of phoneme stimuli beyond their acoustic attributes. None of the studies described here have tested if cortical responses to the F1–F2 components for nonnative vowel sounds show similar sensitivity as native phonemes. Despite these limitations, these studies provide consistent evidence that a perceptually critical aspect of the formant structure of vowels, the F1–F2 relationship, is represented in a spatial map in auditory cortex as early as ~100 ms poststimulus onset.

Another line of evidence has used functional imaging to show the particular regions of the temporal cortex that are sensitive to the formant structure of speech sounds relative to other natural and vocally generated sounds, that is, laughs and coughs (Belin et al., 2000). Cortical responses to natural vocal stimuli were compared to vocal stimuli in which the formant structure of speech was replaced by white noise and scrambled vocal sounds. All stimuli were matched for overall RMS energy. In both of these experimental conditions, the original amplitude envelope of the speech signal modulated the altered spectral information. Results from this experiment indicated that all stimuli activated regions along the superior temporal sulcus (STS), a cortical region consisting of unimodal auditory and multimodal areas that is hypothesized to be a critical speech-processing center subsequent to more rudimentary acoustic processing in structures of the superior temporal plane. However, responses to the natural vocal stimuli were significantly larger and more widespread throughout the STS, particularly in the right hemisphere, than for the spectrally manipulated vocal stimuli. These data indicate that the formant structure of speech deeply affects activity patterns in the STS, a speech-selective region of temporal cortex, even when the temporal components of the signals are held constant. In addition, these data suggest a right-hemisphere bias for processing the formant structure, which supports the more general hypothesis that the right hemisphere is dominant for resolving spectral components in acoustic signals (Zatorre et al., 2002).

An interesting consideration is how cortical asymmetries in response to the acoustic features of speech relate to well-established cerebral asymmetries for higher-order language processing, such as phonemic and semantic processing (Geschwind and Galaburda, 1985), which are strongly lateralized to the left hemisphere. Although a direct link between these forms of asymmetry has not been established, a plausible scenario is that the acoustic-level asymmetries precede, and serve as the input to, phonemic and semantic processing in left-hemisphere language regions. If this is the case, it remains to be seen what physiological advantage a right-hemisphere preference for formant structure processing (Belin et al., 2000) might offer given that phonemic and semantic processing of speech stimuli takes place in the opposite hemisphere, thereby requiring transmission through the corpus callosum. Future studies investigating acoustic-level asymmetries and their interface with higher-order language asymmetries would provide essential information regarding the functional neuroanatomy of speech perception.

Electrophysiological Changes due to Training

Musical training can enhance the brainstem's representation of formant frequencies, and this enhancement is related to important aspects of speech perception. For example, it was recently shown that adult musicians have greater differentiation of brainstem responses for consonant–vowel stimuli that vary according to F2 frequency (Parbery-Clark et al., 2012; Strait et al., in press). Specifically, musicians showed more pronounced brainstem timing differences in response to /da/, /ga/, and /ba/ stimuli compared to nonmusicians, and brainstem differentiation of these stimuli correlated with standardized measures of speech perception in noise. This finding is important for a number of reasons. First, it shows that musicians' goal-directed attention to spectrotemporal features in music promotes neural differentiation of subtle variants in formant structure in speech as well as perceptual benefits for speech in noise. This result is also significant with regard to efficacy of therapy: Whereas many forms of auditory perceptual training fail to generalize to untrained stimuli (Burk and Humes, 2008; Halliday et al., 2012), results from the music literature have consistently shown that musical training generalizes to speech perception tasks in children (Moreno et al., 2009; Thompson et al., 2004) and adults (Schon et al., 2004; Thompson et al., 2004) as well as the neural encoding of speech (Moreno et al., 2009; Schon et al., 2004; Strait and Kraus, 2014). Importantly, results from the Parbery-Clark study show that musical training influences the neural differentiation of subtle formant frequency characteristics, which is fundamental to the identification and discrimination of phoneme contrasts (Peterson and Barney, 1952).

In summary, the brainstem encodes lower formant frequencies, which are critical to vowel perception, with phase-locked responses. Moreover, the representation of these formants is enhanced following long-term musical training, and the strength of these representations is correlated with perceptual benefit for speech in noise. Converging evidence indicates that the cortex encodes a perceptually essential aspect of the formant structure of speech. Specifically, the F1–F2 relationship is spatially mapped in the cortex at ~100 ms poststimulus onset as measured by N100m source

location. In addition, functional imaging data provide evidence that the STS, a nonprimary auditory region of temporal cortex, is more responsive to speech stimuli that contain formant structure than speech in which the formant structure has been replaced with other sounds. Together, these results suggest that both primary and associative regions of temporal cortex are sensitive to aspects of the formant structure that are essential for normal perception.

Frequency Transitions

Acoustic Description and Role in the Perception of Speech

Frequency transitions of the fundamental and formant frequencies are ubiquitous in ongoing speech. In English, modulation of the fundamental frequency typically does not provide segmental cues; rather it provides suprasegmental cues such as the intent (e.g., question or statement) and emotional state of the speaker. In other languages, such as Mandarin and Thai, modulations to the fundamental frequency provide phonetic cues. Formant transitions on the other hand are critical for speech perception of English in that they serve as a cue for consonant identification and signal the presence of diphthongs and glides (Lehiste and Peterson, 1961). Formant transitions have also been shown to play a role in vowel identification (Nearey and Assmann, 1986). The movements of formant frequencies can be distilled to three basic forms that occur during an ongoing sequence of phonemes (taken from Lehiste and Peterson, 1961): (a) The movement of a formant from the initiation of the consonant until the beginning of the vowel in a consonant–vowel combination, (b) the movement of a formant from one vowel to another vowel (i.e., in a diphthong), and (c) formant movement from a vowel until vowel termination for a vowel–consonant combination. The frequency modulations that occur during formant transitions can occur at relatively fast rates (~40 ms) while spanning large frequency ranges (>2,000 Hz in F2 transitions).

Physiological Representation of Frequency Transitions in the Human Brain

Auditory Brainstem

The FFR is able to "track," or follow, frequency changes in speech. This phenomenon was demonstrated in a study of FFR tracking of the fundamental frequency (F0) in Mandarin speech sounds (Krishnan et al., 2004). In this study, FFR to four different tonal permutations of the Mandarin word "yi" was measured in a group of native Mandarin speakers. Specifically, synthetic stimuli consisted of "yi" pronounced with (1) a flat F0 contour, (2) a rising F0 contour, (3) a falling F0 contour, and (4) a concave F0 contour that fell then rose in frequency. In Mandarin, which is a "tonal" language, these four stimuli are different words: The F0 contour provides the only acoustic cue to differentiate them. Results indicated that the FFR represented the fundamental frequency modulations for all of the stimulus conditions irrespective of the form of the frequency contour. These data indicate that the FFR represents phase-locked activity in the brainstem for rapidly changing frequency components in speech, an essential acoustical cue for consonant identification.

A similar methodology was used in another study by Krishnan and colleagues to investigate the role of language experience on auditory brainstem encoding of pitch (Krishnan et al., 2005). FFRs to the "yi" stimuli described above were measured in native Mandarin speakers as well as native speakers of American English, to whom the pitch alterations bear no linguistic value. Results from this study indicate greater FFR pitch strength and pitch tracking in the Chinese subjects compared to the native English speakers across all four of the Mandarin tones. The FFR of the Chinese subjects also indicated increased harmonic representation of the fundamental frequency (i.e., larger neural representation of the harmonic content of the F0) compared to the English speakers. These data indicate that responses from the auditory brainstem reflect the behavioral experience of a listener by enhancing the neural representation of linguistically relevant acoustic features.

An hypothesis proposed by Ahissar and Hochstein (2004) may explain how experience engenders plasticity at low levels of sensory systems. Their "reverse hierarchy" theory proposes that when a naïve subject attempts to perform a perceptual task, the performance on that task is governed by the "top" of a sensory hierarchy. As this "top" level of the system masters performance of the task, over time, lower levels of the system are modified and refined to provide more precise encoding of sensory information. This can be thought of as efferent pathway-mediated tuning of afferent sensory input. Although the reverse hierarchy theory does not explicitly discuss plasticity of the brainstem, this theory could account for the findings of Krishnan. Specifically, because of the importance of extracting lexical information present in pitch contours, native Mandarin speakers are "experts" at encoding this acoustic feature, which is accomplished, at least in part, by extreme precision and robustness of sensory encoding in low levels of the auditory system such as the brainstem. Native English speakers, who are not required to extract lexical meaning from pitch contours, are relative novices at this form of pitch tracking, and consequently their brainstems have not required this level of modification.

An interesting question that was addressed in a subsequent study is whether native Mandarin speakers are better than English speakers at pitch tracking the F0 exclusively for familiar speech sounds or whether Mandarin speakers' superior performance would extend to all periodic acoustic signals, including nonnative speech sounds (Xu et al., 2006). Results show that a lifetime of experience using F0 to extract

linguistic meaning specifically affects auditory responses to familiar speech sounds and does not generalize to all periodic acoustic signals. However, data from the Kraus Lab suggests that another form of long-term auditory experience, musicianship, contributes to enhanced neural encoding of speech sounds in the auditory brainstem relative to nonmusicians (Wong et al., 2007). This finding provides evidence that expertise associated with one type of acoustic signal (i.e., music) can provide a general augmentation of the auditory system that is manifested in brain responses to another type of acoustic signal (i.e., speech) and indicates that auditory experience can modify basic sensory encoding.

Auditory Cortex

Similar to Krishnan's work involving the brainstem, multiple studies have investigated cortical processing of F0 pitch contours and its relationship to language experience. The most convincing of these studies is that by Wong et al. (2004). In this study, native Mandarin and native English speakers underwent PET scanning during passive listening and while performing a pitch discrimination task. Stimuli consisted of (a) Mandarin speech sounds that contained modulations of the fundamental frequency that signal lexical meaning and (b) English speech sounds which also contained modulations to the fundamental frequency; however, F0 modulations never provide lexical information in English. Imaging results indicated that native Mandarin speakers showed significant activation of the left anterior insular cortex, adjacent to Broca's area, only when discriminating Mandarin speech sounds; the homologous right anterior insula was activated when this group discriminated English speech sounds, as well as when native English speakers discriminated both Mandarin and English speech sounds. These data suggest that the left anterior insula is involved in auditory processing of modulations to the fundamental frequency only when those modulations are associated with lexical processing. Moreover, these data suggest that the neural processing of acoustic signals is context dependent and is not solely based on the acoustical attributes of the stimuli.

In addition to studies of the neural representation of F0 modulations, a number of studies have also addressed the cortical representation of formant frequency modulation in humans. As it is known that neurons in auditory cortex do not phase-lock to frequencies greater than approximately 100 Hz (Creutzfeldt et al., 1980), and the formant structure of speech consists of frequencies almost exclusively above 100 Hz, the cortical representation of frequency modulation as measured by evoked potentials is abstract (i.e., not represented with time-locked responses) relative to those described for the auditory brainstem. One cortical mechanism that has received considerable attention for the processing of rapid formant modulations is that of asymmetric processing in the left-hemisphere auditory cortex. A more general hypothesis proposes that left-hemisphere auditory cortex is specialized for all forms of rapid acoustic stimuli and serves as an early acoustic analysis stage at the level of the cortex. A significant piece of evidence in support of this hypothesis was provided in a study of cortical activation patterns for rapid and slow formant frequency modulations (Belin et al., 1998). In this study, nonspeech sounds containing temporal and spectral characteristics similar to speech sounds were presented to listeners as they were PET scanned. Nonspeech sounds were used so that any cortical asymmetry could not be associated with well-known asymmetries for language processing. Results indicated that the left STG and primary auditory cortex showed greater activation than the right STG for rapid (40 ms) formant frequency transitions but not for slow (200 ms) transitions. In addition, a left-hemisphere region of prefrontal cortex was asymmetrically activated for the rapid formant transition, which was corroborated in a separate fMRI study that used nearly identical acoustic stimuli (Temple et al., 2000). These data suggest that left-hemisphere auditory regions preferentially process rapid formant modulations present in ongoing speech.

In summary, modulations in the fundamental frequency of speech are faithfully encoded in the FFR. Moreover, these brainstem responses appear to be shaped by linguistic experience, a remarkable finding that indicates that cognitive processes (e.g., language) influence basic sensory processing. In the cortex, a mechanism for encoding frequency modulation is the specialization of left-hemisphere auditory regions, and results indicate that rapid frequency changes in speech-like stimuli preferentially activate the left hemisphere relative to slower frequency changes. In addition, the anterior insular cortex is activated for the processing of F0 modulations: The left-hemisphere insula is specifically activated when F0 modulations provide lexical information to a native speaker, whereas the right-hemisphere insula is activated when F0 modulations do not provide lexical information. These cortical findings would appear to be contradictory: The former indicates asymmetric activation by left-hemisphere structures is based on physical parameters of the speech signal, irrespective of linguistic content, whereas the latter suggests that linguistic context is essential for left-asymmetric insular processing of F0 modulations. However, Wong et al. (2004) stated that these results can be reconciled if the insular activity shown in their study occurs after the "acoustically specialized" cortical activity described by Belin et al. (1998) and Temple et al. (2000). If this were true, it would indicate two independent levels of cortical asymmetry: One based on the acoustic attributes of the signal and one based on the linguistic relevance to the listener. This hypothesis needs to be tested in future studies.

Electrophysiological Changes due to Training

There is ample evidence that multiple forms of auditory therapy and training have enhancing effects on the neural representation of frequency transitions in speech, including transitions of the fundamental and formant frequencies. Consistent with neural enhancement of formant structure

discussed previously, musical training also strengthens brainstem representations of frequency transitions, including representations of both the fundamental and formant frequencies. As discussed previously, one study showed that adult musicians have enhanced brainstem representations in response to tonal permutations of the Mandarin word "mi," which are characterized by contours to the fundamental frequency (Wong et al., 2007). It is hypothesized that this neural benefit is the result of years of attention to pitch variations in musical stimuli, and again it is significant that this neural advantage generalizes from the music domain to speech. In another study, it was shown that musical training also enhances brainstem representations of formant transitions in speech. For example, young children (3 to 5 years of age) with at least a year of musical training showed earlier brainstem responses to the formant transition portion of a consonant–vowel stimulus compared to age-matched listeners, with the greatest effects of musicianship being evident in the presence of background noise (Strait et al., 2013). Studies examining other forms of auditory training have also shown strengthening of brainstem responses to formant transitions in speech. In one study, two groups of older adults (mean age = 62 years) participated in different training paradigms matched for time and computer use: One group was trained on an adaptive computer-based auditory training program that combined bottom-up perceptual discrimination exercises with top-down cognitive demands whereas an active control group was trained on a general educational stimulation program (Anderson et al., 2013). Results for the auditory training group showed improved resiliency of speech-evoked brainstem responses in background noise, and this resiliency was most pronounced for the formant transition period of the consonant–vowel stimulus. This neural effect in the auditory training group was accompanied by significant improvement in a number of auditory behavioral and cognitive measures, including speech in noise, auditory memory, and processing speed. Importantly, the active control group failed to show improvements on both the neural and behavioral measures. A third study examined brainstem plasticity for yet another type of auditory therapy, in this case the use of assistive listening devices for use by children with reading impairments in the classroom (Hornickel et al., 2012b). The theoretical basis for providing these listening devices to this population is that children with reading impairments have impaired speech perception in noise relative to age-matched children (Bradlow et al., 2003). Importantly, assistive listening devices provide substantial improvements with regard to the signal-to-noise ratio of the teacher voice relative to classroom background noise. Results from this study showed that after using assistive listening devices for one academic year, children with reading impairments showed greater consistency of brainstem responses in the formant transition period of a consonant–vowel stimulus. These children also showed behavioral improvements on standardized measures of phonologic processing and reading ability. A control group, composed of reading-impaired children who did not use assistive listening devices, failed to show improvements in either of these neural or behavioral measures.

Taken together, results from these studies show that the neural representation of frequency transitions in speech is highly malleable in response to very different kinds of auditory training, including musical training, adaptive auditory-based computer programs, and the use of assistive listening devices. This suggests that therapies that sharpen "top-down" brain mechanisms, such as goal-directed attention to auditory stimuli, and "bottom-up" signals, as provided by assistive listening devices, can focus and improve the efficiency of neural mechanisms serving the tracking of frequency modulations. Moreover, the relative abundance of studies showing training effects for neural responses of frequency transitions further suggests that the brain's representation of this acoustical feature is particularly plastic, reflecting a critical auditory mechanism underlying rapid improvement in important auditory skill acquisition.

Acoustic Onsets

Acoustic Description and Role in the Perception of Speech

Acoustic onsets are defined here as the spectral and temporal features present at the beginning (the initial ~40 ms) of speech sounds. Although the acoustics of phonemes are only slightly altered based on their location in a word (i.e., beginning, middle, or end of a word), an emphasis has been put on acoustic onsets in the neurophysiological literature. Consequently, acoustic onsets are discussed here separately, despite some overlap with acoustic features (e.g., frequency transitions) discussed previously.

Onset acoustics of speech sounds vary considerably in both their spectral and temporal attributes. In some cases, the spectral features of the onset are essential for perception (e.g., the onset frequency of F2 for discriminating /da/ vs. /ga/), whereas in other cases temporal attributes of onsets are the critical feature for perception. A frequently studied acoustic phenomenon associated with the latter is that of the voice onset time (VOT), which is present in stop consonants. The VOT is defined as the duration of time between the release of a stop consonant by speech articulators and the beginning of vocal-fold vibration. The duration of the VOT is the primary acoustic cue that enables differentiation between consonants that are otherwise extremely similar (e.g., /da/ vs. /ta/, /ba/ vs. /pa/, /ga/ vs. /ka/).

Physiological Representation of Acoustic Onsets in the Human Brain

Auditory Brainstem

The brainstem response to speech-sound onsets has been studied extensively (Banai et al., 2005; Russo et al., 2004;

Wible et al., 2004). The first components of the speech-evoked ABR reflect the onset of the stimulus (Figure 28.2). Speech onset is represented in the brainstem response at approximately 7 ms in the form of two peaks, positive peak V and negative peak A.

Findings from a number of studies have demonstrated that the brainstem's response to acoustic transients is closely linked to auditory perception and to language function, including literacy. These studies have investigated brainstem responses to speech in normal children and children with language-based LDs, a population that has consistently demonstrated perceptual deficits in auditory tasks using both simple (Tallal and Piercy, 1973; Wright et al., 1997) and complex (Kraus et al., 1996; Tallal and Piercy, 1975) acoustic stimuli. A general hypothesis proposes a causal link between basic auditory perceptual deficits in LDs and higher-level language skills, such as reading and phonologic tasks (Tallal et al., 1993), although this relationship has been debated (Mody et al., 1997). In support of a hypothesis linking basic auditory function and language skills, studies of the auditory brainstem indicate a fundamental deficiency in the synchrony of auditory neurons in the brainstem for a significant proportion of language-disabled subjects.

The brainstem's response to acoustic transients in speech is an important neural indicator for distinguishing LD from typically developing (control) subjects. A number of studies have provided compelling evidence that the representation of speech onset is abnormal in a significant proportion of subjects with LD (Banai et al., 2009). For example, brainstem responses to the speech syllable /da/ were measured for a group of 33 normal children and 54 children with LD, and a "normal range" was established from the results of the normal subjects (King et al., 2002). Results indicated that 20 LD subjects (37%) showed abnormally late responses to onset peak A. Another study showed a significant difference between normal and LD subjects based on another measure of the brainstem's representation of acoustic transients (Wible et al., 2004). Specifically, it was shown that the slope between onset peaks V and A to the /da/ syllable was significantly smaller in subjects with LD compared to normal subjects. The authors of this study indicate that diminished V/A slope demonstrated by LDs is a measure of abnormal synchrony to the onset transients of the stimulus and could be the result of abnormal neural conduction by brainstem generators. In another study (Banai et al., 2005), LD subjects with abnormal brainstem timing for acoustic transients were more likely to have a more severe form of LD, manifested in poorer scores on measures of literacy, compared to LD subjects with normal brainstem responses. In yet another study, it was shown that the timing of children's brainstem onset responses to speech sounds correlated with standardized measures of reading and phonologic abilities across a wide range of reading abilities (Banai et al., 2009).

Taken together, these data suggest that the brainstem responses to acoustic transients can differentiate not only a subpopulation of LDs from normal subjects, but also within the LD population in terms of the severity of the disability. Findings from the brainstem measures also indicate a link between sensory encoding and cognitive processes such as literacy. An important question is whether the link between sensory encoding and cognition is a causal one, and if so, whether brainstem deficits are responsible for cortical deficits (or vice versa). Alternatively, these two abnormalities may be merely coincident. Nevertheless, the consistent findings of brainstem abnormalities in a sizable proportion of the LD population have led to the incorporation of this experimental paradigm into the clinical evaluation of LD and central auditory processing disorders.

Auditory Cortex

Cortical encoding of spectral features of speech-sound onsets has been reported in the literature (Obleser et al., 2006) and indicates that a spectral contrast at speech onset, resulting from consonant place of articulation (i.e., front produced consonant /d/ or /t/ vs. back produced consonant /g/ or /k/), is mapped along the anterior–posterior axis in auditory cortex as measured by N100m source location. This is significant because it indicates that phonemes differentially activate regions of auditory cortex according to their spectral characteristics at speech onset. It was also shown that the discrete mapping of consonants according to onset acoustics is effectively erased when the speech stimuli are manipulated to become unintelligible despite keeping the spectral complexity of the stimuli largely the same. This stimulus manipulation was accomplished by altering the spectral distribution of the stimuli. The authors argue that this latter finding indicates that the cortex is spatially mapping only those sounds that are intelligible to listeners. These data provide important evidence that cortical spatial representations may serve as an important mechanism for the encoding of spectral characteristics in speech-sound onsets. In addition to differences in spatial representations for place of articulation contrasts, cortical responses also showed latency differences for these contrasts. Specifically, it was shown that front consonants, which have higher frequency onsets, elicited earlier N100m responses than back consonants. This finding is consistent with near-field recordings measured from animal models indicating earlier response latencies for speech onsets with higher frequency formants (McGee et al., 1996).

Cortical responses to temporal features of speech-sound onsets have also been reported in the literature, many of which have utilized VOT contrasts as stimuli. These studies were performed by measuring obligatory evoked potentials (N100 responses) to continua of consonant–vowel speech sounds that varied gradually according to VOT (Sharma and Dorman, 1999, 2000; Sharma et al., 2000). Additionally, perception of these phonetic contrasts was also measured using the same continua as a means of addressing whether cortical responses reflected categorical perception of the phonemes.

Neurophysiological results indicated that for both /ba/-/pa/ and /ga/-/ka/ phonetic contrasts, one large negative peak was evident at approximately 100 ms in the response waveform for stimulus VOTs < 40 ms. A second negative peak in the response waveform emerged for stimulus VOTs of 40 ms, and this second peak occurred approximately 40 ms after the first peak and was thought to represent the onset of voicing in the stimulus. Moreover, as the VOT of the stimulus increased in duration, the lag between the second peak relative to the first increased proportionally, resulting in a strong correlation between VOT and the interpeak latency of the two peaks (r = ~0.80). The onset of double peaks in cortical responses with a VOT of 40 ms is consistent with neurophysiological responses measured directly from the auditory cortex of humans (Steinschneider et al., 1999), and an important consideration is that the onset of the double peak occurred at 40 ms for both /ba/-/pa/ and /ga/-/ka/ phonetic contrasts. In contrast, behavioral results require different VOTs to distinguish the /ba/-/pa/ and /ga/-/ka/ phonetic contrasts. Specifically, a VOT of ~40 ms was required for listeners to correctly identify /pa/ from /ba/, whereas a VOT of ~60 ms was required for correct identification of /ga/ from /ka/. Taken together, these data indicate that cortical responses reflect the actual VOT irrespective of the categorical perception of the phonetic contrasts.

Brainstem–Cortex Relationships

In addition to linking precise brainstem timing of acoustic transients to linguistic function, it has also been shown that abnormal encoding of acoustic transients in the brainstem is related to abnormal auditory responses measured at the level of cortex. In addition to their imprecise representation of sounds at the auditory brainstem, a significant proportion of LDs have also consistently demonstrated abnormal representations of simple and complex acoustic stimuli at the level of the auditory cortex. Three studies have linked abnormal neural synchrony for acoustic transients at the auditory brainstem to abnormal representations of sounds in the cortex. In one study, it was shown that a brainstem measure of the encoding of acoustic transients, the duration of time between onset peaks V and A, was positively correlated to auditory cortex's susceptibility to background noise in both normal and LD subjects (Wible et al., 2005). Specifically, the longer the duration between onset peaks V and A, the more degraded the cortical responses became in the presence of background noise. In another study, it was shown that individuals with abnormal brainstem timing to acoustic transients were more likely to indicate reduced cortical sensitivity to acoustic change, as measured by the mismatch negativity (MMN) response (Banai et al., 2005). Finally, a third study showed that brainstem timing for speech-sound onset and offset predicts the degree of cortical asymmetry for speech sounds measured across a group of children with a wide range of reading skills (Abrams et al., 2006). Results from these studies indicate that abnormal encoding of acoustic onsets at the brainstem may be a critical marker for systemic auditory deficits manifested at multiple levels of the auditory system, including the cortex.

In summary, evidence from examining the ABR indicates that acoustic transients are encoded in a relatively simple fashion in the brainstem, yet they represent a complex phenomenon that is related to linguistic ability and cortical function. In the cortex, results indicate that spectral contrasts of speech onsets are mapped along the anterior–posterior axis in the auditory cortex, whereas temporal attributes of speech onsets, as manifested by the VOT, are precisely encoded with double-peaked N100 responses.

Electrophysiological Changes due to Training

A survey of the brainstem and cortical literatures indicates that there is relatively scant evidence that the brain's representation of acoustic onsets is malleable following auditory-based training and therapy, and the primary evidence for plasticity of this feature is from a study of very young children. This study, which was previously described in the Formant Transition section, showed that a year or more of musical training in young children (3 to 5 years of age) resulted in decreased brainstem onset latencies in response to a consonant–vowel stimulus (Strait et al., 2013). Sound onsets are considered to be particularly rudimentary sound features, and the fact that the brainstem's response to acoustical onsets does not appear to be plastic following training (except in very young children) strongly suggests that this neural feature is established early in development and remains largely static irrespective of the experience of the individual. However, subcortical encoding of acoustic onsets does undergo substantial developmental changes across the lifespan, irrespective of training (Anderson et al., 2012; Skoe et al., in press).

The Speech Envelope

Definition and Role in the Perception of Speech

The speech envelope refers to temporal fluctuations in the speech signal under 50 Hz. The dominant frequency of the speech envelope is at ~4 Hz, which reflects the average syllabic rate of speech (Steeneken and Houtgast, 1980). Envelope frequencies in normal speech are generally below 8 Hz (Houtgast and Steeneken, 1985), and the perceptually essential frequencies of the speech envelope are between 4 and 16 Hz (Drullman et al., 1994), although frequencies above 16 Hz contribute slightly to speech recognition (Shannon et al., 1995). The speech envelope provides phonetic and prosodic cues to the duration of speech segments, manner of articulation, the presence (or absence) of voicing, syllabication, and stress. The perceptual significance of the speech envelope has been investigated using a number of methodologies (Drullman et al., 1994; Shannon

et al., 1995) and, taken together, these data indicate that the speech envelope is both necessary and sufficient for normal speech recognition.

Physiological Representation of the Speech Envelope in Auditory Cortex

Only a few studies have investigated how the human brain represents the slow temporal information of the speech envelope. It should be noted that the representation of the speech envelope in humans has only been studied at the level of the cortex, since measuring ABRs typically involves filtering out the neurophysiological activity below ~100 Hz (Hall, 1992). Since speech envelope frequencies are between 2 and 50 Hz, any linear representation of speech envelope timing in brainstem responses is removed with brainstem filtering.

In one EEG study, responses from the auditory cortex to conversational, clearly enunciated, and time-compressed (i.e., rapid) speech sentences were measured in children (Abrams et al., 2008). Results indicate that human cortex synchronizes its response to the contours of the speech envelope across all three speech conditions and that responses measured from right-hemisphere auditory cortex showed consistently greater phase-locking and response magnitude compared to left-hemisphere responses. An MEG study showed similar results; however, in this study, it was shown that these neurophysiological measures of speech envelope phase-locking correlated with subjects' ability to perceive the speech sentences: As speech sentences become more difficult to perceive, the ability of the cortex to phase-lock to the speech sentence was more impaired (Ahissar et al., 2001). These results are in concert with results from the animal literature, which show that neurons of primary auditory cortex represent the temporal envelope of complex acoustic stimuli (i.e., animal communication calls) by phase-locking to this temporal feature of the stimulus (Wang et al., 1995).

A second line of inquiry into the cortical representation of speech envelope cues was described previously in this chapter in the discussion of cortical responses to VOT (Sharma and Dorman, 1999, 2000; Sharma et al., 2000). Acoustically, VOT is a slow temporal cue in speech (40 to 60 ms; 17 to 25 Hz) that falls within the range of speech envelope frequencies. Briefly, neurophysiological results indicated that for both /ba/-/pa/ and /ga/-/ka/ phonetic contrasts, cortical N100 responses precisely represented the acoustic attributes of the VOT. In addition, it was shown that neural responses were independent of the categorical perception of these phonetic contrasts (see the Acoustic Onsets section for a more detailed description of this study).

On the surface, it may appear that the findings from these experiments contradict one another since cortical phase-locking to the speech envelope correlates with perception in one study (Ahissar et al., 2001) whereas phase-locking fails to correlate with perception in the other study (Sharma and Dorman, 1999, 2000; Sharma et al., 2000). These data are not, however, in contradiction to one another. In both cases, an a priori requirement for perception is phase-locking to the speech envelope; there is no evidence for perception in the absence of accurate phase-locking to the temporal envelope in either study. The primary difference between the studies is that despite phase-locking to the temporal envelope in the /ka/ stimulus condition at a VOT of ~40 ms, reliable perception of /ka/ occurs at ~60 ms. This suggests that accurate phase-locking is required for perception; however, perception cannot be predicted by phase-locking alone. Presumably, in the case of the /ka/ VOT stimulus, there is another processing stage that uses the phase-locked temporal information in conjunction with additional auditory-linguistic information (e.g., repeated exposure to /ka/ stimuli with 60 ms VOT) as a means to form phonetic category boundaries. The question of if and how category boundaries are established irrespective of auditory phase-locking requires additional investigation.

CONCLUSIONS

Speech is a highly complex signal composed of a variety of acoustic features, all of which are important for normal speech perception. Normal perception of these acoustic features certainly relies on their neural encoding, which has been the subject of this review. An obvious conclusion from these studies is that the central auditory system is a remarkable machine, able to simultaneously process the multiple acoustic cues of ongoing speech to decode a linguistic message. Furthermore, how the human brain is innately and dynamically programmed to utilize any number of these acoustic cues for the purpose of language, given the appropriate degree and type of stimulus exposure, further underscores the magnificence of this system.

The primary goals of this chapter are to describe our current understanding of neural representation of speech as well as training-related changes to these representations. By exploring these two topics concurrently it is argued that we have provided complementary perspectives on auditory function: The initial descriptions of brainstem and cortical representations of these speech features are thought to reflect "bottom-up" function of the auditory system with minimal consideration for the dynamic interactions provided by top-down connections in the auditory system (Xiao and Suga, 2002); in contrast, the descriptions of training-related changes to these representations provide information regarding how "top-down" cognitive and brain mechanisms sharpen these auditory representations (reviewed in Kraus and Chandrasekaran, 2010). Evidence accumulated across studies provides a complicated, but compelling, account of the malleability of these auditory responses. Results show that brainstem representations of speech can be affected and sharpened by multiple forms of auditory-based experiences, from long-term musical experiences to relatively short-term

auditory-cognitive training paradigms. Importantly, the relative plasticity of these different speech features appears to fall on a continuum: Acoustic onsets, which are largely static following all forms of auditory training, occupy one end of this continuum, whereas neural representations of formant transitions occupy the other end of this continuum, showing enhanced response properties following multiple training paradigms measured in a wide range of subject populations. Consistent with the animal literature (Recanzone et al., 1993), it is plausible that the relative plasticity of these features reflects the behavioral demands of each form of training, and a prediction of this hypothesis is that relatively static neural representations do not significantly contribute to the improvement on these tasks whereas more dynamic neural representations are important for improved performance.

To garner a greater understanding of how the central auditory system processes speech, it is important to consider subcortical and cortical auditory regions as reciprocally interactive. Indeed, auditory processing reflects an interaction of sensory, cognitive, and reward systems. Across the acoustic features described in this review, the brainstem appears to represent discrete acoustic events: The fundamental frequency and its modulation are represented with highly synchronized activity as reflected by the FFR; speech-sound onset is represented with highly predictable neural activation patterns that vary within fractions of milliseconds. Alternatively, the cortex appears to transform many of these acoustic cues, resulting in more complex representations of acoustic features of speech. For example, many of the cortical findings described here are based on the spatial representation of acoustic features (i.e., the relationship between F1 and F2 required for vowel identification; the differentiation of speech transients; the encoding of periodicity). Because cortical neurons are not able to phase-lock to high-frequency events, it is tempting to propose that cortex has found an alternative method for encoding these features based on the activity of spatially distributed neural populations. The extent to which these acoustic features are truly represented via a spatial organization in cortex is a future challenge that will be likely achieved using high-resolution imaging technologies in concert with EEG and MEG technologies.

FOOD FOR THOUGHT

Here, we have described what is currently known about brain representations of key elements of speech that are necessary for normal speech perception. Our review covers information garnered from multiple research methodologies, including brainstem- and cortical-evoked responses using EEG, which provide crucial information regarding the neural timing in response to specific speech features, as well as fMRI research, which provides complementary information regarding "where" in the brain this activity occurs. Furthermore, we have described the relative plasticity of these brain responses as a result of specific behavioral experiences, with an emphasis on musical training. The following are important questions for future research that will enable us to further understand the brain basis of speech perception as well as associated plasticity and impairments.

1. Both the auditory brainstem and cortical regions are highly sensitive to elements of speech structure. An important question is what is the relationship between the integrity of brainstem representations of speech structure and cortical regions beyond auditory cortex that are known to be critical for structural processing of speech? For example, the posterior temporal sulcus is considered "voice-selective cortex" (Belin et al., 2000) and has been proposed to be a critical gateway which enables speech information to access other brain networks that serve semantic, reward, and mnemonic processes (Belin et al., 2011). A better understanding of how lower levels of the auditory hierarchy (i.e., the auditory brainstem) impact voice selectivity in the posterior temporal sulcus would provide important information regarding the function of this extensive network.
2. While humans are drawn to the sounds of speech, it is seldom considered a "rewarding" stimulus. Perhaps for this reason little research has been conducted to study the brain networks that are used for pleasurable speech. For example, what parts of the auditory hierarchy are differentially activated in response to pleasurable compared to neutral speech? Would these pleasurable speech sounds provide altered neural responses across the entire auditory hierarchy, or alternatively would only specific regions of the brain show effects of pleasure?
3. Research described in this chapter has convincingly shown that speech in noise perception is greatly improved through musical training (Parbery-Clark et al., 2012; Song et al., 2012). An exciting question is what are the particular neural mechanisms that enable this effect of musicianship? What aspects of musical training facilitate these behavioral advantages, and how might we harness this information to train individuals of all ages to become better listeners in noisy environments?

ACKNOWLEDGMENTS

We thank Trent Nicol for his comments on a previous draft of this chapter. This work is supported by F32DC010322, DC011095, and the Mosbacher Foundation (DAA) and the Hugh Knowles Center, NIH RO1DC10016, R01HD069414, NSF 0921275, NSF1057566, and NSF1015614 (NK).

REFERENCES

Abrams DA, Nicol T, Zecker S, Kraus N. (2008) Right-hemisphere auditory cortex is dominant for coding syllable patterns in speech. *J Neurosci.* 28, 3958–3965.

Abrams DA, Nicol T, Zecker SG, Kraus N. (2006) Auditory brainstem timing predicts cerebral asymmetry for speech. *J Neurosci.* 26, 11131–11137.

Ahissar E, Nagarajan S, Ahissar M, Protopapas A, Mahncke H, Merzenich MM. (2001) Speech comprehension is correlated with temporal response patterns recorded from auditory cortex. *Proc Natl Acad Sci USA.* 98, 13367–13372.

Ahissar M, Hochstein S. (2004) The reverse hierarchy theory of visual perceptual learning. *Trends Cogn Sci.* 8, 457–464.

Anderson S, Parbery-Clark A, Yi HG, Kraus N. (2011) A neural basis of speech-in-noise perception in older adults. *Ear Hear.* 32, 750–757.

Anderson S, Parbery-Clark A, White-Schwoch T, Kraus N. (2012) Aging affects neural precision of speech encoding. *J Neurosci.* 32 (41), 14156–14164.

Anderson S, White-Schwoch T, Parbery-Clark A, Kraus N. (2013) Reversal of age-related neural timing delays with training. *Proc Natl Acad Sci USA.* 110, 4357–4362.

Banai K, Hornickel J, Skoe E, Nicol T, Zecker S, Kraus N. (2009) Reading and subcortical auditory function. *Cereb Cortex.* 19, 2699–2707.

Banai K, Nicol T, Zecker SG, Kraus N. (2005) Brainstem timing: Implications for cortical processing and literacy. *J Neurosci.* 25, 9850–9857.

Belin P, Bestelmeyer PE, Latinus M, Watson R. (2011) Understanding voice perception. *Br J Psychol.* 102, 711–725.

Belin P, Zatorre RJ, Lafaille P, Ahad P, Pike B. (2000) Voice-selective areas in human auditory cortex. *Nature.* 403, 309–312.

Belin P, Zilbovicius M, Crozier S, Thivard L, Fontaine A, Masure MC, et al. (1998) Lateralization of speech and auditory temporal processing. *J Cogn Neurosci.* 10, 536–540.

Bradlow AR, Kraus N, Hayes E. (2003) Speaking clearly for children with learning disabilities: sentence perception in noise. *J Speech Lang Hear Res.* 46, 80–97.

Burk MH, Humes LE. (2008) Effects of long-term training on aided speech-recognition performance in noise in older adults. *J Speech Lang Hear Res.* 51, 759–771.

Creutzfeldt O, Hellweg FC, Schreiner C. (1980) Thalamocortical transformation of responses to complex auditory stimuli. *Exp Brain Res.* 39, 87–104.

Cunningham J, Nicol T, King CD, Zecker SG, Kraus N. (2002) Effects of noise and cue enhancement on neural responses to speech in auditory midbrain, thalamus and cortex. *Hear Res.* 169, 97–111.

Diesch E, Luce T. (1997) Magnetic fields elicited by tones and vowel formants reveal tonotopy and nonlinear summation of cortical activation. *Psychophysiology.* 34, 501–510.

Drullman R, Festen JM, Plomp R. (1994) Effect of temporal envelope smearing on speech reception. *J Acoust Soc Am.* 95, 1053–1064.

Geschwind N, Galaburda AM. (1985) Cerebral lateralization. Biological mechanisms, associations, and pathology: I. A hypothesis and a program for research. *Arch Neurol.* 42, 428–459.

Hall JH. (1992) *Handbook of Auditory Evoked Responses.* Boston, MA: Allyn and Bacon.

Halliday LF, Taylor JL, Millward KE, Moore DR. (2012) Lack of generalization of auditory learning in typically developing children. *J Speech Lang Hear Res.* 55, 168–181.

Hornickel J, Anderson S, Skoe E, Yi HG, Kraus N. (2012a) Subcortical representation of speech fine structure relates to reading ability. *Neuroreport.* 23, 6–9.

Hornickel J, Zecker SG, Bradlow AR, Kraus N. (2012b) Assistive listening devices drive neuroplasticity in children with dyslexia. *Proc Natl Acad Sci USA.* 109, 16731–16736.

Houtgast T, Steeneken HJM. (1985) A review of the MTF concept in room acoustics and its use for estimating speech intelligibility in auditoria. *J Acoust Soc Am.* 77, 1069–1077.

Johnson K. (1997) *Acoustic and Auditory Phonetics.* Cambridge, MA: Blackwell Publishers Inc.

Killion MC, Niquette PA, Gudmundsen GI, Revit LJ, Banerjee S. (2004) Development of a quick speech-in-noise test for measuring signal-to-noise ratio loss in normal-hearing and hearing-impaired listeners. *J Acoust Soc Am.* 116, 2395–2405.

King C, Warrier CM, Hayes E, Kraus N. (2002) Deficits in auditory brainstem pathway encoding of speech sounds in children with learning problems. *Neurosci Lett.* 319, 111–115.

Kraus N, Chandrasekaran B. (2010) Music training for the development of auditory skills. *Nat Rev Neurosci.* 11, 599–605.

Kraus N, McGee TJ, Carrell TD, Zecker SG, Nicol TG, Koch DB. (1996) Auditory neurophysiologic responses and discrimination deficits in children with learning problems. *Science.* 273, 971–973.

Kraus N, Nicol T. (2005) Brainstem origins for cortical 'what' and 'where' pathways in the auditory system. *Trends Neurosci.* 28, 176–181.

Krishnan A. (2002) Human frequency-following responses: representation of steady-state synthetic vowels. *Hear Res.* 166, 192–201.

Krishnan A, Xu Y, Gandour J, Cariani P. (2005) Encoding of pitch in the human brainstem is sensitive to language experience. *Brain Res Cogn Brain Res.* 25, 161–168.

Krishnan A, Xu Y, Gandour JT, Cariani PA. (2004) Human frequency-following response: representation of pitch contours in Chinese tones. *Hear Res.* 189, 1–12.

Lehiste I, Peterson GE. (1961) Transitions, glides, and diphthongs. *J Acoust Soc Am.* 33, 268–277.

Lin FR, Yaffe K, Xia J, Xue QL, Harris TB, Purchase-Helzner E, et al. (2013) Hearing loss and cognitive decline in older adults. *JAMA Intern Med.* 173, 293–299.

Lu T, Liang L, Wang X. (2001) Temporal and rate representations of time-varying signals in the auditory cortex of awake primates. *Nat Neurosci.* 4, 1131–1138.

Makela AM, Alku P, Makinen V, Valtonen J, May P, Tiitinen H. (2002) Human cortical dynamics determined by speech fundamental frequency. *Neuroimage.* 17, 1300–1305.

McGee T, Kraus N, King C, Nicol T, Carrell TD. (1996) Acoustic elements of speechlike stimuli are reflected in surface recorded responses over the guinea pig temporal lobe. *J Acoust Soc Am.* 99, 3606–3614.

Mody M, Studdert-Kennedy M, Brady S. (1997) Speech perception deficits in poor readers: auditory processing or phonological coding? *J Exp Child Psychol.* 64, 199–231.

Moreno S, Marques C, Santos A, Santos M, Castro SL, Besson M. (2009) Musical training influences linguistic abilities in 8-year-old children: more evidence for brain plasticity. *Cereb Cortex.* 19, 712–723.

Naatanen R, Picton T. (1987) The N1 wave of the human electric and magnetic response to sound: a review and an analysis of the component structure. *Psychophysiology.* 24, 375–425.

Nearey TM, Assmann PF. (1986) Modeling the role of inherent spectral change in vowel identification. *J Acoust Soc Am.* 80, 1297–1308.

Nilsson M, Soli SD, Sullivan JA. (1994) Development of the Hearing in Noise Test for the measurement of speech reception thresholds in quiet and in noise. *J Acoust Soc Am.* 95, 1085–1099.

Nusbaum HC, Morin TM. (1992) Paying attention to differences among talkers. In: Tohkura Y, Sagisaka Y, Vatikiotis-Bateson E, eds. *Speech Perception, Production, and Linguistic Structure.* Tokyo: Ohmasha Publishing; pp 113–134.

Obleser J, Elbert T, Lahiri A, Eulitz C. (2003) Cortical representation of vowels reflects acoustic dissimilarity determined by formant frequencies. *Brain Res Cogn Brain Res.* 15, 207–213.

Obleser J, Scott SK, Eulitz C. (2006) Now you hear it, now you don't: transient traces of consonants and their nonspeech analogues in the human brain. *Cereb Cortex.* 16, 1069–1076.

Parbery-Clark A, Tierney A, Strait DL, Kraus N. (2012) Musicians have fine-tuned neural distinction of speech syllables. *Neuroscience.* 219, 111–119.

Peterson GE, Barney HL. (1952) Control methods used in a study of the vowels. *J Acoust Soc Am.* 24, 175–184.

Recanzone GH, Schreiner CE, Merzenich MM. (1993) Plasticity in the frequency representation of primary auditory cortex following discrimination training in adult owl monkeys. *J Neurosci.* 13, 87–103.

Rosen S. (1992) Temporal information in speech: acoustic, auditory and linguistic aspects. *Philos Trans R Soc Lond B Biol Sci.* 336, 367–373.

Russo N, Nicol T, Musacchia G, Kraus N. (2004) Brainstem responses to speech syllables. *Clin Neurophysiol.* 115, 2021–2030.

Sachs MB, Young ED. (1979) Encoding of steady-state vowels in the auditory nerve: representation in terms of discharge rate. *J Acoust Soc Am.* 66, 470–479.

Schon D, Magne C, Besson M. (2004) The music of speech: music training facilitates pitch processing in both music and language. *Psychophysiology.* 41, 341–349.

Schreiner CE. (1998) Spatial distribution of responses to simple and complex sounds in the primary auditory cortex. *Audiol Neurootol.* 3, 104–122.

Shannon RV, Zeng FG, Kamath V, Wygonski J, Ekelid M. (1995) Speech recognition with primarily temporal cues. *Science.* 270, 303–304.

Sharma A, Dorman M. (2000) Neurophysiologic correlates of cross-language phonetic perception. *J Acoust Soc Am.* 107, 2697–2703.

Sharma A, Dorman MF. (1999) Cortical auditory evoked potential correlates of categorical perception of voice-onset time. *J Acoust Soc Am.* 106, 1078–1083.

Sharma A, Marsh C, Dorman M. (2000) Relationship between N1 evoked potential morphology and the perception of voicing. *J Acoust Soc Am.* 108, 3030–3035.

Shestakova A, Brattico E, Soloviev A, Klucharev V, Huotilainen M. (2004) Orderly cortical representation of vowel categories presented by multiple exemplars. *Brain Res Cogn Brain Res.* 21, 342–350.

Skoe E, Kraus N. (2010) Auditory brain stem response to complex sounds: a tutorial. *Ear Hear.* 31 (3), 302–324.

Skoe E, Krizman J, Anderson S, Kraus N. (in press) Stability and plasticity of auditory brainstem function across the lifespan. *Cereb Cortex*. doi: 10.1093/cercor/bht311

Smith AJ, Blumenfeld H, Behar KL, Rothman DL, Shulman RG, Hyder F. (2002) Cerebral energetics and spiking frequency: the neurophysiological basis of fMRI. *Proc Natl Acad Sci USA*. 99, 10765–10770.

Smith JC, Marsh JT, Brown WS. (1975) Far-field recorded frequency-following responses: evidence for the locus of brainstem sources. *Electroencephalogr Clin Neurophysiol.* 39, 465–472.

Song JH, Banai K, Russo NM, Kraus N. (2006) On the relationship between speech- and nonspeech-evoked auditory brainstem responses. *Audiol Neurootol.* 11, 233–241.

Song JH, Skoe E, Banai K, Kraus N. (2012) Training to improve hearing speech in noise: biological mechanisms. *Cereb Cortex.* 22, 1180–1190.

Steeneken HJ, Houtgast T. (1980) A physical method for measuring speech-transmission quality. *J Acoust Soc Am.* 67, 318–326.

Steinschneider M, Volkov IO, Noh MD, Garell PC, Howard MA 3rd. (1999) Temporal encoding of the voice onset time phonetic parameter by field potentials recorded directly from human auditory cortex. *J Neurophysiol.* 82, 2346–2357.

Stillman RD, Crow G, Moushegian G. (1978) Components of the frequency-following potential in man. *Electroencephalogr Clin Neurophysiol.* 44, 438–446.

Strait DL, Kraus N. (2014) Biological impact of auditory expertise across the life span: musicians as a model of auditory learning. *Hear Res*. 308, 109–121.

Strait DL, O'Connell S, Parbery-Clark A, Kraus N. (in press) Musicians' enhanced neural differentiation of speech sounds arises early in life: developmental evidence from ages three to thirty. *Cereb Cortex*. doi:10.1093/cercor/bht103.

Strait DL, Parbery-Clark A, O'Connell S, Kraus N. (2013) Biological impact of preschool music classes on processing speech in noise. *Dev Cogn Neurosci.* 6, 51–60.

Sweetow RW, Sabes JH. (2006) The need for and development of an adaptive Listening and Communication Enhancement (LACE) program. *J Am Acad Audiol*. 17, 538–558.

Tallal P, Miller S, Fitch RH. (1993) Neurobiological basis of speech: a case for the preeminence of temporal processing. *Ann N Y Acad Sci.* 682, 27–47.

Tallal P, Piercy M. (1973) Defects of non-verbal auditory perception in children with developmental aphasia. *Nature.* 241, 468–469.

Tallal P, Piercy M. (1975) Developmental aphasia: the perception of brief vowels and extended stop consonants. *Neuropsychologia.* 13, 69–74.

Temple E, Poldrack RA, Protopapas A, Nagarajan S, Salz T, Tallal P, et al. (2000) Disruption of the neural response to rapid acoustic stimuli in dyslexia: evidence from functional MRI. *Proc Natl Acad Sci USA*. 97, 13907–13912.

Thompson WF, Schellenberg EG, Husain G. (2004) Decoding speech prosody: do music lessons help? *Emotion.* 4, 46–64.

Wang X, Merzenich MM, Beitel R, Schreiner CE. (1995) Representation of a species-specific vocalization in the primary auditory cortex of the common marmoset: temporal and spectral characteristics. *J Neurophysiol.* 74, 2685–2706.

Wible B, Nicol T, Kraus N. (2004) Atypical brainstem representation of onset and formant structure of speech sounds in children with language-based learning problems. *Biol Psychol.* 67, 299–317.

Wible B, Nicol T, Kraus N. (2005) Correlation between brainstem and cortical auditory processes in normal and language-impaired children. *Brain.* 128, 417–423.

Wong PC, Parsons LM, Martinez M, Diehl RL. (2004) The role of the insular cortex in pitch pattern perception: the effect of linguistic contexts. *J Neurosci*. 24, 9153–9160.

Wong PC, Skoe E, Russo NM, Dees T, Kraus N. (2007) Musical experience shapes human brainstem encoding of linguistic pitch patterns. *Nat Neurosci.* 10, 420–422.

Wright BA, Lombardino LJ, King WM, Puranik CS, Leonard CM, Merzenich MM. (1997) Deficits in auditory temporal and spectral resolution in language-impaired children. *Nature.* 387, 176–178.

Xiao Z, Suga N. (2002) Modulation of cochlear hair cells by the auditory cortex in the mustached bat. *Nat Neurosci.* 5, 57–63.

Xu Y, Krishnan A, Gandour JT. (2006) Specificity of experience-dependent pitch representation in the brainstem. *Neuroreport.* 17, 1601–1605.

Zatorre RJ, Belin P, Penhune VB. (2002) Structure and function of auditory cortex: music and speech. *Trends Cogn Sci.* 6, 37–46.

CHAPTER 29

Central Auditory Processing Evaluation: A Test Battery Approach

Kim L. Tillery

INTRODUCTION

Audiologists routinely administer peripheral hearing assessments and a growing number also administer central auditory processing (CAP) tests. It seems logical that audiologists should consider to assist individuals with difficulty in "hearing" the auditory message, whether it is because of a peripheral dysfunction, central dysfunction, or both. After all, the hearing system is complex and we should be able to assess the integrity of the entire hearing system to better serve those who struggle in communication, language, and learning functions. It appears that the educational training dealing with central auditory processing disorder (CAPD) in clinical doctorate of audiology (Au.D.) programs has increased the awareness of CAPD in audiologists and in the importance of the test battery approach in forming a comprehensive understanding.

BACKGROUND

CAPD was first officially described in 1992 by the American Speech-Language-Hearing Association (ASHA). The general definition described CAPD as having difficulty in retrieving, transforming, analyzing, organizing, and storing information from audible acoustic signals (ASHA, 1992). This simple definition was later expanded when ASHA initiated a task force to discuss and derive the first professional consensus of several issues involving central auditory processing disorders (CAPDs) (ASHA, 1995, 1996). The issues included a definition, basic auditory science, assessment, clinical application, and developmental and acquired communication problems. CAPD was defined as involving deficits in localization, lateralization, auditory discrimination, pattern recognition skills, temporal processing, and performance decrements with competing or degraded auditory signals.

The consensus provided recommendations for determining the presence of CAPD and its functional deficits, emphasizing a team approach and the delineation of developmental or acquired CAPD deficits. Management approaches were focused on enhancing processing skills by increasing the language knowledge base and improving processing speed. Intervention goals were to bridge linguistic and cognitive perception (within the realm of the speech pathologist) and the acoustic properties of speech (within the realm of the audiologist), thus enabling the client with a CAPD to function better with a minimum of behavioral deficits. The consensus document encouraged collaborative efforts between clinicians and researchers to improve our understanding of CAPD.

ASHA provided an updated technical report on CAPD in 2005. This report recognized the previously accepted ASHA definition and detailed a number of additional topics, including a review of basic science advances, audiometric assessment, developmental and acquired communication problems associated with CAPD, and the use of diagnostic information to indicate specific interventions. Although the Bruton Conference (Jerger and Musiek, 2000) suggested removing the word "central" from the title of this disorder, the ASHA (2005) report did not take a stand on the preferred title but rather indicated that both were acceptable. They recommended that the word "central" remain in the title, as (central) auditory processing disorder or CAPD, because most of the tests administered for CAPD diagnosis involve the central auditory nervous system (CANS), which is reiterated in the American Academy of Audiology (AAA) 2010 guidelines.

A second clarification provided in the ASHA (2005) Technical Report addressed the "modality-specific" approach to diagnosing CAPD (Cacace and McFarland, 2005; Jerger and Musiek, 2000). This approach, initiated by one group of researchers over the last decade (Cacace and McFarland, 2005; McFarland and Cacace, 1995, 1997), hinges on whether a CAPD evaluation should be purely auditory or might include other sensory and supramodal systems, such as language (Katz and Tillery, 2005; Musiek et al., 2005). The ASHA (2005) report provides substantial research and reasoning that a diagnostic criterion to rule out all other perceptual factors is not consistent with brain organization and central nervous system (CNS) processing and that assessment of multimodality function is not within

the scope of one professional discipline. In addition, the report stated that influences of maturational delays, extent of neurobiologic disorders, social and environmental factors, and neurologic disorders or diseases most certainly can impact different individuals with the same auditory deficit in different ways. It was concluded that CAPD involves a neural processing deficit of auditory stimuli that may "coexist with, but *is not the result of,* dysfunction in other modalities" (ASHA, 2005, p 3). Thus, CAPD is described as a distinct clinical entity that relates to complex skills including speech, reading, and other functions.

Five years later, the AAA (2010) guidelines elaborated on the potential influence of poor motivation, fatigue, or attention issues as sources that might cause a decreased test performance toward the end of the 45- to 60-minute test battery. The guidelines stress the need to use more than one test, in particular, to be conscious that sensitivity may be raised when increasing the number of tests whereas specificity may be reduced. The audiologist must be aware that the more tests that are used the more likely it is to have errors because of attention or fatigue. The purpose of an evaluation is to (1) identify strengths and weaknesses in the auditory system and (2) differentiate normal versus abnormal performance.

Successive position statements support the importance of diagnosis and treatment of children and adults. This was echoed most recently by the 2012 Canadian Guidelines on CAPD which incorporates the British Society of Audiology Position Statement (2011) and the International Classification of Functioning, Disability, and Health (ICF) of the World Health Organization (WHO, 2001). The latter ensures two main principles to be considered. First, the focus should involve assessment and intervention in meeting the needs of the individual and family. Second, the end result should consider clinical, social, vocational, educational, and community needs. In other words, is there community support for remediation and how do the processing deficits influence one's life? Secondly, there should be a consideration of clinical, social, vocational, educational, and community needs. Further consideration should include developmental CAPD over time, acquired CAPD, and secondary CAPDs (i.e., peripheral hearing impairment or transient hearing issues because of otitis media or progressive presbycusis). Although all position papers show agreement on many of the whys and why not issues in proper diagnosis and intervention, the Canadian Guidelines offer more thorough information on the above models than the previous position statements.

Various authors have described the severity and variety of functional behavioral limitations caused by CAPD and coexisting disorders (Chermak et al., 1999; Keller, 1992, 1998; Keller and Tillery, 2002, 2014). Questionnaires can be useful for indicating the types of functional limitations present and assisting in appropriate referrals (Moore et al., 2013; Tillery, 1998). Given the associated language, communication, and learning difficulties frequently associated with CAPD, a multidisciplinary approach can lead to more accurate diagnoses, thereby enhancing effective treatment and management plans (Keller and Tillery, 2002, 2014). Although a team approach is recommended to determine the problems associated with the client's communication skills (Keller and Tillery, 2014), it is the audiologist who administers tests to determine the integrity of the CANS (AAA, 2010; ASHA, 1995, 2005; Canadian Guidelines, 2012).

CENTRAL AUDITORY PROCESSING TEST BATTERY APPROACH

As early as 1954, Mykelbust suggested that children with language disorders may have an auditory deficit beyond peripheral hearing and that the clinician should assess for such possibilities (Mykelbust, 1954). This early suggestion came when there were no audiologic tests to determine auditory functioning beyond the peripheral system. Today, there are quite a few central tests, and the test battery approach continues to be well recognized for CAP assessment (Domitz and Schow, 2000; Rawool, 2013).

The intent of CAP evaluations is to assess the CANS system at different levels. The efficacy of any test is determined by examining how it compares with different assessment tools (AAA, 2010; ASHA, 2005). Such comparisons may indicate that two or three auditory processing tests provide the same conclusions as six or seven other tests (AAA, 2010; Musiek and Lamb, 1994).

CAP tests have been in use for decades. The reader is referred to the following for a review of these tests: Bellis (2003), Katz (1994), Rawool (2013), and Weihing et al. (2013). Table 29.1 lists the CAP tests with their targeted processes and CANS sensitivity.

In addition to tests and test batteries that provide insights into CANS system issues, we now recognize that such an approach can help us to determine the possible underlying auditory mechanisms. For example, Schow et al. (2000) analyzed various central tests in comparison to the ASHA (1996) processes listed and determined the following measurable auditory behaviors: (1) Auditory pattern temporal ordering (APTO), (2) monaural separation/closure (MSC), (3) binaural separation (BS), and (4) binaural integration (BI). In addition, these authors suggested that CAPD testing can also evaluate auditory discrimination, localization/lateralization, and temporal tasks (resolution, masking, and integration). Table 29.2 provides definitions of these measurable auditory behaviors with associated CAP tests. The Schow et al. (2000) study is an example that illustrates the selection of tests, in this case based on auditory behaviors that should be assessed according to ASHA (1995, 2005); however, as discussed in the following sections, researchers have developed test batteries based on various conceptualizations of what they sought to examine.

TABLE 29.1

Summary of Central Auditory Processing Tests with Targeted Process and Central Auditory Nervous System Sensitivity to Specific Sites

	Monaural	Targeted Processes	Sensitive To:
Low-Pass Filtered Speech Tests			
Band-Pass Filtered	X	Auditory closure	Brainstem/cortical lesions
Compressed Speech	X	Auditory closure	Primary auditory cortex
Speech Recognition in Noise	X	Auditory closure	Brainstem to cortex
Dichotic Speech Tests			
Staggered Spondaic Word		Binaural integration	Brainstem/cortical/corpus callosum
Dichotic Digits		Binaural integration	Brainstem/cortical/corpus callosum
Synthetic Sentence Identification w/Contra Competing Message		Binaural separation	Cortical vs. brainstem
Competing Sentences		Binaural separation	Language processing
Dichotic Sentence Identification		Binaural integration	Brainstem/cortical
Dichotic Rhyme		Binaural integration	Interhemispheric
Dichotic Consonant Vowels		Binaural integration	Cortical
Temporal Patterning Tests			
Pitch Pattern Sequence (PPS)	X	Temporal ordering Linguistic labeling	Cerebral hemisphere lesions Interhemispheric transfer
Duration Patterns	X	Temporal ordering Linguistic labeling Duration discrimination	Cerebral hemisphere lesions Interhemispheric transfer
Random Gap Detection Test		Temporal resolution	Left temporal/cortical
Gaps-in-Noise	X	Temporal resolution	Interhemispheric transfer
Frequency Pattern (FP)	X	Temporal ordering Linguistic labeling Frequency discrimination	Cerebral hemisphere lesions
Other Tests			
Binaural Fusion		Binaural integration	Low brainstem
Masking Level Difference		Binaural interaction	Low brainstem
Rapid Alternating Speech		Binaural interaction	?Low or high brainstem

CENTRAL AUDITORY PROCESSING TEST BATTERY MODELS

Some CAP models that have been in use for the past several years or longer will be discussed below. Some were developed to determine the underlying auditory difficulties that relate to communicative and academic deficits (Bellis and Ferre, 1999; Katz and Smith, 1991), others tended to emphasize a medical framework (Jerger and Musiek, 2000), whereas others are based on the intertwining of cognitive, language, and auditory processes (Medwetsky, 2011).

These models incorporate different tests depending on the desired outcome of the applied construct. Three of the four models deliver subtypes or profiles that describe the CAPD, rather than pointing to a general CANS diagnosis. These models provide more information about an individual's functional limitations and, in turn, suggest effective treatment opportunities. Regardless of the underlying construct, all of these models rely on CANS tests being administered in a manner that controls for fatigue and attention since these can affect test performance (AAA, 2010; Moore et al., 2013; Tillery, 2013; Tillery et al., 2000).

Minimal Test Battery

Jerger and Musiek (2000) discussed a possible test battery that would include both behavioral and electrophysiological testing. The authors suggested this battery as a minimum:

- Immittance audiometry (tympanometry and acoustic reflex threshold testing) to ascertain the status of the middle ear as well as auditory neuropathy differential diagnosis
- Otoacoustic emissions to diagnose inner ear problems

TABLE 29.2

Measurable Auditory Processing Behavioral Processes Recommended by Schow et al. (2000) and Adopted by the Bellis/Ferre Model

Process	Measurable Auditory Processing Performance	Types of Tests	Bellis/Ferre Profiles
Auditory pattern/ temporal ordering [APTO]	Auditory discrimination of frequency or duration/order and sequencing/temporal processes/interhemispheric integration	1. Frequency pattern tests 2. Duration pattern tests 3. Pitch Pattern Sequence Test	Prosodic deficit
Monaural separation/ closure [MSC]	Performance with degraded signals	1. Filtered or time-compressed speech 2. Ipsilateral competing signals	Auditory decoding deficit
Binaural integration [BI]	Ability to properly respond to all competing signals directed to both ears	Dichotic tests	Integration deficit
Binaural separation [BS]	Ability to attend to stimulus in one ear while ignoring the stimulus in the other ear	Competing sentences	Integration deficit
Sound localization/ lateralization	Ability to describe location of stimuli in relation to position of one's head	Brainstem-level binaural interaction tests [masking level difference [MLD]]	Integration deficit
Auditory discrimination	Ability to describe when two stimuli are different	1. Difference limens for frequency/duration/ intensity or speech stimuli 2. Speech-sound or word discrimination tests	Auditory decoding deficit
Temporal resolution Temporal masking Temporal integration	Discrimination of speech and nonspeech, prosodic elements of speech, localization/lateralization	Need for research in developing more tests; possibly random gap detection/forward and backward masking	Auditory decoding deficit

- Auditory brainstem and middle latency evoked responses to assess brainstem and cortical level integrity
- Puretone audiometry to evaluate the integrity of the peripheral hearing system
- Performance-intensity functions for word recognition ability
- A dichotic task consisting of dichotic words, dichotic digits, or dichotic sentences (assessing the communication between hemispheres)
- Duration pattern and a temporal gap detection test to assess temporal processing aspects of CAPD

The authors further state that the above tests recommended in the minimal test battery (MTB) are a "reasonable compromise" (Jerger and Musiek, 2000, p 472) of tests until research can provide analogous measures in the visual modality and neuroimaging results can be applied to the clinical utility of CAPD testing. Note that this model does not describe specific processing-related difficulties, but rather the goal is to ascertain whether CAPD is present.

Concerns were voiced about the MTB stating that a pure medical (diagnostic) model, as described in the Jerger and Musiek (2000) paper, would not delineate the CAP problems (Katz et al., 2002). Katz and colleagues pointed out that the tests lacked national CAP norms (at that time) and most had limited clinical use with the target population. In addition, the MTB did not address the educational concerns of children.

Bellis/Ferre Model

Initially, this model was called CATfiles (the CAT acronym stands for "categories" of CAPD) (Ferre, 1992). This was

later developed into a broader expansion of profiles (Bellis, 1999; Bellis and Ferre, 1999) with even further published changes (Bellis, 2002; Ferre, 2002), based on the Schow et al. (2000) criteria (Bellis, 2002). The current Bellis/Ferre CAPD subprofiles include three primary deficits—auditory decoding, prosodic, and integration—with secondary subprofiles that include associative deficit and organization-output deficit. The profiles are based on a conceptualization of the underlying neurophysiology in the CANS for encoding the auditory signal with the goal of identifying dysfunction in the left hemisphere, right hemisphere, and interhemispheric pathways. Bellis (2003) suggests that by examining the pattern of results across auditory processing functions, cognition, language, and learning, one can glean the underlying CAPD difficulties/profile.

The Bellis/Ferre profiles may be seen in isolation or together, with Bellis (2002) cautioning that one profile is typically primary in nature; however, another profile can be present because of possible overlap in the adjacent cortical structures. Electrophysiological tests are not used in the Bellis/Ferre model. Table 29.2 outlines this model, whereas the reader is referred to Bellis (2003) and Chapter 30 for an in-depth review of these profiles and for therapies. Following are descriptions of the various Bellis/Ferre CAPD subprofiles.

Auditory Decoding Deficit

According to Bellis (2003, p 291), auditory decoding deficit is possibly "the only true CAPD." This subprofile involves weak phonemic representations, poor discrimination and blending of sounds, and an inability to remember the learned phonemes. Determination of this profile is based on weaker right ear versus left ear test performance on low-redundancy speech and speech-in-noise tests (Bellis, 1996). Bellis (2002) describes the additional components of weak reading, vocabulary, and spelling skills, as well as concomitant behaviors such as auditory fatigue, and performance being improved with good visual perceptual skills. Site-of-lesion and electrophysiological research has suggested the primary auditory cortex within the left hemisphere as the probable site of dysfunction (Bellis, 1996). A later report (Bellis, 2002) found this deficit to be associated with diminished right ear/both ear performance on the Dichotic Digits Test (Musiek, 1983) (labeled a BI weakness) and the Competing Sentence Test (Willeford and Burleigh, 1985) (labeled a BS weakness).

Prosodic Deficit

Prosodic deficit is characterized by (1) difficulty in perceiving and recognizing nonverbal information, such as tonal patterns; (2) weak left ear test performance on dichotic tests showing weak BI and BS abilities; and (3) good speech-in-noise ability because of intact decoding ability (Bellis, 2002). Associated problems include weak singing ability (such as poor replication of melodies), poor social communication skills (i.e., difficulty understanding body language, facial gestures), flat voicing patterns, and diminished ability on visual-spatial tasks. Academic concerns involve weakness in mathematics, reading, sequencing, and spelling and poor sight-word abilities.

Integration Deficit

Integration deficit is characterized as a struggle involving interhemispheric skills, such as drawing, understanding dictation, dancing, and multimodal tasking (Bellis, 1996). Integration deficits may be the result of an immature corpus callosum or other structures related to the transfer of interhemispheric information. Auditory test results observed for this profile include weak left ear results on dichotic tasks and poor nonverbal test performance scores. Bellis (2002) further elaborates that BI and BS deficits are also often seen with this profile of CAPD, with weak sound localization abilities.

Secondary Profiles

Auditory Associative Deficit

The secondary profile known as an auditory associative deficit was observed in the original work of Bellis and Ferre (1999) as a CAPD profile, but it more recently has been classified as a secondary profile of CAPD (Ferre, 2002). This deficit consists of an inability to use rules of language with acoustic information, with the most severe cases replicating receptive childhood aphasia (Bellis, 1996). Performance on speech-sound discrimination tasks is normal; however, weak word recognition and dichotic test findings are observed bilaterally. Receptive language struggles are seen in vocabulary, semantics, and syntax. Inefficient communication between primary and associative cortical regions may be the causal aspect of this category (Bellis, 1996) and realized as significant auditory–language-processing difficulties (Ferre, 2002). The individual exhibits functional communication deficits when there is no specific-language impairment (Ferre, 2010).

Output-Organization Deficit

Another secondary profile is the output-organization deficit, which involves an inability to properly sequence, plan, and organize information (Bellis, 1996). Test performance requiring one simple response (e.g., monaural low-redundancy tests) will be good, whereas performance on tests with multiple components, such as those required on dichotic, frequency, or duration pattern tests, will be poor because of the use of complex stimuli (Bellis, 1996). Individuals with this type of deficit exhibit fine motor difficulties as well as sequencing and sound-blending errors. Reading comprehension is generally good for those who exhibit only this subprofile. At

the time this subprofile was proposed, the site of dysfunction for this category was not known, although an efferent (motor planning) hypothesis was proposed because of the weak skills observed on motoric tasks seen with this type of CAPD (Bellis, 1996).

Buffalo Model

This model, first reported in the early 1990s, consists of four CAPD subtypes (Katz, 1992; Katz and Smith, 1991). The Buffalo Model comprises of three tests, in which the Staggered Spondaic Word (SSW) test (Katz, 1962, 1968) is the center of the battery; the other two tests include the Phonemic Synthesis (PS) (Katz and Harmon, 1982) and Speech-in-Noise (Mueller et al., 1987) tests. This test battery provides 34 quantitative and qualitative indicators. Quantitative indicators are the number of errors seen in each of the three tests, whereas qualitative indicators refer to inter/intratest comparisons and the behavioral struggles seen during testing. Test results are compared to independent parent–teacher assessments (Katz and Zalweski, 2013) to determine if the test results relate to the same concerns of the family and school. The combination of the test performance indicators and academic and social behaviors (particular clusterings of each being associated with cortical anatomic sites) results in four CAPD subtypes that are not mutually exclusive: Decoding, tolerance-fading memory (TFM), integration, and organization (Katz, 1992, 2001; Katz and Smith, 1991). Clinicians may administer other tests (Medwetsky, 2011; Stecker, 1998) in addition to the Buffalo Model tests. See Table 29.3 for the test indicators for the Buffalo Model types of CAPD.

Decoding

Decoding has been described as the most common type, but it may not be quite as prevalent as it was in the late 1980s and early 1990s because the whole language approach is no longer used in the school system (Stecker, 2004) and more emphasis is being placed on phonemic awareness now (Tillery, 2005). The decoding type involves a breakdown at the phonemic level, causing a weakness in identifying, manipulating, and remembering phonemes. Weak oral reading or word accuracy and spelling skills are usually found in this subtype. Rapid incoming speech adds to the confusion of processing the message, and response delays are common because of the individual needing additional time to determine the verbal message. Weak discrimination and vocabulary result in the misperceptions of the heard auditory stimuli. Reported site of dysfunction for this category is the phonemic zone (Luria, 1966) of the left posterior temporal lobe, also known as the auditory cortex. Test results associated with this subtype include weak SSW right competing (RC) and left noncompeting (LNC) scores and poor Phonemic Synthesis results (Katz, 1992). Qualitative signs include delayed responses, nonfused answers, and quiet rehearsals (described in Table 29.3).

TABLE 29.3

Qualitative and Quantitative Test Indicators of Buffalo Model Central Auditory Processing Disorders (CAPD) Types

CAPD Types	Primary Indicators			Secondary Indicators			Qualifying Indicators		
	SSW	PS	SN	SSW	PS	SN	SSW	PS	SN
Decoding	RC errors LNC error Order L/H Ear H/L	Below normal	Mild		Nonfused, quiet rehearsals, delays		Delays Perseverations Smush	Delays Perseverations O/L	Mild/moderate in poorer ear
TFM	Order H/L Ear L/H		Moderate or severe in poorer ear	LC errors			Quick AYR/Y TTW Smush	Omission error on first sounds	Moderate in poorer ear
Integration	Type A			Sharp LC peak of errors	May be severe score		Extreme delays		
Organization	Significant Reversals	Significant Reversals							

Abbreviations: SSW, Staggered Spondaic Word Test; PS, Phonemic Synthesis Test; SN, Speech-in-Noise Test (Katz, 2001a); RC, right competing; LC, left competing; LNC, left noncompeting; H/L, high/low; L/H, low/high; O/L, whereby client produces an /o/ sound for the /l/ sound; TFM, tolerance-fading memory; AYR, "are you ready" response; Y, "yes" response; TTW, tongue twister.

Tolerance-Fading Memory

This CAPD subtype has been considered the second most common in the general population (Katz, 1992). The theorized loci involve the frontal lobes and the anterior temporal region, which houses the hippocampus and amygdala, and are associated with memory and the limbic system (Katz, 1992; Isaacson and Pribram, 1986). Functional behavioral limitations include a weak short-term auditory memory and difficulty hearing auditory information in the presence of noise (the *tolerance* aspect of TFM), that is, individuals with TFM may exhibit significantly increased difficulty tolerating and understanding in noise as compared to individuals with other types. Other limitations associated with frontal lobe dysfunction include expressive language and difficulty inhibiting impulsive responses. Qualitative signs include quick responses, smush responses (combining the competing words of an item into a single word, e.g., "sea *shore out*side" = "sea *shout* side"), an inability to refrain from repeating carrier phrases ("Are you ready?"), and omission or errors on the first word (or the omission of the first sound on the PS test). Individuals with attention deficit/hyperactivity disorder (ADHD) are commonly found to exhibit TFM (Keller and Tillery, 2002, 2014), probably because of the close association of the frontal and the anterior temporal lobes (Katz and Smith, 1991). The frontal lobe houses executive function that serves to regulate and coordinate behaviors to the environment, inhibits irrelevant responses, and oversees cognitive processes (Barkley, 1998), which are affected by ADHD. Recent studies support the close association between short-term auditory memory and speech-in-noise difficulties (Brannstrom et al., 2012; Yathiraj and Maggu, 2013).

Integration

The integration category is considered the most severe type of CAPD. Earlier integration was divided into two subtypes, but in time it became clear that each category needed to be addressed equally and so it was unnecessary to have this division. Generally, the more severe integration case probably involves the posterior corpus callosum and/or the angular gyrus of the parietal-occipital region, which are regions thought to be associated with dyslexia (Geschwind and Galaburda, 1987). The integration problems that likely involve more anterior regions of the corpus callosum tend to be somewhat less severe. An integration sign is said to be present when one displays a type-A SSW pattern, that is, a severe peak of errors usually in one particular column of the eight columns on the SSW test response form (column F, a left competing condition). Type-A indicates difficulty in transferring interhemispheric information. To determine the likely behavioral impact of the type-A, one needs to look at the rest of the test battery findings and the Buffalo Model Questionnaire—Revised (Katz and Zalweski, 2013). In addition, a qualitative sign in those with integration difficulties includes extremely long response times on the SSW items that are generally seen in daily life activities as well. Functional behavioral limitations include severe reading and spelling problems and difficulty in integrating visual and auditory information, and they are often diagnosed with dyslexia. Integration is often more resistant to therapeutic intervention therapy than the other three categories.

Organization

This CAPD subtype was first reported by Lucker (1981), who recognized that reversals on the SSW test are observed in individuals who are disorganized. A reversal is said to occur when stimuli (i.e., words, sounds) are repeated out of sequence. Both the SSW and Phonemic Synthesis tests have norms for determining the presence of a significant number of reversals (ages 5 or 6 years to 69). Reversals are considered a more anterior sign (Katz, 1992). Note that those with attention disorders tend to exhibit weak organization, planning, and sequencing, all of which are associated with dysfunction in the Rolandic region (Luria, 1970, Efron, 1963). Indeed, Tillery (1999) found SSW reversals to be very common in her studies of children with ADHD.

Spoken-Language–Processing Model

This model, developed by colleagues at Rochester Speech and Hearing Clinic, New York, expands on the Buffalo Model to include a broader perspective beyond auditory processing to better understand how one perceives and processes spoken language. Medwetsky (2011) considers auditory processing to be a component of spoken-language–processing (S-LP) and limited to those perceptual mechanisms involved in the initial acoustic analysis of the incoming signal. Table 29.4 shows a summary of the S-LP Model. The CAPD diagnosis may result in the following areas of concern: Lexical decoding, fading memory, auditory-linguistic integration, sequencing, short-term memory span, prosodic perception, attention and phonologic problems.

ELECTROPHYSIOLOGICAL MEASURES AND A CENTRAL AUDITORY PROCESSING TEST BATTERY?

The proposed MTB indicated a need for electrophysiological testing that the proposers felt should be included in all CAPD test batteries (Jerger and Musiek, 2000). This recommendation was based on the fact that CANS neural synchrony in response to auditory stimuli is assessed through the application of a number of electrophysiological procedures, including auditory brainstem response (ABR), middle latency response (MLR), mismatch negativity (MMN), and late evoked potentials (LEP), including P300. However, an

TABLE 29.4

Processes Assessed through the Spoken-Language Processing (S-LP) Model (Medwetsky, 2011)

Process	Definition	Test
Temporal resolution	Ability to detect rapid changes in the speech signal	Random Gap Detection Test; Gaps-in-Noise Test
Lexical decoding speed	Ability to process words quickly and accurately	Staggered Spondaic Word (SSW) Test–Decoding Signs
Short-term memory (STM)/ working memory	Severity/patterns of how information is maintained in conscious memory (i.e., initial vs. later presented information)	SSW Test–Fading Memory Signs
STM/working memory span	Amount of units/information retained in STM	Test of Auditory Perceptual Skills–Revised: 1. Auditory Number Memory–Forward; 2. Auditory Word Memory; 3. Auditory Sentence Memory
Sequencing	Ability to maintain speech sounds, words, and directions in order	SSW Test (organization), Phonemic Synthesis Test (reversals), Token Test, Pitch Pattern Sequences Test
Auditory-linguistic integration	Ability to integrate information (supra-segmental/visual/verbal) across processing regions	1. Digit Span–Rhythm Task; 2. SSW Test–Integration Sign; 3. Competing Sentences Test–right ear dominance; 4. Pitch Pattern Sequences Test (non-verbal/verbal discrepancy)
Prosodic perception	Ability to perceive/replicate rhythmic patterns	Pitch Pattern Sequences Test (significant nonverbal sign) + flat voicing patterns
Selective auditory attention	Ability to focus and recall target stimuli in presence of competition	Figure-ground tests (i.e., speech embedded in noise) and binaural separation such as on Competing Sentences Test
Divided auditory attention	Ability to recall both competing stimuli presented	SSW Test, Competing Sentences Test, Competing Words from Screening Test for Auditory Processing Disorders (SCAN)/SCAN–Revised
Sustained auditory attention	Ability to maintain attention to verbally presented information over a period of time without a break	Auditory Continuous Performance Test
Higher order phonologic skills		
Phonemic synthesis	Ability to blend individually presented speech sounds and derive the target whole word	Phonemic Synthesis Test
Sound–symbol associations	Ability to discriminate/sequence/represent speech sounds with symbols	Lindamood Auditory Conceptualization Test 3

abnormality of the CANS determined through electrophysiological measures does not provide specific information as to the type of CAPD or auditory behavioral deficits that can be expected based on the results obtained. That is, although electrophysiological tests may show clinical utility in assessing the CANS (Jirsa, 2002), there is a paucity of research in understanding the abnormalities of these tests relative to the presence of learning disabilities (Cacace and McFarland, 2002). For example, clear relationships have not yet been found between the auditory behavioral limitations observed in individuals suspected of having CAPD and neural dyssynchrony ascertained via electrophysiological measures. In addition, research has revealed little evidence of an increased prevalence of abnormal ABRs or MLRs to click stimuli/tone

bursts in CAPD populations. Furthermore, it is questionable as to what information can be provided with application of traditional electrophysiological testing when providing intervention recommendations (AAA, 2010; Bellis, 2003). Obviously, electrophysiological tests control for attention, fatigue, and motivation influences when assessing the CANS, even though these areas can usually be identified and controlled for during behavior tests (Bellis, 2003; Katz and Tillery, 2005).

Some recent studies have investigated the application of electrophysiological procedures to determine clinical utility in a CAPD diagnosis. For example, MMN has been found to (1) verify neurophysiological changes because of listening training that may accompany observable auditory behaviors (Tremblay et al., 1997; Tremblay, 2007); (2) assist in differentiating phonemic (low) levels and language (higher) levels during auditory processing (Dalebout and Stack, 1999); and (3) differentiate children with and without learning problems (Banai et al., 2005). It has also been suggested that LEP measures can (1) differentiate attention disorders from other problems (Kraus et al., 1995); (2) show increased latency and decreased amplitude on P300 for children with APD when compared to those without APD (Jirsa and Clontz, 1990); (3) be used to study developmental processes in children and adults with hyperactivity (Satterfield et al., 1984); and (4) examine children with language/speech disorders (Mason and Mellor, 1984).

However, the most impressive research to date concerning the use of electrophysiological procedures and speech processing comes from Krause and colleagues (Kraus and Chandrasekaran, 2011; Kraus and Nicol, 2005; Russo et al., 2005). Please refer to Chapter 28, and Krause and Hornickel (2013).

CENTRAL AUDITORY PROCESSING SCREENING

CAP screening assesses the possibility of existence of a CAPD and, in turn, can lead to possible referral for a comprehensive CAPD evaluation. Psychologists and speech-language pathologists are two professional groups that would likely screen for CAP on a routine basis. As part of the screening process, teachers and parents may be asked to provide information on the child's behavioral functional limitations through the use of questionnaires.

Questionnaires

Questionnaires are a common tool for ascertaining the likelihood that an individual exhibits functional behavioral limitations in his/her communication, language, and learning. Because of possible bias, we must take into consideration who is rating the child's behaviors on the questionnaire. A teacher may give ratings that indicate weak attention or motivation of the student as being the possible reason for "poor listening." However, CANS tests may indicate that CAPD is associated with the student's "listening difficulty." On the other hand, parents may insist that their child has a CAPD and reflect this bias on the questionnaire ratings for their child to receive preferential services or a referral for testing. Following is a list of questionnaires which are available through the Educational Audiology Association:

1. Fisher Auditory Problems Checklist (Fisher, 1985). This was the first developed CAP screening questionnaire, with normative data available from kindergarten to grade 6. It has been designed to rate 25 items of concern. Many of the items listed on this questionnaire are commonly used in other CAPD profiles.
2. Children's Auditory Processing Performance Scale (CHAPPS) (Smoski et al., 1992). There are six listening situations (ideal, quiet, attention, memory, noise, and multiple inputs), and the rater (parent or teacher) compares the student to children of similar age and background. There are a total of 36 questions, and the choices vary from +1 (less difficulty than others) to −5 (cannot function in the situation). Scores can range from +36 to −180, and the more negative the score, the more difficulty that is noted. A child who receives a total score of −12 to −180 is at risk for CAPD.
3. Screening Instrument for Targeting Educational Risk (SIFTER) (Anderson, 1989). There are 15 questions over five category areas: Communication, academics, attention, class participation, and social behavior. Scoring consists of 15 points per category, resulting in a failure if one is rated at or below 6 or 7 (depending on the category).
4. Buffalo Model Questionnaire—Revised (BMQ-R) (Katz and Zalweski, 2013). The questionnaire contains 39 questions dealing with CAPD including categories/subcategories: Decoding, Noise, Memory, Various TFM, Integration, Organization, and General (more than one category). Data are provided for three age groups (<6, 6 to 18, >18) with 122 controls and 213 who have CAPD. The characteristics that were most common were for Decoding (understands oral directions) and Memory (remembers oral directions), both of which had 79% hit rates in the CAPD group. BMQ-R is useful prior to the evaluation, following the evaluation to compare with the test findings, before therapy, and independently validating the progress in therapy.

Screening Tests

Historically, screening test performance scores have sometimes been used to label a child with CAPD, rather than to refer the child for further testing (Jerger and Musiek, 2000) by an audiologist to rule in or out the diagnosis of CAPD. In general, screening tests have been designed to have high

sensitivity (Jerger and Musiek, 2000) (i.e., those having CAPD are readily identified); however, this can also lead to a high false-positive rate (i.e., identify individuals as possibly having CAPD when, in fact, they do not).

Obviously, attention, fatigue, and status of an unchecked peripheral hearing system can influence screening test findings. It is recommended that screening tests be administered in a room without any noise distractions and during the morning to control for attention and fatigue. When possible, screening tympanometry and puretone thresholds should be obtained to improve the reliability of the screening results. It is essential to obtain a thorough case history of the child or adult prior to the evaluation for medical, academic, and functional behavioral deficit information. We need to understand the individual's problems that may be related to CAPD (AAA, 2010). The following is a description of some of the CAP screening tests available:

1. The original Screening Test for Auditory Processing Disorders (SCAN) (Keith, 1986) developed over the years. Currently, for ages 5 to 12 years the SCAN-3 for Children: Tests for Auditory Processing Disorders (SCAN-3:C) (Keith, 2009b) and for ages 13 to 50 years, the SCAN-3 for Adolescents and Adults: Tests for Auditory Processing Disorders (SCAN 3:A) (Keith, 2009a) are used. Both SCAN protocols contain three screening measures (Gap Detection, Auditory Figure-Ground, Competing Words), four diagnostic tests (Filtered Words, Auditory Figure-Ground, Competing Words, Competing Sentences), and three supplementary tests (Auditory Figure-Ground at +12 dB SNR and at 0 dB SNR, Time-Compressed Sentences).

   Although psychologists and speech-language pathologists typically administer the SCAN series as a screening tool for CAPD, the inclusion of the diagnostic portion of the SCAN-3:C or SCAN-3:A offers audiologists to use these instruments as a portion of their test battery. A cassette player, headphones, and a quiet environment are necessary to administer these screening procedures. I have consulted and advised psychologists to administer the SCAN in the morning and that it be the first test in their test battery to control for fatigue and attention; otherwise, fatigue and inattention could influence the occurrence of false-positive test results.
2. The Differential Screening Test for Processing (DSTP) (Richard and Ferre, 2006) was developed to differentiate skills associated with three neurologic levels of processing that are integrated depending on the communication task: (1) Perception of primary acoustic characteristics of auditory signals; (2) identification of acoustic aspects related to the phonemic portion of language; and (3) the ability to attribute meaning to language.

   The authors indicate that the first level is evaluated by tests that target (a) the ability to discriminate speech sounds (auditory discrimination); (b) binaural integration in which the client is asked to repeat numbers presented dichotically to assess communication between hemispheres, and (c) the ability to recognize acoustic patterns found in verbal communication (temporal patterning) by verbally indicating the sequence of the two presented tones (high and/or low pitched, such as high–high or low–high.

   The second level is evaluated by using two subtests: "Phonemic" manipulation and "phonic" manipulation. Phonemic manipulation provides two to four sounds in which the child must properly recognize (a) the number of discrete phonemes in a provided word, (b) blend the sounds into a word, and (c) change discrete sounds when asked. Phonic manipulation assesses sound–symbol associations by providing three tasks that target (a) proper spelling with supplied tiles, (b) the ability to synthesize phonemes with the use of tiles, and (c) the ability to modify the tile representation when provided a new target word.

   The third level assesses meaning to the auditory signal by providing three subtests: Antonyms, prosodic interpretation, and language organization. To assess antonym knowledge, the child must provide the opposite word to the provided target word. To assess prosodic interpretation, the child verbally responds with a "yes" or "no" to the sincerity of the message. For instance, the phrase "I am happy to be here" is provided in a sad tone of voice. The child would respond "no" because there is a discrepancy between the prosodic information and the provided statement. To assess language organization, the child must respond successfully to two different tasks. Task 1 provides eight different sentences, such as, "It's what you sit on at a table or a desk." The proper answer is chair, stool, or seat. For task 2, the child is provided pictures of objects and must describe the objects or what the objects do. For instance, a picture of a flower may be provided. The proper response can be any of the following: Smells good, attracts bees, blooms, grows in a garden, has pollen, and so on.

   The DSTP was standardized by presenting the subtests to 509 students aged 6.0 to 12.11 years old, reflecting a balance across race, age, gender, grade, and all socioeconomic groups. Poor test performance in any area suggests the need for additional diagnostic evaluation(s) to establish the presence of a deficit.
3. The Auditory Skills Assessment (ASA) (Geffner and Goldman, 2010) is the first tool developed to assess the skills of young children (3.6 to 6.11 years) as CAPD weaknesses, most certainly, influence language and academic skills.

   The ASA consists of six subtests: Speech discrimination in noise (+6 dB SNR, mimicry (repeat a nonsense word), blending (morphemes or phonemes are given first with a visual cue followed with no visual cue), rhyming

awareness, tonal discrimination (ability to distinguish same or different musical instrument notes), and tonal patterning (points to the picture of a piano or an oboe to indicate which sound was heard last).

The ASA was standardized by analyses of data from 475 children aged 3.6 to 6.11 years. Reading, language, and learning issues can result from a deficit found on the above ASA measures. Thus early identification coupled with intervention will assist this young population.

4. There are other possible screening tools. Speech-language pathologists routinely use the Test of Auditory Perceptual Skills—Revised (TAPS-R; Gardner, 1996), whereas psychologists typically use some form of digit span test (Wechsler, 1991) or the Visual-Aural Digit Span Test (Koppitz, 1975). Bellis (2003) indicates that the TAPS-R may be an instrument that can provide some indication of auditory perceptual ability, but it does not indicate the specific underlying auditory processing difficulties. Keller et al. (2006) found a correlation with test performance on digit span (Wechsler, 1991) and CAPD. This indicates that psychologists should refer individuals for a CAPD evaluation when a client shows weakness on tests sensitive to short-term auditory memory span.

   The Dichotic Digit Test (DDT) (Musiek, 1983), administering two digits per ear, may also be a useful CAPD screening tool because it is a very quick test to administer (4-minute task) and uses very familiar items (digits) that even young children will readily recognize (Jerger and Musiek, 2000).

Combined screening measures may assist in minimizing over-referrals because of high false-positive findings (Jerger and Musiek, 2000). For example, this can be accomplished by using the combination of a questionnaire and CAP screening test measure. Another possibility posed by Jerger and Musiek (2000) is to administer both the DDT and a gap detection test; however, the authors stress the need for research to assess this possibility as a screening measure.

CENTRAL AUDITORY PROCESSING DISORDER TESTS

Auditory tasks administered to assess for auditory processing function consist of monotic (stimuli presented separately to each ear), diotic (same stimuli presented to both ears simultaneously), and dichotic (different stimuli presented to each ear simultaneously) tests. Audiologists generally choose their tests by the processes they wish to assess. Refer to the CAPD Test Battery Model discussed in earlier sections of this chapter. Table 29.1 lists CAP tests with their associated targeted process and CANS sensitivity, whereas Table 29.2 defines the function assessed by the CAP tests. The reader is referred to the previous cited test battery publications and the more recent publications that provide a thorough description of the CAP tests seen in the various models (Chermak and Musiek, 2014; Geffner and Ross-Swain, 2013; Musiek and Chermak, 2014).

REPORTING CENTRAL AUDITORY PROCESSING DISORDER TEST RESULTS

An evaluation report must be accurate, concise, and well written. These reports communicate to families and professionals (such as physicians, speech-language pathologists, teachers, tutors, occupational and physical therapists, and psychologists) an explanation of the various test battery procedures, test performance results, and recommendations for remediation or compensations for the disorder. Professionals appreciate reports that are organized, consistent in format style, and provide details on the test performance data; in turn, this allows them to know exactly where to find a specific summary or fact, thus saving them time and effort. The reports should provide the raw scores, number of standard deviations (SD), and explanation of findings in terms that are understood by all those who read the report. When applicable, information should include both qualitative and quantitative results, severity of findings, overall implications (e.g., comorbidity associations, educational and medical aspects), and resources for the reader to consult. Reports should be sent within a reasonable time frame.

In summary, the report is likely the best opportunity to educate others about the diagnosis of CAPD; facts regarding the administered test battery; social, medical, and educational ramifications; and recommendations for assisting with the client's auditory, learning, and communicative functional behavioral limitations.

THIRD-PARTY REIMBURSEMENT

The ASHA (2005) CAPD Technical Report is the first publication to provide information on how to submit for payment for these evaluations. Perhaps this is because current procedural terminology (CPT) codes implemented in January 2005 for the first time reflected the professional time and services provided in a CAPD evaluation. The AAA (2010) document provides detailed information on reimbursement, including counseling and report writing. The first hour of administering, interpreting, and/or providing test results falls under the CPT code 92620, with each additional 15-minute increment to be billed under code 92621. These codes do not include the peripheral hearing assessment, which is billed under each individual peripheral hearing measurement administered. There are differences in insurance reimbursement seen in the United States and other countries. In the United States, the audiologist should

be well informed of what procedures are covered by the third-party insurers. Families are unaware of the differences among insurers' policies and rely on the professional to be informed.

In the past, reimbursement involved submitting for each central test administered, each with its own CPT code. Such billing was frustrating because some CAP tests would only allow a $3.00 reimbursement for a test that took 15 minutes to administer, whereas others provided a $25.00 reimbursement for a 10-minute test. Another billing problem in the past was that speech-language pathologists and audiologists had to bill for language and/or auditory processing evaluations under a single CPT code. Such procedures led to confusion and misrepresented CAPD test assessment. To reconcile this billing dilemma, improved reimbursement procedures were developed for CAP assessment, which ultimately led to the new CPT codes. In October 2014, the United States will be using new diagnostic codes. The codes for a CAPD diagnosis include four new codes: One for a left ear CAPD deficit, a right ear CAPD deficit, both, or nonspecified.

Reimbursement Concerns

Insurance companies are not obligated to reimburse for testing, intervention, and report writing that fall under an educational-related diagnosis or experimental applications. Some insurance companies indicate that CAPD is related to educational factors (Excellus, 2002) and, therefore, is not covered, even under the diagnostic code 388.40: Abnormal Auditory Perception. When an educational-based reason is used as a reason for denial of payment of service or when an insurance company has outdated information and claims that CAPD is experimental (Aetna, 2012), information should be provided to these insurance companies that includes the most current up-to-date facts, such as studies showing the clinical utility of CAPD testing. During this interim period, the clinician would submit for payment to the insurance company for the peripheral and central assessment procedures. The client will be responsible for payment of any uncovered assessment measures.

Another concern is the need for evidenced-based research to address the types or subprofiles of CAPD in terms of both medical and educational outcomes. Insurance companies rely on evidence-based research and technical reports to justify medical needs for services rendered. At the present time, all CAPD models indicate some form of educational basis: Poor reading and spelling, weak organization, poor or inconsistent academic performance, weak expressive language written skill, and so on. If this continues to be stressed in the models of CAPD, without the medical counterpart, then insurance companies may prematurely conclude that there are only educational components of CAPD and thus not realize the medical concerns. In turn, this will continue to result in denials of reimbursement for services associated with the current diagnostic code 388.40 Abnormal Auditory Perception.

As professionals, we are obligated to provide evidence-based research regarding areas of concern related to differential diagnosis to indicate a medical need for testing and application of intervention. Differential diagnosis involves collaboration with the psychologist, speech-language pathologist, audiologist, and possibly the physician. The end result may be a child with only ADHD who may need medication to assist with the functional behavioral limitations associated with ADHD (Keller and Tillery, 2014; Tillery, 2013). However, the auditory problems of a child with CAPD alone will not improve with medication (Tillery et al., 2000). The child with both CAPD and ADHD will need a variety of therapeutic measures to assist ADHD (i.e., medication, tutoring, behavioral modification, counseling) and unique therapeutic measures for CAPD. This example illustrates the concept of "win–win," with both the client and insurance company benefiting from the CAP evaluation and recommendations. The insurance company will not have to provide coverage for medication for someone with a diagnosis of CAPD (which could cost the insurance company thousands of dollars over the course of many years), whereas the client hopefully will obtain the treatment that will best meet his or her needs.

FUTURE CONCERNS IN AUDITORY PROCESSING TEST BATTERIES

The selection of CAP tests or a test battery approach relies on the comfort, experience, and education of the clinician, as well as the availability of a multidisciplinary team in the geographic area in which one resides. Consensus and position papers (AAA, 2010; ASHA, 2005; Canadian Guidelines, 2012) recommend that testing be done for children 7 years of age and older. However, the Buffalo and S-LP models provide qualitative data congruent with quantitative data for children as young as 5 years of age. Such testing can result in the categorization of types of auditory processing problems, initiation of therapy, and addressing educational and communication concerns before major problems occur. Hopefully, in time, there will be a general understanding that the earlier one is diagnosed the better the opportunity to provide appropriate intervention. The identification of dysfunction among specific auditory processes is the basis of the Bellis/Ferre Model and provides specific categories of auditory problems that coincide with educational and communication concerns. Some clinicians broaden these models. For instance, Medwetsky's S-LP Model (2011) uses the qualitative and quantitative data of the Buffalo Model as

a foundation and further includes attention, memory span, and phonologic awareness/phonics test performance for further analysis (Medwetsky, 2011). Stecker (1998) discusses the application of additional tests to assess localization and/or low brainstem assessment beyond the Buffalo Model. Those who work with psychologists in a team approach may not need to administer attention tests such as the Auditory Continuous Performance Test (Keith, 1994) since it is routinely administered by the referring psychologist, as is the case in Western New York.

SUMMARY

A test battery approach is recommended for the assessment of the CANS when a client presents with functional behavioral limitations in auditory, learning, and communication skills. Currently, evaluating the CANS is not a routine application of assessment among audiologists; however, it is hopeful that this will soon change with the educational opportunities offered by Au.D. programs. Research should concentrate on the application and results of CAP tests that indicate specific types of CAPD, as seen in the current models discussed which would lead to appropriate intervention. The past decade has focused on improving our awareness of the reliability of CAP test performance and the success in remediating the functional deficits associated with CAPD. The next decade should focus on improved normative data for using electrophysiological tests in certain populations with attention deficit, dyslexia, and the subtypes of CAPD. These procedures would augment current behavioral test batteries by providing objective evidence of underlying processing deficits, help determine auditory training candidacy, and, in turn, continue to evaluate the effectiveness of such therapy.

FOOD FOR THOUGHT

Perhaps in the future, there will be additional evidence-based research to validate the specific types of CAPD (ASHA, 2005). In the meantime, clinicians will continue to administer CAPD tests that are known to provide information regarding CANS dysfunction. As we work together to learn the "best of the best" in diagnosing CAPD, here are some thoughts we should be aware of and try to answer:

1. Can one test provide a diagnosis of CAPD? Although position statements indicate to be aware of the number of tests in a test battery, ASHA (2005) indicates that one test failure at 3 standard deviations below the mean or a failure of two tests by a minimum of 2 standard deviations below the mean is sufficient for a diagnosis of CAPD in the presence of functional behavioral limitations. The use of one test failure was considered to be a lax approach, but most would agree that such a failure constitutes a dysfunction in only the specific auditory process being assessed. If the clinician controls for attention, motivation, and fatigue, then a failure of two tests at a minimum of 2 standard deviations below the mean or one test failure at 3 standard deviations below the mean would seem appropriate for the profession to consider as a criterion for the diagnosis of CAPD.
2. Please discuss what advantages you see in providing a test battery approach. Based on what you know about CAPD give some examples of how the test battery approach would be advantageous?
3. The various models for types or profiles of CAPD have many commonalities and a few differences. Actually, it is interesting that there are more similarities than differences. All of the models agree on decoding and integration subtypes of CAPD. The TFM type of CAPD is seen in the Buffalo Model, and FM is seen in the S-LP Model, whereas the prosodic category is only in the Bellis/Ferre and S-LP Models. Intervention is being used to successfully remediate the above CAPD subtypes. Tests have been found to identify auditory difficulties for designing intervention (AAA, 2010).

   There is agreement with respect to the organization category among the different models. However, only one CAP test (the SSW test) provides norms for reversals. What does it mean when one reverses on other tests, especially if those tests were developed to identify those with learning disabilities, such as the Pitch Pattern Sequence Test? What does it mean when an individual passes all the CAP tests with the exception with reversals? Is there a CAPD in this case or are the reversals related to attention deficit or learning disorders?
4. Would the inclusion of electrophysiological tests assist with profiling specific types of CAPD?

Questionnaires listed in this chapter are available at Education Audiology Association (EAA): www.edaud.org.

KEY REFERENCES

thePoint A full list of references for this chapter can be found at http://thePoint.lww.com. Below are the key references for this chapter.

Aetna. (2012) Clinical policy bulletins. Auditory processing disorder (APD). No. 668, 1–9.

American Academy of Audiology (AAA). (2010) Clinical practice guidelines: diagnosis, treatment and management of children and adults with central auditory processing disorder. Available online at: www.audiology.org

American Speech-Language-Hearing Association (ASHA). (1992) *Issues in Central Auditory Processing Disorders: A Report from ASHA Ad Hoc Committee on Central Auditory Processing.* Rockville, MD: ASHA.

American Speech-Language-Hearing Association (ASHA). (1995) *Central Auditory Processing: Current Status of Research and Implications for Clinical Practice. A Report from the ASHA Task Force on Central Auditory Processing.* Rockville, MD: ASHA.

American Speech-Language-Hearing Association (ASHA) Task Force on Central Auditory Processing Consensus Development. (1996) Central auditory processing: current status of research and implications for clinical practice. *Am J Audiol.* 5, 41–54.

American Speech-Language and Hearing Association (ASHA). (2005) *(Central) Auditory Processing Disorders. A Technical Report.* Rockville, MD: ASHA.

Bellis TJ. (2002) Developing deficit-specific intervention plans for individuals with auditory processing disorders. *Semin Hear.* 23, 287–295.

Bellis TJ. (2003) *Assessment and Management of Central Auditory Processing Disorders in the Educational Setting: From Science to Practice.* 2nd ed. Clifton Park, NY: Thompson Learning.

Brannstrom KJ, Zunic E, Borovac A, Ibertsson T. (2012) Acceptance of background noise, working memory capacity, and auditory evoked potentials in subjects with normal hearing. *J Am Acad Audiol.* 23 (7), 542–552.

British Society of Audiology. (2011) Position statement: auditory processing disorders. Available online at: http://www.thebsa.org.uk/docs/docsfromold/BSA_APD_PositionPaper_31March11_FINAL.pdf

Cacace AT, McFarland DJ. (2002) Middle-latency auditory evoked potentials: basic issues and potential implications. In: Katz J, ed. *Handbook of Clinical Audiology.* 5th ed. Philadelphia, PA: Lippincott Williams & Wilkins; pp 349–377.

Cacace AT, McFarland DJ. (2005) The importance of modality specificity in diagnosing central auditory processing disorder (CAPD). *Am J Audiol.* 14, 112–123.

Canadian Guidelines on Auditory Processing Disorders in Children and Adults: assessment and intervention. (2012) Available online at: www.speechandhearing.ca

Chermak GD, Musiek FE. (2014) *Handbook of Central Auditory Processing Disorder; Comprehensive Intervention.* Vol. 2. 2nd ed. San Diego, CA: Plural Publishing, Inc.

Ferre JM. (2002) Managing children's central auditory processing deficits in the real world: what teachers and parents want to know. *Semin Hear.* 23, 319–326.

Ferre JM. (2010) Aural rehabilitation & central auditory processing disorders (CAPD): outcome evidence among school-age children. *Educ Audiol Rev.* 27, 8–17.

Geffner D, Goldman R. (2010) *Auditory Skills Assessment (ASA).* San Antonio, TX: Psychological Corporation.

Geffner D, Ross-Swain D. (2013) *Auditory Processing Disorders: Assessment, Management, and Treatment.* 2nd ed. San Diego, CA: Plural Publishing.

Jerger J, Musiek FE. (2000) Report of Consensus Conference on the Diagnosis of Auditory Processing Disorders in School-Aged Children. *J Acad Audiol.* 11, 467–474.

Katz J. (1994) *CAPD Test Battery.* Vancouver, WA: Precision Acoustics.

Katz J. (2001) *Central Test Battery: Tester's Manual.* Vancouver, WA: Precision Acoustics.

Katz J, Smith PA. (1991) A ten minute look at the CNS through the ears: using the SSW test. In: Zappulla R, LeFever FF, Jaeger J, Bildern R, eds. *Windows on the Brain: Neuropsychology's Technical Frontiers. Ann N Y Acad Sci.* 620, 233–252.

Katz J, Tillery KL. (2005) Can central auditory processing tests resist supramodal influences? *Am J Audiol.* 14, 124–127.

Katz J, Zalweski T. (2013) *Buffalo Model Questionnaire – Revised (BMQ-R).* Denver, CO: Educational Audiology Association.

Keith RW. (2009a) *SCAN 3:A: for Adolescents and Adults: Tests for Auditory Processing Disorders.* San Antonio, TX: The Psychological Corporation.

Keith RW. (2009b) *SCAN 3:C: for Children: Tests for Auditory Processing Disorders.* San Antonio, TX: The Psychological Corporation.

Keller W, Tillery KL. (2002) Reliable differential diagnosis and effective management for auditory processing and attention deficit hyperactivity disorders. *Semin Hear.* 23, 337–347.

Keller W, Tillery KL. (2014) Central auditory processing disorder and attention deficit hyperactivity disorder: a psychological perspective on intervention. In: Chermak G, Musiek F, eds. *Handbook of Central Auditory Processing Disorders, Volume II: Comprehensive Intervention.* 2nd ed. San Diego, CA: Plural Publishing Inc.; pp 571–587.

Keller W, Tillery KL, McFadden S. (2006) Auditory processing disorder in children diagnosed with nonverbal learning disability. *Am J Audiol.* 15, 108–113.

Kraus N, Chandrasekaran B. (2011) Music training for development of auditory skills. *Nat Rev Neurosci.* 11, 599–605.

Kraus N, Hornickel J. (2013) cABR: a biological probe of auditory processing. In: Geffner D, Ross-Swain D, eds. *Auditory Processing Disorders: Assessment, Management, and Treatment.* 2nd ed. San Diego, CA: Plural Publishing.

Kraus N, Nicol T. (2005) Brainstem origins for cortical 'what' and 'where' pathways in the auditory system. *Trends Neurosci.* 28, 176–181.

Medwetsky L. (2011) Spoken language processing model: bridging auditory and language processing to guide assessment and intervention. *Lang Speech Hear Serv Sch.* 42, 286–296.

Moore DR, Rosen S, Bamiou DE, Campbell NG, Sirimanna T. (2013) Evolving concepts of developmental auditory processing disorder (APD): a British Society of Audiology APD special interest group 'white paper'. *Int J Audiol.* 52, 3–13.

Musiek FE, Chermak GD. (2014) *Handbook of Central Auditory Processing Disorders, Volume I: Auditory Neuroscience and Diagnosis.* 2nd ed. San Diego, CA: Plural Publishing.

Rawool VW. (2013) Temporal processing in the auditory system. In: Geffner D, Ross-Swain D, eds. *Auditory Processing Disorders: Assessment, Management, and Treatment.* 2nd ed. San Diego, CA: Plural Publishing; pp 227–249.

Richard GJ, Ferre JM. (2006) *Differential Screening Test for Processing.* East Moline, IL: LinguiSystems, Inc.

Russo NM, Nicol GT, Zecker SG, Hayes EA, Kraus N. (2005) Auditory training improves neural timing in the human brainstem. *Behav Brain Res.* 156, 95–103.

Schow R, Seikel J, Chermak G, Berent M. (2000) Central auditory processes and test measures: ASHA 1996 revisited. *Am J Audiol.* 9, 1–6.

Tillery KL. (2005) CAPD characteristics in a large sample with and without CAPD. Paper presented at the American Speech-Language-Hearing Association (ASHA) Annual Convention, San Diego, CA.

Tillery KL. (2013) Use of medication with auditory processing disorders. In: Geffner D, Ross-Swain D, eds. *Auditory Processing Disorders: Assessment, Management, and Treatment.* 2nd ed. San Diego, CA: Plural Publishing; pp 719–729.

Tillery KL, Katz J, Keller W. (2000) Effects of methylphenidate (Ritalin) on auditory performance in children with attention and auditory processing disorders. *J Speech Lang Hear Res.* 43, 893–901.

Tremblay K. (2007) Training-related changes in the brain: evidence from human-auditory-evoked potentials. *Semin Hear.* 28, 120–132.

Weihing J, Bellis TJ, Chermak GD, Musiek FE. (2013) Current issues in the diagnosis and treatment of CAPD in children. In: Geffenr D, Ross-Swain D, eds. *Auditory Processing Disorders: Assessment, Management and Treatment.* 2nd ed. San Diego, CA: Plural Publishing; pp 3–32.

World Health Organziation. (2001) *International Classification of Functioning, Disability and Health (ICF).* Geneva: Author.

Yathiraj A, Maggu AR. (2013) Screening test for auditory processing – a preliminary report. *J Am Acad Audiol.* 24(9), 867–878.

CHAPTER 30

Central Auditory Processing Disorder: Therapy and Management

Jack Katz, Jeanane Ferre, William Keith, and Angela Loucks Alexander

INTRODUCTION

In recent years, interest and involvement with central auditory processing disorders (CAPDs) has increased among audiologists, speech-language pathologists, and others. The auditory training (AT) aspect of CAPD has perhaps grown even more rapidly, as those who are drawn to rehabilitation have begun to see the benefits of this rewarding work. Providing a re/habilitative component makes the CAPD evaluation itself much more important, especially because the issues that are uncovered can be addressed and improved.

AT is based on the concept that improved central nervous system (CNS) function can increase our ability to focus on, and decode, what we want to hear. It can also increase the likelihood that the auditory information that is received will be remembered and organized accurately, as well as improve our ability to combine auditory input with other information. Improved central auditory processing (CAP) skills ultimately support higher cognitive functions (see Chapter 28). The most important auditory input we hear is speech, but because of its rapid and complex structure many people find it a major challenge in effectively communicating and in achieving academic success. Those of us who provide therapeutic services generally involve speech in some way, because of its face validity and its potential for directly improving important processing functions.

Neural plasticity enables the AT to positively change one's auditory performance. This is especially effective with repetitive stimulation, permitting our brains to facilitate processing (Skoe and Kraus, 2010).

We are not always able to choose when we can provide AT and working with older, not necessarily elderly, individuals may have some benefits. Those who know that they need the help are likely to be more determined/highly motivated compared to young children. So, age alone should not disqualify a person from getting rehabilitative services.

One of the major confounding issues for AT for those with CAPD is when the person continues to have middle ear fluctuations, which are important contributors to the faulty or vague information that the brain has stored (Bennett et al., 2001). Ordinarily, AT has a long-term effect, but when a person continues to have middle ear issues we often see deterioration that tends to nullify the improvement.

An important aspect of working with those who have CAPD is to provide optimal hearing. This can be provided in a number of ways including hearing assistive technologies (HATs), speaking a little louder, and reducing the distance between speaker and listener.

The techniques discussed in this chapter have been used successfully for many years. They are rather simple and positively effect important communicative and/or academic functions. In addition to those improvements, we find that the individuals attend better and are much more confident when they understand what is going on around them. In a sample of 67 children who were seen (by the first author) for CAPD therapy 57% of the parents reported greatly improved self-confidence, 30% moderately improved, and just 1% with no improvement. Each of the authors has had great success and enormous satisfaction from doing this work and seeing the positive results.

BUFFALO MODEL THERAPIES

The Buffalo Model was formalized after many years of evaluating and providing therapy for those with CAPD (Katz and Smith, 1991). There are four major categories of CAPD (Katz, 1992), which are reviewed in Chapter 29. The two basic and most common categories are decoding, which is the ability to quickly and accurately analyze what is said, and tolerance-fading memory (TFM), which is primarily a difficulty in understanding speech-in-noise and with short-term auditory memory (STAM) tasks. In this section, two therapies will be described for each category.

Decoding is generally the most common CAPD category and so this therapy applies to almost all of the individuals we see. In addition, decoding is such a basic function that it is generally the first concern in therapy, along with TFM functions. Decoding is closely associated with the auditory cortex (Katz and Smith, 1991) which Luria (1970) refers to as the phonemic region of the brain. It is in the left upper,

mid-posterior region of the temporal lobe. We conceptualize these CAPD problems as vague or inaccurate encoding in the auditory cortex because of early otitis media or other etiologies. To improve on the inaccuracies and inefficiencies that are noted in understanding speech, learning to read fluently, and other functions, the therapy is directed to narrowing the phonemic boundaries (i.e., sharpening the perception) of the affected sounds.

We see that by improving the accuracy and speed of processing phonemes, it generalizes to understanding speech more broadly (Russo et al., 2005). This is likely because the therapy helps to replace the inaccuracies and inefficiencies in the way the brain perceives. Of course, what information the person has lost in the past or has misunderstood will not automatically improve and for the most part, the earlier the training, the better.

Phonemic Training Program

The Phonemic Training Program (PTP) is a basic program to improve auditory decoding. Observers are often surprised that such simple procedures can make such a significant difference in the ability, not only to process speech, but to result in improved reading word accuracy, auditory spelling, and even the clarity of the person's speech. Indeed PTP is so simple that the technique can be used from preschoolers to the elderly. Working with very young children or those with severe problems may require some modifications of the procedures. Further details of PTP and other Buffalo Model therapy procedures and forms are available elsewhere (Katz, 2009).

The purpose of PTP is to improve the speed and accuracy of processing speech. Although, the purpose is to improve speech understanding, in general, most of this work is carried out with individual speech sounds (phonemes). The procedure is given live voice at a fairly close distance. Figure 30.1 shows this close proximity to maximize the clarity of speech and the use of an embroidery hoop that is covered with acoustically transparent material (loudspeaker material) which prevents the listener from using visual cues. On the table you will see cards with letters signifying the speech sounds. Capital letters are used to simplify the visual association and diacritical marks identify long and short vowels. Some sounds that are easily confused with others have key words written at the bottom of the card.

The general plan for PTP is to start with an easy task and gradually increase the difficulty as the person improves in auditory processing of the sounds. We begin by introducing some of the most difficult sounds that have been observed for the individual. This may seem to contradict the first rule, but it does not. Initially, the difficult sounds are presented, but not contrasted with competing sounds or letters. For example, we often start with /d, ɛ, /, m / that are among the more difficult for those we see for CAPD evaluations. Although they may be difficult these four sounds are easily distinguishable from one another. We determine which of the sounds are most difficult by the use of a phonemic error analysis (PEA). PEA is based on the speech-sound errors on the three tests of the Buffalo Model which contains 926 phonemes.

FIGURE 30.1 A typical setting for PTP with the therapist obscuring much of the lower face by holding an acoustically transparent screen (*hoop*) and presenting specific sounds to which the listener responds by pointing to the appropriate letter. For most of the procedures there are four or fewer cards, but in review there are eight in the General review.

Principles

1. *Phonemes.* If phonemes are weak, vague, or inaccurately imprinted on the brain this forms a poor foundation for communication and academic success. When the foundation is improved the dependent functions also benefit. In PTP the emphasis is on improving phonemic perception.
2. *Repetition.* Starting in infancy the brain begins encoding phonemes, correctly or not, and after several years these models are strongly imprinted. Therefore, they cannot be improved simply with a cognitive approach. We cannot simply say, stop hearing the /ɛ/ as an /ɪ/. Rather it should be retrained the way physical therapists reteach a person to walk. They start slowly and simply and gradually work up from there. Gradual increases are built into PTP along with repetition, but it need not be boring.
3. *Over time.* As in the case of repetition, do not expect a great deal of improvement with just one or two presentations. Just like proficiency in playing golf or tennis, improved speech-sound perception requires practice over time. PTP is surprisingly quick but still requires sufficient time to master it.
4. *Easy.* Start at an easy level and work up gradually. It is generally important for those who have less confidence in what they hear or have had academic failures to start with success and then go forward.

5. *Four sounds.* Introduce four new sounds each visit. For most children and adults, this has been quite effective. Accommodations should be made for age and severity.
6. *Consider severity.* Based on the severity of the problem (e.g., on the PEA) begin with the more difficult items first. We begin with the difficult sounds while the brain is not crowded with other sounds and because we have more opportunities to repeat them in subsequent sessions.
7. *Visual confusions.* Be mindful of visual challenges as well as auditory (e.g., a long-A and a short-A differ only by a little marking above them).
8. *Auditory confusions.* When a person has confusion in distinguishing between two sounds (e.g., f/θ or ɪ/ɛ), branch from the program and use a "focus" or "itch" technique as described below.
9. *Teach accurate sounds.* This is to replace vague or poorly encoded sounds with as clear and accurate sounds as possible. Therefore, when the therapist says the consonants they should not have an added vowel (e.g., /b/ and not /bə/).

PTP Steps

With these principles in mind there are three major steps to PTP. The order of these steps differs for the first three visits.

1. *New.* For the first session there were no previous sounds so the first step is to introduce four sounds (e.g., /d, ɛ, /, m/). The person is told that they will hear a sound a few times and just to listen carefully to how it sounds but not to repeat it.
   a. *Introduction without bias.* With lower face concealed say the first sound clearly, usually three times, as this will increase the person's ability to identify it. Then without the hoop show the card with that symbol and say, "That was the /d/ or the D-sound as we hear in 'dog'."
   b. *Repetition with pointing response.* Then place the card in front of the person and indicate that each time you say /d/ you want them just to (quickly) touch the card. Behind the hoop say the sound once with a response and then again. There should be no error as there is only one card to point to. Then introduce a "foil." A foil is used occasionally to maintain attention. The therapist says, "That was good, but what if I said /s/? There is no card for /s/ so you point over here (off to one side) to let me know that I did not fool you." Then practice pointing to the /d/ again and next give an easily distinguished sound for a foil. Use foils sparingly from time to time especially when attention is lagging or the person is ready to point to a card before the sound is said.
   c. *Introduce a new sound and discriminate.* Remove the D-card from direct view and give the next sound in the same way without bias. But after doing the reinforcement with just the second sound, bring back the previous sound in front of the person. This discrimination task just slightly increases the challenge but is mainly to hear it again and associate the letter and point. After one presentation of the two sounds remove them and introduce the third sound. Then give the discrimination task with the three sounds, remove them and introduce the fourth sound, and finally discriminate all four sounds.
2. *Brief review.* On the next session give a brief review of the sounds from the previous session in the same way and in the same order but perhaps a little faster.
   a. This helps to remind or reinforce the sounds from the previous lesson before any New sounds are given. The procedure is not meant to be difficult, rather to gradually teach the person what the sounds sound like individually and to associate them with their letters.
   b. Then give the same procedures as in (a), (b), and (c) above with the New group of four sounds.
3. *General review.* A third procedure is added to the previous two on the third visit. After the Brief review of the sounds from the second lesson the General review increases the challenge and lets us know if there are sounds which confuse the person. The General review, unlike the two previous steps, presents the sounds in random order from all of the ones that have gone through the New and Brief review steps in the previous sessions. Over time most of the sounds will be contrasted with one another.
   a. *Open discrimination 1.* From the deck of randomly ordered cards for the General review sounds for that session take the first four cards from the deck for the person to discriminate. To include the person in the process hand all four to the person to place them face up to discriminate. Without visual cues the therapist says each sound and the person points to the card. Usually the prior training enables the person to give the proper responses, but note confusions for possible repair training. If there is an error repeat the sound, but if it is still difficult indicate the card and move on. We do not want to reinforce confusions.
   b. *Open discrimination 2.* Remove the first four cards and give the person the next four. But after this discrimination leave those cards and bring back the previous four (that were temporarily removed) and have these cards in the second row (N, AW, L, R as shown in Figure 30.1). The discrimination with eight cards is called *The Big Test.* This increases the variety and challenge of discriminations that must be made. If there are more cards go on to Open discrimination 3.
   c. *Open discrimination 3.* Retire the cards from Open discrimination 1 and temporarily remove the second group. Give the next four cards to the person and after discriminating them bring back the group 2 cards and give The Big Test.
   d. If there are more cards continue as before (i.e., remove Open discrimination 2 cards and temporarily remove group 3 and start with group 4).

e. *Reduce the cards.* When there are as many as 20 cards or so in the General review deck, it is well to maintain that number by eliminating the easier sounds.

Branching Strategies

When we hit a significant bump in the road or a brick wall it is best to provide some special help for about two to five visits.

Focus

If a person confuses /ɪ//ɛ/ we can increase the distinction in a rather simple way. We assume in this case that the /ɪ/ is the easier of the two. It usually is but if unsure ask the individual which is easier to hear. Generally, they are correct. When they have two sounds that are weak, there is no "anchor" so we first improve the easier one and then we can improve the weaker one.

a. *Build up the strong sound.* The two cards are placed in front of the person and they are told they will hear the /ɪ/ sound three times and then the /ɛ/. Knowing which sounds beforehand will help to ensure that they respond correctly. This is important because they are often confused so they get off to a good start.
b. Once the sounds have been introduced in this way the next step is to practice. Now, they are told the procedure will be the same. The /ɪ/ will always be first but the number of times it is said will vary and then the other sound will be said. This task is meant to be easy, especially if we start with the easier sound.
c. For this second step, say the /ɪ/ one to four times and then the /ɛ/. The purpose is not to trick them as they are already confused.
d. When the responses are accurate and quick, this step is completed and in the next session reverse the procedure.
e. Indicate that the /ɛ/ will be given three times and then the /ɪ/. Then, as before, explain that the /ɛ/ will be said three times and then the /ɪ/ once. After that introduction indicate that the /ɛ/ always will be given first but you will not tell the person how many times before the /ɪ/.
f. The procedure, starting with the more difficult sound, may well have to be repeated on the next visit and perhaps more.

Itch Cards

Itch cards are key-word cards. When a person begins therapy, there is not much to be done with only four new sounds. Because these are difficult sounds for the individual it is a good opportunity to reinforce the correct sounds.

a. After the PTP procedure, the same four sounds can be given. Behind the hoop the therapist indicates that some sounds will be said and then they will be told to touch/point to the card and say the "Itch Word." The name Itch Word comes from the first card that was developed for this procedure. Itch cards show a key word with the critical letter(s) underlined and/or in a different color.
b. After saying /d/, now without the hoop, say that, "the Itch Word for /d/ is Dessert" and show the card. When the person hears the sound they should point to the card and say the word "Dessert." Place the card in front of the person and practice once or twice.
c. Then remove the card and introduce the next one in the same way. After practicing one or two times bring back the first card to discriminate among the two sounds and the person pointing and saying the word for each (one time is enough).
d. Finish up with the last two sounds in the same way.
e. If there is a second group of cards to be reinforced do them separately in the same way.

The other decoding procedure in the Buffalo therapies is Phonemic Synthesis (PS). Generally, we do not do these tasks back to back. Rather one of the other procedures (e.g., words-in-noise training (WINT)) would be given to work on a different aspect.

Phonemic Synthesis

PS was the first of the therapy procedures in Buffalo Model. It is a sound-blending type task in which the listener hears individual speech sounds and must put them together to form the given word. This procedure both reinforces the PTP program and takes the training to a higher level. It requires not only accurate decoding of the sounds, but also memory for the sounds, maintenance of proper sequence, and relating the results to words.

Luria (1970) indicated that the auditory cortex is the center for phonemic processing. He found in his study of soldiers with gunshot wounds that the only place in the brain that was associated with phonemes was the auditory cortex (also see Chapter 28). The specific skills that he enumerated were phonemic discrimination, phonemic memory, and phonemic synthesis or analysis. Several highly respected professionals in various fields have noted the beneficial effects of sound blending–type training for reading and articulation problems. These include such luminaries as Van Riper, Orton, and others (see Katz and Harmon, 1981). Additional references for this chapter can be found on http://Katze7…

Benefits and Disadvantages of Recorded Phonemic Synthesis Program

In general, the recorded program has many more positive characteristics. It is recorded so it can be delivered in the exact same way and repeated without concern that there are variations in how the sounds were spoken. Speech sounds are produced correctly by an experienced speaker. The program was carefully designed to take a person with almost

no skill to a very high level of performance by gradually increasing the level of difficulty of sounds, as well as, combinations of sounds, and words. Difficult words are often given with clues or explanations and then repeated later in the program to be sure they are processed properly and reinforced. The program is sprinkled with humor to get an occasional chuckle. This recording has been in continuous use by many professionals since 1982.

There are two disadvantages to the recorded program. The first is that it cannot be varied for the individual and the second is that the therapist's speech might be richer than the recording and inflected emphasis cannot be given to call attention to some aspect. Fortunately, using the recording does not prevent the therapist from replaying an item, skipping items, or giving parts of the program live voice when needed as well as giving cautions or instructions to focus or alert the person. In some severe cases, the program can start live voice and then the recorded program can be administered when the person is better trained.

Description of Phonemic Synthesis Program and Basic Approach

The recorded PS program* has 15 lessons that start with two very easy picture choices (i.e., distinguishing the given word "she (/ʃ…i/)" from the word "pencil" which is the foil). It is given again and then four other items are presented in the same way. All five items are also repeated later on to be sure the person knows these easy sounds and words as this program builds on itself. The next lesson has the same five words with three picture choices. The third lesson begins with the same three picture choices and then the person says the words without picture support. However, after hearing the sounds of these words so many times it is easy for almost all children (or adults even with severe/profound challenges) to make the transition to generating their own answers.

For most people with CAPD, it is not necessary to begin with lesson one. We determine this based on their PS test performance. Most people start with lesson four or five. However, the program assumes that the listener knows the previous words. Those who skip earlier lessons are given some brief training on the words that were skipped before starting the recorded program.

The words are gradually expanded (e.g., "cow"' from the original five words to "couch") or changed and harder sounds (e.g., more short vowels and liquids /l, r, j, w/) are more likely to be used. Toward the end of the program, phonemic analysis is introduced. In this procedure the person is given a word and they are asked to break them up into their component sounds.

*Precision Acoustics, 13410 SE 26th Circle, Vancouver, WA 98683, (360) 447-8403; paaudproducts@gmail.com, www.paaudproducts.com

The score for each lesson is recorded on a summary sheet on which there are two sets of marks for each lesson. Toward the top of the chart are two heavy lines indicating the completion level. If a person reaches the completion level, that lesson can be considered finished. Further down the column there is a dashed line that shows the lower limit of the target zone. The person is expected to score above that level if they have been doing fairly well on the previous lessons. If the score is below the target zone there will only be frustration and lack of learning if they go on to the next lesson. So either further training is needed on that lesson (with special help, see later) or if very poor or frustrating the person needs to go back to an easier level and work their way back gradually.

On the answer sheet we currently mark PS items with an X-circled to show that there was a delay. As the individuals improve they reduce their delays and also get more items correct. Therefore, we consider both speed and accuracy when assessing improvement. This has been extremely helpful. If an item is in error, we do not show the delay.

Branching Strategies

General

It is permissible to repeat an item in this program, especially after the first administration of a lesson. The second time the task is generally easier. To improve the chances of getting the correct answer when an item is repeated the person may be alerted, "Listen carefully to the first sound/the vowel, etc." If it is less likely that the person will process the word correctly they could be told what the word is (e.g., jump) and say, "Listen carefully and see if you can hear 'jump, jump'." If the person hears the recorded version soon after the word was said live voice the chances for an accurate response are increased. On the score sheet it is helpful to show the initial error and a slash to show that it was given again and a dot if it was correct the second time (but count only the first response).

Word Chart

When a person makes errors on a lesson, it is extremely helpful to address this on the following visit when their brains are clear of this confusion. We fold a piece of paper in half top down twice and then left to right. This gives us eight boxes to show four pairs (the actual word and the error given). Figure 30.2 shows an example of a word chart in which the test and error words cannot be determined from the position. The paper is folded (with the aid of a paper clip) to show one pair at a time. To be sure that the person does not stop listening after pointing to the first word, that word can be repeated a second time for a large percentage of the presentations. Then we have the Big Test in which all of them (as many as four pairs) are give at once. The person is to point to the word that was presented randomly, one sound at a time behind the hoop.

lock	rock
end	and
ball	bowl
sand	stand

FIGURE 30.2 In the Phonemic Synthesis program, a word chart is used in the session following the errors on the PS lesson. The correct and error words are shown side by side in an order so the person does not know which one is the word on the program. The *dashed lines* show where the paper was folded so that each pair can be given one word at a time, sound by sound and then the Big Test with all eight words showing.

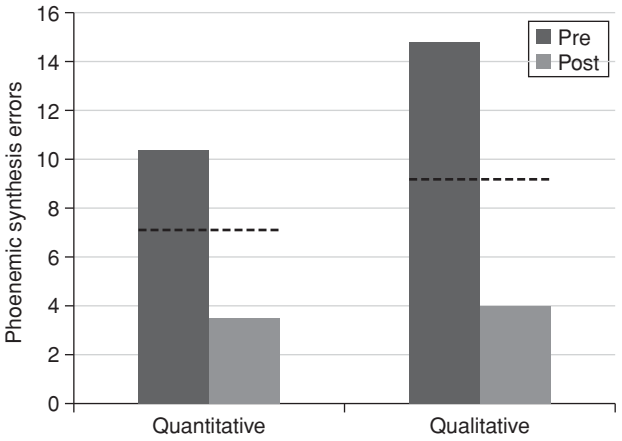


FIGURE 30.3 Phonemic Synthesis quantitative and qualitative error scores before and after the first round of therapy ($N = 95$). Normal limits for each measure are shown by *dashed lines* for the group's mean age (9 years).

Results of Decoding Therapy

In the Buffalo Model, we assess the results in three ways: How the person performed on the therapy materials themselves, how the person performed on the retest compared to the pretest, and how the person is performing on the initial concerns that brought the person for assessment originally.

Figure 30.3 shows the test–retest results for the PS test for 95 children 6 to 18 years of age who completed the first round of therapy. The average number of sessions was 13 and the total time spent on decoding skills per child was less than 6 hours. The good results that were obtained over this short period of time were supported by the assessment of parents and teachers following the first round of therapy. Figure 30.4 shows the parent–teacher assessments ($N = 88$) for that therapy period. The decoding therapies appear to have generalized by having a major effect on these skills that are associated with decoding.

Words-in-Noise Training

This is the first of the two TFM procedures that will be discussed here. A large percentage of those with CAPD have difficulty understanding speech in noise. Having better decoding skills makes the task in noise much easier and therefore both types of therapy are often given at the same time. However, WINT is designed to address the ability to focus in on speech and to pull out the speech from the background of noise. This particular skill seems rather fragile because it is susceptible to factors such as poor sleep the night before, anxiety from a bad day at school, and inattention. This therapy is also called "desensitization training." The anxiety and tension that are often seen in those with significant reactions to noise also appear to be addressed to some extent with this therapy.

Overview of WINT

This program begins with single syllable words that are presented, one at a time, at a comfortably loud presentation level with no noise and the listener is asked to repeat the words. Then the task is repeated with the next group of words along with a mild noise. Then the level of the noise is increased gradually until a signal-to-noise ratio (SNR) of zero is reached (i.e., equal to the speech level).

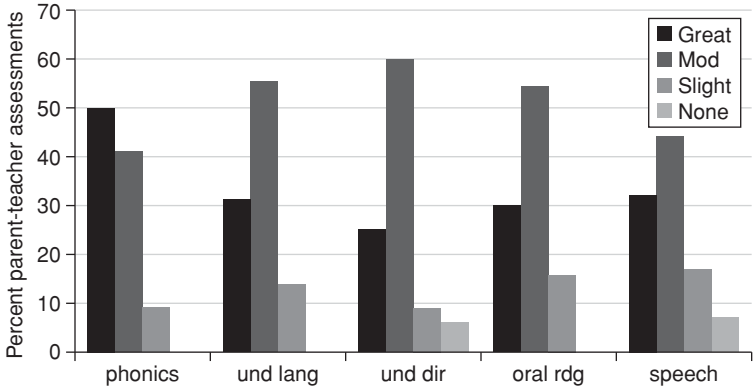


FIGURE 30.4 Parent–teacher assessments of improvement on decoding-associated skills after one round of therapy ($N = 88$). The highest percent of moderate or great improvement was noted for phonics (91%) and the fewest was speech (76%). The others were understanding language, understanding directions, and oral reading.

Description of WINT

There are two versions of WINT[†]. The WINT-3 recording is used with a CD player and a two-channel audiometer. WINT-1 is preprogrammed so that it can be administered without an audiometer. The basic procedure for both programs is essentially the same. We will discuss the WINT-3 first. Both programs can be presented through one or more loudspeakers or through earphones. It is best when both options are available.

One track of this program is made up of 600 primary level words that are divided into 60 subgroups of 10 words each. The other track is multitalker, eight-speaker babble. A series is approximately eight subgroups that are presented in one session. Each track of the CD has eight subgroups except the last track that has four. Figure 30.5 is the scoring form for track 1. As in the other Buffalo Model procedures, a dot represents a correct answer and an incorrect response is written out for most words. If a word might be ambiguous it is spelled phonetically or phonemically.

The WINT-3 Procedure

Most individuals who have CAPD have a positive score on the Speech-in-Noise test of the Central Test Battery-CD and speech-in-noise concerns on the Buffalo Model Questionnaire that the family filled out. One can choose to start therapy with any track and move along from one track to the next and after eight tracks back to the first track again as the dates show in Figure 30.5. The person is told that they will be hearing a man say some words and just to repeat them as clearly as possible. Some people will mumble and try to hide their errors. Have them repeat the word, spell it, or tell you what it means. Sometimes, the parents, if present, can clarify the response. Also, have the person face you and turn up the talkback microphone.

The first 10 items are given with no noise and speech presented at a comfortable level. The same speech level is used for subsequent visits, if possible. Enter both errors and delays on the score sheet so that the person's performance can be assessed, reviewed, and analyzed, if desired. Next the person is told that there will be some noise in the background and not to pay attention to it and just repeat the words. For the next subgroup we start with a SNR of about +12 dB. Then on each subsequent sublist, increase the noise by 2 dB until the SNR is zero. After the first few sessions the +12 noise level may be quite easy. This level can be omitted on future visits to save time, if desired.

Those who have the poorest scores initially tend to make the most gains in therapy. Generally, an average of four to six total errors for last one or two series suggests good performance. At a later time this can be rechecked to be sure that the performance remains good. This is especially important to check if the person has middle ear problems or persistent allergies.

Additional Procedures

a. Usually the first two or three series are given with little or no correction to establish baseline performance. After that corrective procedures can be used. Give preference to errors at low noise levels because they are usually the easiest to correct. Do not correct for too many errors as this can discourage the person, which will not help speech-in-noise training.
b. After an error, stop the CD and simply repeat the item. If a hint will make it more likely to get the repeated item correct, the person can be instructed to listen to the first/last sound or the vowel, and so on.
c. Instead of (b), if more assistance is likely to be needed, the person can be told the word and replay the item. It is good to say the word live voice once to three times just before the recorded word is heard. This will increase the person's chances of perceiving the word correctly.
d. When an error is persistent, or the individuals are sure that they are correct, turn off the noise channel so the word can be heard without interference.
e. Do not "beat a dead horse." If the person continues to hear the word incorrectly indicate that it will be worked on in the future and go on.

WINT Results

Figure 30.6 shows the average performance on the WINT-3 series in the first round of therapy. The average improvement is from about 18 errors initially to 6 at the end of the first round. On average, there is a rather steep decline in errors for the first five series after which the improvement is much more gradual. Most of the initial improvement is likely the "limbic effect," that is, the person getting used to the task and feeling more at ease with listening in noise. The gradual gains are likely because of the increasing ability to separate the words from the noise and to understand the words better.

Figure 30.7 shows the parent–teacher assessment of improvement. They indicated primarily great or moderate improvement for the 74 children for each of the three questions related to noise issues. For Understanding in Noise, 93% indicated great or moderate improvement. For Distracted by Noise and Hypersensitive to Noise, the ratings were 88% and 77%, respectively.

The WINT-1 Procedure

WINT-1 can be used without an audiometer as it is preprogrammed. The first seven series/tracks of WINT-1 have no noise to +12 dB SNR and then in 2-dB steps up to 0 dB SNR. The scoring is the same as for WINT-3 and all of the Additional Procedures above, except turning off the noise, can be used.

The eighth series/track of WINT-1 is available for those with severe problems or those who need to be introduced to the noise more gradually. After the no noise sublist, the noise

[†]Upstate Advanced Technologies, 12 Shadow Vale Drive, Penfield, NY 12526; gsbusat@frontiernet.net.

WORDS-IN-NOISE-TRAINING 3 (WINT-3)

Speech 62 dB HL

Track 1 date:	June 03, 2012		Oct 14, 2012	Track 1 date:	June 03, 2012		Oct 14, 2012
dBN/Transducer	NO / FF	/	NO / FF	dBN/Transducer	56 / FF	/	56 / FF
eight 0:05	•		•	sun 3:45	•		•
chin	•		•	gas	•		•
crawl	•		•	hide	ⓧ •		•
peach	•		•	made	•		•
cold	•		•	wheel	ⓧ •		ⓧ •
glass	•		•	mean	•		•
duck	•		•	crash	•		•
leg	•		•	new	•		•
bird	•		•	child	•		•
of	•		•	soeak	•		•
Σ errors (delays)	( )	( )	( )	Σ errors (delays)	( )	( )	( )
dBN/Transducer	50 / FF	/	50 / FF	dBN/Transducer	58 / FF	/	58 / FF
shoe 0:60	•		•	slap 4:40	slam		•
chase	•		•	bleed	leed		•
nut	knot		•	feet	heat		•
cute	•		•	hand	•		•
bones	•		•	move	•		•
house	•		•	fill	•		•
mud	•		•	smash	black		•
share	•		•	bench	•		•
men	nin		min	mouse	ⓧ •		•
stone	•		•	try	•		•
	( )	( )	( )		( )	( )	( )
dBN/Transducer	52 / FF	/	52 / FF	dBN/Transducer	60 / FF	/	60 / FF
belt 1:55	ⓧ •		•	rest 5:35	ⓧ •		•
was	•		•	train	•		•
ring	•		•	bell	•		•
no	•		•	none	•		•
can	•		•	how	ⓧ •		•
taste	•		•	said	•		•
earth	•		•	meet	•		ⓧ •
hose	•		•	high	•		•
my	•		•	couch	howtch		•
pain	en		•	gum	numb		dumb
	( )	( )	( )		( )	( )	( )
dBN/Transducer	54 / FF	/	54 / FF	dBN/Transducer	62 / FF	/	62 / FF
tank 2:50	•		•	mail 6:30	ⓧ •		•
great	•		•	lap	black		•
five	funny		•	tight	•		•
hit	•		•	fudge	•		•
paint	ain't		haint	red	bread		•
day	•		•	each	•		beach
street	•		•	road	rose		•
hold	ⓧ •		•	card	•		•
one	•		•	stick	•		•
broom	vroom		•	roof	root		•
	( )	( )	( )		( )	( )	( )
date:				date:			

©Jack Katz Ph.D., 2008

FIGURE 30.5 Score sheet for WINT-3 demonstrating the features and scoring. After the person goes through the 60 sublists they begin again where they started using a second column. If therapy continues in a second round then it might require the use of the third column. *Dots* represent correct responses and *words* show the errors. ⊗ indicates a delayed response on a word that was correct.

training starts with an SNR of +22 dB that should be suitable for most people. The sublists go up to +14 dB SNR. This may require a few series before the person is quite successful with the low levels of noise. When the person shows good performance for track 8, the program can continue with tracks 1 to 7.

Short-Term Auditory Memory Program

Short-Term Auditory Memory Program (STAMP) is the second TFM training program. STAM is a critical aspect of

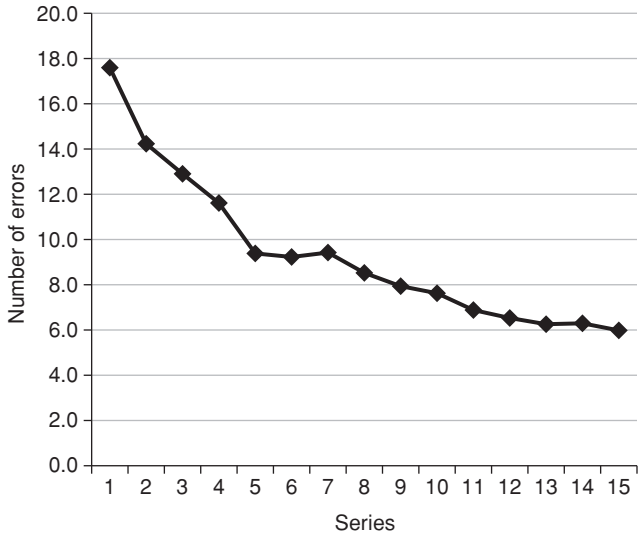


FIGURE 30.6 Mean number of errors across 15 WINT-3 series. The first five means show a sharply descending pattern. This is primarily associated with the "limbic effect," a reduction of anxiety and accommodating to background noise. The 10 more gradual reductions are primarily associated with increased success in understanding speech in noise.

CAPD. We often want or need to remember many things of value that we hear. A large percentage of those with CAPD have STAM issues and therefore are not able to remember nearly as much as most people. Importantly, STAM like the other aspects of CAPD responds well to AT.

Overview of STAMP

The purpose of STAMP is to increase short-term memory. In the Buffalo Model procedures we emphasize memory for digits, words, and working memory. Our purpose is to increase memory span by one unit and if that is successful we might try to stretch memory further. Generally, digits are the easiest and working memory the hardest. Working memory is remembering something and at the same time working with it in some way. Various tasks can be used for working memory training.

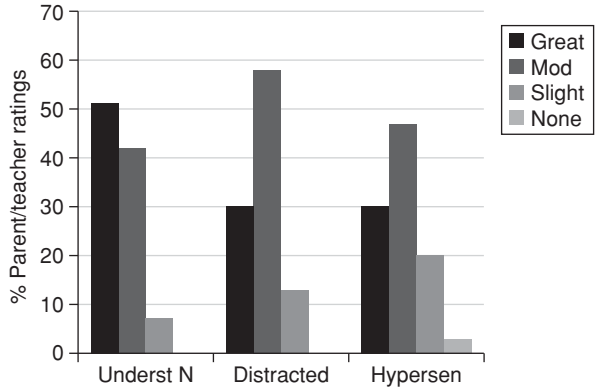


FIGURE 30.7 Ratings of parent–teachers regarding improvement in noise issues: Moderate or great improvement was noted in Understanding in noise (93%), Distracted by noise (88%), and Hypersensitive to noise (77%).

STAMP Procedures

Based on pretest scores we start with a level that the person can handle well (e.g., perhaps on a pretest 90% to 100% for three digits) and plan to expand their memory span by one unit (e.g., for four digits from 30% to perhaps 90% to 100%).

Figure 30.8 shows a sample of a memory response sheet for words. That plan is for working with someone who is quite good with remembering four digits, to both strengthen that level and to increase performance for the next level (in this case five digits). There are four increasing levels of difficulty from sections A to D, that is, the items tend to be more challenging. In addition, the number of easier level items is reduced from four to one so that there is more and more training at the higher level as the person gets stronger and stronger.

The following table shows the percent correct scores for "Sam" initially:

Percent Correct for Task and Units

Units	Digits	Words	Working Memory
3	100	90	80
4	90	80	50
5	10	0	0

We might start memory training working with Sam on digits because that is the easiest type, and work from four to five units. If this is successful, it will facilitate training for words from four to five units and this in turn can increase the likelihood of success for working memory but from three to four units. Figure 30.8 shows typical results for a person using the STAMP procedure.

Branching Strategies

a. Generally, it is a good idea to administer a sublist without correction the first time to establish a baseline and to see the issues.
b. When a person makes a small error it may be corrected by a simple repetition. To make the error more obvious one might emphasize the unit that was in error if this is thought to be necessary. Show the improvement on the response form but count only the first try.
c. For greater challenges it is well to tell the person the error and then repeat the item.
d. If this is still difficult or there are several errors at a particular level, one can use a "modified" procedure that will

A ↓	1. Date: Jan. 08, 2013 2.					X	R	X	R	Comments
1	**bird**	**mouse**	**cow**	**dog**		•	R		R	(1) 4W 4/4 = 100%
2	**red**	**brown**	**green**	**blue**		•	R		R	5W 5/6 = 83%
3	**table**	**floor**	**chair**	**wall**		•	R		R	
4	**mother**	**uncle**	**sister**	**cousin**		•	R		R	
5	**pen 1**	**pencil 4**	**paper 3**	**book 2**	**desk 5**	•	(R)		R	(2) 4W /4 =
6	**shoe**	**sock**	**coat**	**hat**	**shirt**	•	R		R	5W /6 =
7	**walk**	**run**	**jump**	**fall**	**climb**	•	R		R	
8	**leg**	**white**	**red**	~~**pink**~~	**silver**	—	R		R	
9	**house**	**school**	**car**	**bus**	**road**	•	R		R	
10	**ball**	**bike**	**swim**	**play**	**fun**	•	R		R	
B ↓	1. Date: Jan. 15, 2013 2. Date: Jan. 22, 2013					X	R	X	R	
1	**bread**	**butter**	**jame**	**milk**		•	R	•	R	(1) 4W 3/3 = 100%
2	**lettuce**	**tomato**	**salt**	**pear**		•	R	•	R	5W 4/7 = 57%
3	**rock**	**water**	**sky**	**star**		•	R	•	R	
4	**feet**	**ankle**	**knee**	**elbow**	**hand**	•	R	•	R	
5	**brother**	**father**	**cousin**	**uncle**	~~**aunt**~~	—	R	•	R	(2) 4W 3/3 = 100%
6	**blue**	**black**	**purple**	**pink**	**yellow**	•	R	•	R	5W 6/7 = 86%
7	**dog**	**cat**	**rat**	**sheep**	**cow**	•	R	•	R	
8	**spoon**	**knife**	**fork**	**plate**	**cup**	•	R	•	R	
9	**dress**	**pants**	**shirt**	~~**belt**~~	**shoes**	—	R	•	R	
10	**airplane**	**truck**	**bus**	~~**car**~~	**train**	—	R	—/•	R	
C ↓	1. Date: Feb. 2, 2013 2.					X	R	X	R	
1	**boat**	**fish**	**water**	**bird**		•	R	•	R	(1) 4W 2/2 = 100%

FIGURE 30.8 A section of the Word Memory Training form for four and five words show the scoring procedures. A *dot* [•] is correct, a *dash* [**–**] is an omission, a substitution is written in after the printed word, and the incorrect sequence is designated by *numbers next to words.* An *R that is circled* designates a reversal. When the person is given a second try at an item a slash [/] is shown and then the second score [if any] is shown. Only the first administration is considered in the scoring to the right. Because of the errors initially in sublist B it was administered again showing some improvement.

simplify the task to enable the person to achieve an accurate response. Then the regular items can be given again. Some considerations in modifying the items are given below.

e. For digits, numbers 1 to 5 are generally easier than 0 or 6 to 10; giving two in order (e.g., 4, 5) is generally easier than 3, 5 or 5, 4.
f. For words, shorter, more common nouns are easier than other words.
g. For working memory, consider (a) and (b) above. Also teach the tasks individually (e.g., putting digits in order from small to large and then add a word to the task)
h. For each of the procedures when a person performs well with perhaps three memory units, but four units are very hard, modify the four units by first giving the first three units and when successful then indicate that you will just add one unit at the end.

Results

STAMP is the most recent addition to the Buffalo Battery therapies. We have seen positive results in therapy with both children and adults. We have received very good feedback from others who are using these procedures which supports our findings (see Katz, 2009). For example, one adult with a degenerative neurologic disorder after working with digit memory began word memory going from three to four units. He quickly improved from 88% to 100% for three words and from 78% to 97% for four words. He is now at 100% for four words and at 62% for five words. Instead of degenerating, he continued to improve (Figure 30.9).

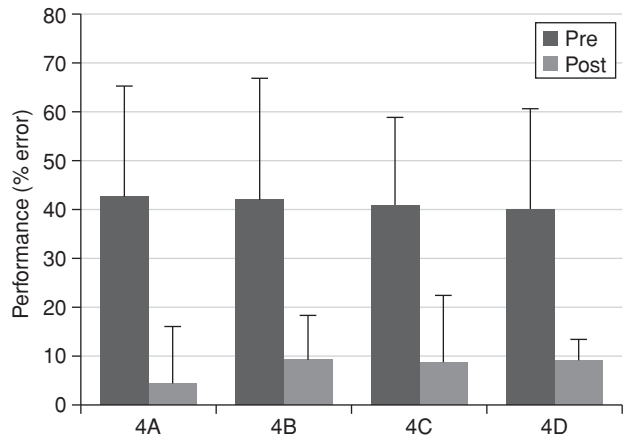


FIGURE 30.9 Memory training means and standard deviations are shown for four-digit items for 21 to 12 children for sublists 4A to 4D with a median of 17 children. Data for three digits are not shown as the target was four digits. If the therapy had not begun with 4A and ended with 4D we might have expected the initial scores to have been poorer initially for the later sublists.

SUMMARY OF BUFFALO MODEL THERAPY PROCEDURES

The Buffalo Model therapies have been highly successful when the specific categories have been identified. It is also a rather brief therapy averaging 13 (50-minute) sessions for the first round of therapy. Most children with mild and moderate CAPD complete the program in one round. Therapeutic benefits are determined by improvements on the therapy materials themselves, test–retest comparisons, and independent assessments by parents and or teachers regarding the observed changes.

M3 THERAPIES

The M3 model for remediation of auditory processing deficits is an integrated approach to treatment that uses a combination of bottom-up and top-down activities to improve specific auditory skills and to empower listeners to manage any residual adverse effects of the deficit on their lives (Ferre, 1997, 1998). The program conceptualizes communication as the interaction of three factors—the *message,* what we listen to, the *medium,* the environment in which we listen, and *me,* what the listener brings to the communication event. Negative changes in any of these three will adversely affect communication, for example, an acoustically or linguistically unclear signal, an excessively noisy or reverberant environment, or poor listening habits or impaired auditory processing skills of the listener. Conversely, positive changes will enhance communication. Applicable to all types of auditory processing deficits, the M3 model teaches the client (i.e., top-down activities) to effect positive change in themselves, the listening environment, and/or the message to maximize communication and *trains* the system (i.e., bottom-up activities) to work in a more efficient, age-appropriate manner.

MESSAGE refers to "what we hear," including speech sounds, words, phrases, sentences, patterns, and conversations. Therapy activities include minimal pairs auditory discrimination training, temporal pattern recognition, dichotic listening exercises, rhyming, sound-blending exercises, auditory closure exercises, and identification of key elements in targets. Intertwined with these activities is a discussion of familiarity, redundancy, and using contextual, syntactic, semantic, and nonauditory cues to assist recognition and listening comprehension.

MEDIUM refers to the environment in which we listen with sessions focusing on impact of noise, reverberation, distance, and lighting on speech recognition. Therapy activities include listening in noise and using visual cues.

ME refers to the unique combination of strengths and weaknesses that a listener brings to any communication event. Discussion focuses on using visual cues and active listening strategies, advocating for oneself, and training those auditory (and related) skills that may be deficient. Parents and caregivers are integral to the model, extending benefits of therapy beyond an individual session by regularly engaging the student at home in these same activities or analogous commercially available games.

For many students, computer-assisted auditory and/or multimodality training is included as an adjunct to the traditional therapeutic program. Detailed discussions of the specific activities noted above can be found elsewhere in this book and in other sources (Baran, 2013; McAleer-Hamaguchi, 2013). This section will describe the application of the model for specific auditory processing deficits, particularly as it relates to the student's need to meet the educational Common Core Standard for speaking and listening (CCSSI, 2012). Goals and representative examples of therapy benchmarks are provided for each type.

The student with auditory decoding deficit (Bellis and Ferre, 1999) exhibits poor discrimination of fine acoustic differences in speech with behavioral characteristics similar to those observed among children with peripheral hearing loss. The deficit can create secondary difficulties in communication (e.g., vocabulary, syntax, semantics, and/or second language acquisition) and/or academic (e.g., reading decoding, spelling, note-taking, and/or direction following) skills. The individualized education plan (IEP) for this student should include goals for improved auditory discrimination and closure, use of visual cues, noise tolerance, sound blending, auditory vigilance, and use of metalinguistic/metacognitive and active listening strategies. Metalinguistic strategies refer to the listener's ability to apply higher order linguistic rules when confronted with adverse listening situations. These include auditory closure (i.e., using context to fill-in missing pieces), schema induction (i.e., using expectations and experience to fill-in the message), use of discourse cohesion devices (e.g., learning to "key-in" to tag words and conjunctions), and prosody training (i.e., learning to use the rhythmic and melodic features of the signal to "get the message"). Metacognitive strategies refer to the listener's ability to think about and plan ways to enhance spoken language comprehension. These include attribution training (i.e., self-identification of the sources of listening difficulties), use of metamemory techniques (e.g., chunking, mnemonics), and self-advocacy (i.e., learning to modify one's own listening environment). Taken together, metalinguistic and metacognitive strategies enable the listener to be an active, rather than passive, participant in a communication event. The listener learns to use all available cues as well as their own knowledge and experience, altering behavior as needed, to enhance communication and improve processing. For a detailed discussion of metacognitive and metalinguistic therapies, the reader is referred to Chermak (1998) and/or Bellis (2003).

Auditory discrimination and closure. Student will recognize speech presented under a variety of listening conditions.

Benchmark examples. Student will discriminate minimally contrasted phoneme pairs presented auditorily only

(i.e., no lipreading/speechreading cues) in a background of multispeaker babble that is of equal loudness (i.e., at a 0 SNR) and emanates from the same location as the target signal with 90% accuracy. Activity examples: Student is given two choices, for example, *shuh – chuh* printed on individual cards. Therapist says one word at a time that either begins or ends with the phonemes and student determines which phoneme was spoken at the beginning of the word or at the end (e.g., *ditCH* – student points to *ch* card, *SHoe* – student point to *sh* card). Student will recognize words presented with visual cues in a background of multispeaker babble that is much louder than the target with 90% accuracy.

Use of visual cues. Student will use visual cues to improve speech recognition.

Benchmark examples. Student will discriminate same-difference for target presented visually only with 90% accuracy. For example, therapist "mouths" two words and student determines if the two words "look" the same or look different (e.g., *fail-rail—different, fail-fail—same).* Student will identify target compound word presented visually only (i.e., no auditory input) from among a closed set of no more than 30 printed words or picture choices with 90% accuracy. That is, therapist mouths a compound word and student identifies word from among a printed word list.

Noise tolerance. Student will recognize speech under adverse listening conditions.

Benchmark examples. Student will recognize everyday sentences presented *without* visual cues in a background of equal loudness noise with 85% accuracy. Student will recognize everyday sentences presented *with* visual cues in a background of noise that is much louder than the target with 75% accuracy.

Sound blending/synthesis. Student will recognize and manipulate multiple auditory targets.

Benchmark examples. Given a word and using a "phoneme list," student will create as many rhymes as possible within 2 minutes. Student will smoothly blend three nonsense syllables (e.g., *puh-tuh-kuh, spruh-struh-skruh*) using equal stress on each phoneme (or varying stress across phonemes, e.g., *SPRUH-struh-skruh*).

Auditory vigilance. Student will recognize change in a string of auditory targets.

Benchmark examples. Given a string of random words (or phonemes), student will indicate through hand signal each occurrence of predetermined "target" word (e.g., target word is *TREE*—string is *house, car, boat, TREE, dog, mom, TREE*). Student will indicate through hand signal "rare" or different target from within a string of common targets (e.g., *buh-buh-**dee**-buh-buh-buh-buh-**dee***). That is, student indicates when there is a change in stimulus.

Use of metalinguistic strategies (listening comprehension). Student will recognize and use key elements in spoken targets.

Benchmark examples. When given a sentence, student will state what information is conveyed by key (i.e., stressed) word with 90% accuracy (e.g., My sister baked two dozen chocolate chip cookies on TUESDAY. Key word: Tuesday. Information conveyed: When).

When given a sentence, student will state what information is conveyed by two (or more) key (i.e., stressed) words with 90% accuracy (e.g., MY sister baked TWO DOZEN chocolate chip cookies on Tuesday. Key words: My, two dozen. Information conveyed: Who, how many).

When given a short passage, student will answer fact-based questions posed by speaker with 80% accuracy (e.g., *who, what, amount, date, place names*).

Use of metacognitive strategies (active listening). Student will demonstrate understanding of active listening strategies.

Benchmark examples. Student will state two "self-help" strategies for use in highly noisy or reverberant environments. Student will state two "self-help" strategies for use when signal message is acoustically or linguistically unclear.

Therapy activities given above can be supplemented through the use of simple low-cost or no-cost games such as the "telephone game," *A Rhyme in Time,* and the "ending sound game," in which each player says a word beginning with the last sound of the previous word (e.g., caT-ToP-PiG-GuM-MuD-DoG). Regular use of these games extends the training beyond the individual session to maximize benefit.

The student with integration deficit, likely because of inefficient interhemispheric communication, struggles to perform tasks that require intersensory and/or multisensory communication. The child does not synthesize information well, may complain that there is "too much" information, and, without adult assistance, has difficulty determining task demands, starting complex tasks, transitioning from task to task, or completing tasks in a timely fashion. Impact on communication is variable with, typically, observation of academic effects in reading, spelling, writing, and other integrative tasks. In therapy, this student needs activities designed to improve interhemispheric integration, including dichotic listening, intersensory integration (e.g., use of visual cues), sound blending, listening comprehension and working memory/rapid recall, vigilance, and active listening (i.e., using metalingusitc and metacognitive strategies). Dichotic listening underlies our ability to listen actively in a classroom and is a necessary first step in a protocol to improve auditory processing and classroom listening abilities.

Dichotic listening. Student will recognize dichotically presented targets.

Benchmark examples. Student will repeat two digits presented simultaneously, one to each ear, with 90% accuracy for each ear. Example: Right ear—*6,* left ear—*8,* student repeats *6,8*). Student will repeat four words presented simultaneously, two to each ear, with 80% accuracy for each ear. Example: Right ear—*house, car;* left ear—*goat, dig* where *house* and *goat* overlap and *car* and *dig* overlap. Students repeat all four words.

When given two sentences, presented simultaneously, one to each ear, student will repeat sentence directed to right ear only (or to left ear only) with 90% accuracy (binaural separation).

Interhemispheric integration. Student will increase communication between the two hemispheres.

Benchmark examples. Given an array of common objects, student will name object without looking at it with 90% accuracy. Given an array of common objects, student will "find" a named object without looking at it with 90% accuracy.

Listening comprehension. Student will recognize and use key elements within a spoken target.

Benchmark examples. When given a sentence, student will identify stressed word in the sentence with 90% accuracy. When given a sentence, student will identify two stressed words with 90% accuracy. Student will follow two-part, three-element verbal directions (e.g., *point to the large white square and the small blue triangle*), presented without visual cues in a background of equal loudness multispeaker babble with 90% accuracy.

Working memory/recall. Student will synthesize and manipulate auditory and auditory–visual information.

Benchmark examples. Given a "deck" of 50 word cards, student will generate single rhyming word for printed target within 2 minutes with 90% accuracy. Given a list of 50 words, student will generate two rhymes for each word within 3 minutes with 90% accuracy.

Active listening. Student will demonstrate ability to use active listening strategies. Active listening requires taking responsibility for one's listening success or failure by understanding the impact of the auditory impairment in one's life, recognizing those aspects of the communication experience that are under the listener's control, displaying effortful listening behaviors, and taking overt steps to avoid or correct potential communication mishaps.

Benchmark examples. Student will state two difficult listening situations that he/she has encountered. For a self-reported difficult listening situation, student will state (and practice) one strategy to minimize the listening difficulty.

Outside of the "therapy room," integration and related functions can be enhanced through regular use of commercially available games/toys such as Twister, Bop-It, Simon, and interactive video games (e.g., Wii system games).

Prosodic deficit is characterized by deficiency in using prosodic features of a target, a predominantly right hemisphere function. This student exhibits difficulty in auditory pattern recognition, important for perceiving running speech and following directions. Student may have difficulty recognizing and using other sensory patterns (e.g., visual, tactile). Adverse effects are observed in pragmatic language (e.g., reading facial expressions, body language, and gestures or recognizing or using sarcasm or heteronyms), rhythm perception, music, and nonverbal learning. Therapy goals focus on improving right-hemisphere–based auditory pattern recognition, recognition and interpretation of rhythm and stress cues in words and sentences (e.g., using prosodic cues), use of visual cues to assist recognition, and application of metacognitive and metalinguistic strategies, and self-advocacy.

Temporal pattern discrimination and recognition. Student will discriminate and recognize auditory patterns presented in quiet or noise.

Benchmark examples: Discrimination. Student will determine same-difference for two-, three-, or four-tone sequences composed of soft and loud (e.g., loud-soft), high and low (e.g., high-low-high), short and long (e.g., short-short-long-short) tones with 90% accuracy.

Identification. Student will identify three-tone sequence presented in quiet in a four-alternative forced choice (4AFC) format using printed choices with 90% accuracy for loudness, pitch, or duration sequences.

Imitation/recognition of tonal patterns. Student will imitate two-, three-, or four-tone patterns, presented with equal stress in quiet with 95% accuracy.

Imitation/verbal labeling of tonal patterns. Student will attach verbal label to two-, three-, or four-tone sequences presented in quiet, varying in pitch, loudness, or duration with 90% accuracy.

Use of prosody. Student will discriminate, recognize, and interpret stress in speech.

Benchmark examples. (a) Same-different discrimination—student will determine same-difference for two- or three-phoneme combinations presented in quiet with 95% accuracy. Example: Therapist says *muh-muh-muh* and *muh-luh-muh,* student states whether targets are same or different. (b) 3AFC identification—student will identify three-phoneme sequence from among a closed set of three choices, when presented in quiet, with 90% accuracy. (c) Open set recognition of stress—student will imitate (exactly) three-phoneme sequences presented in quiet with 85% accuracy. (d) Student will judge intent of statements presented in quiet with 85% accuracy, including sincerity/insincerity and emotion conveyed (e.g., anger, happiness, fear, sadness).

Use of visual cues. Student will use visual cues to assist message comprehension.

Benchmark example. Given picture choices, student will match "emotion" word/phrase, for example, *They are frightened,* with corresponding picture with 90% accuracy. Given printed sentences, student will identify and imitate the "prosodic" marker in the sentence with 90% accuracy (e.g., identify the *?* in a sentence to denote questioning/rising intonation and imitate the same).

Metalinguistic/metacognitive goals for students with temporal patterning/prosodic deficit are similar to those of students with impaired integration or auditory–language association (see below). Self-advocacy goals are similar across all processing deficit types in that all students should be able to demonstrate an understanding of the nature of their deficit and describe the uses of self-help strategies. At

home, students can practice temporal processing/prosodic skills using musical instrument training and games such as *MadGab* and *Simon.*

The associative deficit profile is not true central auditory impairment but instead represents significant auditory-*language* processing difficulties. Children with this deficit do not infer and/or apply the rules of language as well as their peers. Although normal processors too often do not "think outside the box," these children rarely are "in the box"; the "box" being those rules of language, both explicit and implicit, that we use to "get the message" of an auditory–verbal target. They may exhibit specific language impairments in syntax, vocabulary, semantics, verbal and/or written expression, pragmatics, or social communication. More importantly, though, they exhibit functional communication deficits even in the absence of specific language impairment.

A key behavioral characteristic is a finding of adequate academic performance in early elementary grades with increasing difficulty as linguistic demands increase in upper grades. This child may present with subaverage to subnormal intellectual potential when assessed using standard (language-biased) intelligence tests. This student's overall rehabilitation program will include an array of goals and benchmarks (implemented by the speech-language pathologist) addressing specifically impaired language and language processing skills as well as functional communication. Applicable goals within the M3 model appropriate for this student include those that address listening comprehension, working memory and recall, use of visual cues, and self-advocacy as opposed to those that train auditory-specific skills.

Listening comprehension. Student will use stress cues to interpret auditory information.

Benchmark examples. Given a short passage, student will write three key or stressed words or phrases within the passage with 90% accuracy.

Working memory/recall. Student will use key linguistic elements in a target.

Benchmark examples. Given no more than three clues, student will recognize word (e.g., given *white, fluffy, falls* students would respond *snow*) with 90% accuracy. When given a sentence, student will state what information is conveyed by two (or more) key (i.e., stressed) words with 90% accuracy (e.g., *MY sister baked TWO DOZEN chocolate chip cookies on Tuesday.* Key words: *My, two dozen.* Information conveyed: Who, how many).

Use of visual cues. Student will use visual cues to comprehend message.

Benchmark examples. Given printed or picture clues, student will "guess" target word or phrase (e.g., *the$$bank = money in the bank, thecccccc = the seven seas*) with 90% accuracy.

To extend the benefit of these language usage goals and activities beyond the therapy session, teen listeners can practice recognition and use of visual cues to "get the message" using the road signs section of *Rules of the Road* books. Additionally, students with auditory-language association issues should be encouraged to play language-based board games, such as *Scattergories, Password, and Taboo,* and verbal problem-solving games such as *Clue* and rebus-type and crossword puzzles.

Another secondary type of processing deficit exhibiting a unique pattern on central auditory tests is the output-organization deficit. This deficit is characterized by difficulty on tasks requiring efficient motor path transmission/motor planning and is likely a behavioral manifestation of impaired efferent or executive function. Behaviorally, the child may be disorganized, impulsive, and a poor planner/problem solver. Difficulties in expressive language, articulation, and syntactic skills may be observed as well as educational problems in direction following, note-taking, and remembering assignments. Like their peers presenting with the associative profile, students with output-organization issues benefit from activities to enhance use of visual cues, working memory, rapid recall, listening comprehension, especially as it relates to note-taking, and self-advocacy (see previous examples). Additionally, this student needs therapy to improve sequencing and, often, noise tolerance.

Sequencing. Student will apply verbally mediated strategies to sequence and organize auditory information.

Benchmark examples. Given a single word target, student will create as many rhyming words as possible in 2 minutes and in alphabetical order with 90% accuracy. Student will execute three-step sequential directions, in which each direction has one or two critical elements, with 90% accuracy when presented in a quiet environment. For example, *First, draw a straight line, then draw a circle below the line, and then draw a red star above the line.*

Noise tolerance. Student will tolerate extraneous noise/reverberation of varying loudness levels.

Benchmark examples. Student will repeat monosyllables presented without visual cues in a background of multitalker babble that is louder than target with 90% accuracy. Student will execute multistep, single-element sequential directions with 90% accuracy when presented without lipreading cues in a background of equal loudness multitalker babble. Games and activities that can enhance organization and sequencing skills include *Bop-it, Twister,* Simon Says, and "treasure hunt" games.

Treatment Effectiveness

To document treatment effectiveness, there must be evidence that change has occurred because of the treatment and not maturation or some uncontrolled factor (Goldstein, 1990). A growing body of research supports the use of top-down and/or bottom-up treatment to reduce or resolve specific auditory processing impairments and to support development of compensatory strategies (Bellis, 2003; Bellon-Harn, 2011; Chermak, 1998). Ferre (2010) examined change in

performance on a degraded speech task (Low Pass Filtered Speech) and a dichotic listening task (dichotic digits) for two groups of children diagnosed with CAPD characterized by impaired auditory discrimination and/or impaired binaural integration/separation. Twenty students received either weekly individual hour-long aural rehabilitation sessions supplemented with 60 minutes per week of the same therapy exercises administered by parents (120 minutes per week therapy) or 120 minutes per week of computer-assisted AT. Test scores obtained at initial evaluation (pre) and again following 30 hours of treatment (post) indicated significant improvement following treatment for both groups on both tasks, with scores for most students at or very near normal limits for age at post-test. Improvement noted in dichotic listening, despite neither group engaging in specific dichotic listening training, was hypothesized to be related to impact of poor discrimination/closure on the ability to recognize dichotically presented words. That is, these students exhibited poor dichotic listening on initial CAP evaluation not because of (truly) impaired binaural integration but because of poor decoding/discrimination. As discrimination/closure abilities improved, ability to recognize dichotically presented targets also improved. The results support the notion that therapy also can improve other related skills that were not targeted specifically.

Also of interest was the finding that while all students exhibited significant improvement, individualized "live" treatment/training provided greater overall improvement than computer-based training alone for these auditory skills. It is likely that the best possible therapy outcomes will be realized through the combination of individual treatment supplemented by computer-assisted training.

Summary of M3

Having defined as clearly as possible the disorder's nature and impact through the assessment process, one can develop deficit-specific intervention strategies designed to minimize the adverse effects of the deficit on the listener's life and (re)habilitate the system. The intervention process must meet each child's unique functional needs, be provided in a timely manner, use resources effectively, and be extended beyond the therapeutic environment into all aspects of the listener's daily life. In *M3* therapy, "top-down" strategies designed to teach the listener how to maximize auditory skills and compensate for residual effects of the processing disorder are paired with "bottom-up" techniques designed to improve specific auditory (or related) skills. The breadth and depth of the activities and strategies chosen will be unique to each student depending on specific processing deficit type and daily listening needs. All students, regardless of deficit type, will benefit from exercises to improve self-advocacy, empowerment, and active listening. Supplementing individual therapy with auditory, language, or multisensory games as well as computer-assisted auditory and/or multimodality training appears to maximize treatment outcomes, allowing students to meet their "auditory" goals in relatively short periods of time.

OTHER TREATMENTS

Treatment for Amblyaudia

Amblyaudia, abnormal interaural asymmetry on dichotic tasks, affects half or more of children with CAPD. As amblyaudia may not be corrected by remote microphone hearing aid use or general AT, it often requires specific remediation. Methods include the Dichotic Interaural Intensity Difference (DIID) procedure (Musiek and Schochat, 1998) and Auditory Rehabilitation for Interaural Asymmetry (ARIA, Moncrieff and Wertz, 2008), among others. Treatment of amblyaudia is similar to the treatment of the visual analogue, amblyopia. In amblyopia the dominant eye is inhibited by application of drops or an eye patch to reduce sensory input. In amblyaudia, input to the dominant ear is reduced by decreasing the stimulation intensity on dichotic training tasks. A variety of dichotic training materials can be used and presentation can be by loudspeakers (ARIA) or either headphones or loudspeakers (DIID). With the intensity fixed at a comfortable level in the nondominant ear, the level of competition in the dominant ear is gradually increased from a low starting level over a number of training sessions until the nondominant ear can achieve normal performance in the presence of equal intensity stimulation in the dominant ear. Binaural separation and integration tasks are used. Early trials involved training a number of times per week over many weeks. However, the ARIA procedure has been refined to require only four 1-hour sessions over 4 weeks in the majority of cases. Amblyaudia treatment appears to work by releasing the nondominant pathway from suppression by the dominant side.

Software and Other Materials for Auditory Training and Language Therapy

CAPD therapy can be categorized as bottom-up or top down. Bottom-up treatments encompass strategies to enhance signal quality, such as amplification and discrimination training, and include training of psychoacoustic skills (Sharma et al., 2012). Treatments which use higher level processes such as cognition, language, and metacognitive functions to understand the auditory message are classified as top down. They include therapy to improve vocabulary, word understanding, prosody perception, inferencing, reasoning, working memory, verbal rehearsal, summarizing, language, reading, and other high-level skills. As members of the multidisciplinary team involved in treating CAPD, audiologists tend to concentrate on bottom-up treatments and speech-language pathologists on top-down approaches.

There are numerous training packages, workbooks, and software programs promoted for CAPD treatment. Many are in a game format. Some are advertised direct to consumers. Auditory training games are also increasingly becoming available as mobile applications. Not all software packages, and few if any mobile applications, are evidence based, and some programs that have been extensively investigated are the subject of both positive and negative reviews. No single program is likely to meet all of a child's training and therapy needs and a particular child is unlikely to need all of the subcomponents in a particular package or software program. Hence, they are best used as home training adjuncts to clinician-directed therapy with clinician guidance to ensure that a child works at an appropriate level and on appropriate subtasks within any given program. Some of the popularly recommended programs for CAPD, for example, Earobics (Houghton Mifflin Harcourt), are primarily reading programs with auditory processing and phonics subcomponents. Some are generic adult brain training programs which happen to have auditory processing subcomponents. CAPD textbooks list many of the programs available but clinicians are still advised to review available evidence. The What Works Clearinghouse (Institute of Education Sciences, US Department of Education) is a useful source of reviews.

LiSN & Learn auditory training software (National Acoustic Laboratories, Australia) is a game-format evidence-based software training program specifically designed to remediate a particular central auditory deficit, spatial processing disorder (hearing in noise). Like the LiSN-S test of spatial stream segregation developed by the same research group, LiSN & Learn produces a virtual three-dimensional environment under headphones. Through a variety of games children learn to attend to target stimuli and suppress background noise.

Sound Auditory Training (Plural Publishing) is a software tool to enable clinicians to customize web-based auditory skills training for individual clients. Tasks train intensity, frequency, and temporal detection, discrimination, and identification using a variety of nonverbal and minimally loaded verbal stimuli. Immediate feedback for error correction and reinforcement is provided through animations in a game format.

Listening and Communication Enhancement (LACE, Neurotone) is an adaptive AT program designed to improve listening and communication skills. It is oriented to adults with sensory hearing loss and is evidence based. It contains useful training materials for adults and older children with CAPD.

CLASSROOM ACCOMMODATIONS AND HEARING ASSISTANCE

The Classroom Environment

Classrooms are a critical auditory environment for children yet many do not provide favorable conditions for hearing. The classroom environment is one that must be controlled to provide favorable conditions for hearing. There are three important variables to be noted: Noise, reverberation, and distance from the teacher. Sources of classroom noise may include the children themselves, furniture noise, ventilation systems, and external ambient noise. Ambient noise levels often exceed an optimum 35 dBA (unoccupied), and hard surfaces can reduce hearing effectiveness by increasing reverberation time beyond an optimum maximum of 0.3 to 0.6 seconds (American Academy of Audiology; AAA, 2011a). Signal level and SNR decrease with distance from the signal source. The primary signal decreases by 6 dB with each doubling of distance. However, in a reverberant environment the overall signal level may decline less, because of reverberation enhancement. Early reflections enhance the signal. Conversely late reverberation, while increasing the amplitude, degrades intelligibility. For all these reasons audibility in classrooms is best at a close distance to the teacher.

Children require a greater SNR than adults for speech recognition. Young children require speech levels that are at least 20 dB above those of interfering noise and reverberation (AAA, 2011b). In practice, this is difficult to achieve without amplification. Consequently, even children with normal hearing may experience difficulty hearing in class. Many children with CAPD particularly have difficulty hearing in background noise. The noise level does not need to be loud to disrupt auditory input. Adults with CAPD describe how noise from a fan or refrigerator can interfere in properly decoding speech. Some children with CAPD are overwhelmed by all classroom noise levels, becoming distressed and unable to function. Such children are sometimes withdrawn from school.

Sometimes, minor modifications to a classroom, for example, sealing obvious entry points of external noise and introduction of absorbent materials, may improve the acoustic classroom environment, but are unlikely to sufficiently improve the audibility for a child with CAPD. This is because some children with CAPD may need amplification of the primary signal, not just an improved SNR, to hear well (see Section "Amplification"). Hearing assistive technologies (HATs) and in particular remote microphone systems can alleviate or overcome all three sources of signal degradation in the classroom: Noise, reverberation, and distance from the talker.

Other Environments

Children with CAPD have difficulty when speech is rapid or degraded by distance, acoustic conditions, or accent, when information streams are complex or lengthy, and when competing sounds are present. It follows that HAT can be helpful to them in many aspects of their lives besides the school environment. Moreover, given the positive neuroplastic changes that occur over time from wearing amplification (Friederichs and Friederichs, 2005; Hornickel et al.,

2012), children with CAPD should be encouraged to use their HAT as much as possible.

Amplification

Terminology

The majority of recent studies of amplification for children with CAPD have used remote microphone hearing aids, with body- or head-worn receivers, which receive a signal from a microphone worn by the speaker. The transmission medium has typically been frequency modulation (FM). Hearing systems of this type are usually referred to as "personal FM systems." This term is ambiguous, because it refers to accessory FM systems used by wearers of conventional hearing aids or cochlear implants. Furthermore, FM is increasingly being replaced by digital modulation (DM) technology. From the point of view of advocacy as well as accuracy, use of the term "remote microphone hearing aids" reinforces the point that children with "central deafness" require amplifying hearing aids in much the same way as do children with peripheral hearing loss.

Until remote microphone hearing aids become recognized as simply another type of hearing aid they remain classified as assistive listening devices (ALDs) or, in more current terminology, a type of hearing assistive technology (HAT).

Remote Microphone Hearing Aids

Low-powered remote microphone hearing aid systems designed specifically for children with normal or near-normal peripheral hearing include the Phonak iSense Micro and the Oticon Amigo Star. In each case there is a choice of transmitter microphones which transmit to behind-the-ear receivers (Figure 30.10). Body-worn systems are also available though less popular. Placement of the transmitter microphone at chest level or beside the mouth in the case of boom microphones provides speech input levels of 80 to 85 dB SPL and 90 to 95 dB SPL, respectively. The high-level input and additional amplification, if required, enable output levels in the ear to be maintained at levels of 70 to 90 dB SPL. SNRs of the order of 20 dB can be achieved. Adaptive technology in some systems actively maintains the signal-to-noise advantage by varying the signal level up or down according to the background noise level. The signal level is enhanced in noisier conditions to maintain an optimal SNR. The systems are output limited at approximately 100 dB SPL. Eiten (2010) recommends that peak real-ear saturation response should not exceed 105 dB SPL when fitting ears with normal puretone hearing thresholds.

Research

The discovery of a therapeutic benefit of amplification for children with CAPD is one of the most exciting research findings in the treatment of CAPD. The assistive benefits have been long known, but recent studies repeatedly show improved auditory skills after a period of use of amplification when tested without the hearing aids, reflecting a neuroplastic change as a consequence of amplification use.

Friederichs and Friederichs (2005) followed 20 children with CAPD and comorbid ADHD over 1 year and assessed them on behavioral and electrophysiological measures. The experimental group of 10 wore binaural EduLink remote microphone hearing aids for at least 5 hours per day at school throughout the year. The experimental group showed continuing improvement relative to the control group on a variety of measures as the year progressed. Significant improvements were seen on teacher and parent assessments of Understanding the Teacher, Focus, School Results, and Dictation. Social behavior and attentiveness improved, with significant improvements on two of five psychoacoustic measures, frequency discrimination, and binaural temporal resolution. On auditory-evoked response measures using tones and an oddball paradigm there was impressive maturation of the N1 P2 (see Chapter 17) complex over time in the experimental group, only with the morphology improving

FIGURE 30.10 Remote microphone hearing aid systems. Phonak iSense Micro [**left**] and Oticon Amigo Star FM receivers with optional transmitters [**right**].

and P2 amplitude increasing from test to test. All tests were carried out without the remote microphone hearing aids on. The results provided evidence of improved ability to hear, improved ability to access learning, and neuroplastic development, as a result of the hearing aid use.

Johnston et al. (2009) studied 10 children with CAPD compared to a normal control group on measures of speech perception, academic performance, and psychosocial status. The experimental group wore binaural EduLink remote microphone hearing aids at home and school for at least 5 months. All children in the experimental group improved on measures of speech perception both in quiet and in noise (from spatially separated locations) irrespective of whether they had a specific hearing in noise deficit at the outset. These results are indicative of improvement in hearing ability because of beneficial treatment–induced neuroplastic development. The experimental group also improved on measures of academic performance and psychosocial status.

Smart et al. (2010) studied 29 children with CAPD, some with comorbidities. The children wore EduLink remote microphone hearing aids at school for 5 months. They were tested on a CAPD test battery and the Lexical Neighborhood Test presented with background noise from spatially separated loudspeakers. Pre- and post-treatment teacher and parent report data were also collected. Teachers and parents reported positive benefits. Significant positive improvements in auditory skills were reported post-treatment on two tests: The Frequency Pattern Test and the Lexically Controlled Words presented in noise. The post-treatment improvements were observed without the hearing aids which indicated positive neuroplastic changes as a result of the amplification. Umat et al. (2011) showed improvement on auditory working memory and Yip and Rickard (2011) showed improvement on spatial stream segregation ability from remote microphone use in children with central auditory deficits.

Sharma et al. (2012) carried out a randomized controlled trial of bottom-up versus top-down therapy on children with CAPD and comorbidities on 55 participants of an initial cohort of 90 children. Two subgroups additionally used EduLink remote microphone hearing aids at school during the 6-week course of therapy. Amplification was not tested independently, but as a supplement to therapy. The results were limited by the small group sizes and short period of amplification. Nonetheless, therapy plus amplification was shown to significantly improve some measures of core language and phonologic awareness compared to therapy alone. The authors suggested that remote microphone hearing aid use provided additional benefit over therapy alone.

Hornickel et al. (2012) studied 38 children with dyslexia and normal peripheral hearing over an academic year. CAPD is believed to contribute to the impairments in phonologic awareness and phonologic memory seen in children with dyslexia. Nineteen children comprising the experimental group wore EduLink remote microphone hearing aids at school during the year. Nineteen matched controls also with dyslexia attended the same schools. The children were assessed on reading ability, phonologic awareness, and auditory brainstem function. The auditory brainstem response stimuli were synthesized ba, da, and ga syllables. The children in the experimental group improved on phonologic awareness and reading, and their auditory brainstem responses demonstrated significant improvements in neural consistency. Moreover, the children who demonstrated the greatest improvement in phonologic awareness also demonstrated the greatest improvement in auditory brainstem function. In addition, neural response consistency was predictive of improvement in phonologic awareness. All tests were carried out without the use of remote microphone hearing aids. The matched control group did not demonstrate similar effects on any of the measures. The results provide strong evidence of auditory neuroplastic improvement as a result of amplification.

In combination, the studies reported above show improved performance on the following measures (recorded without the use of remote microphone hearing aids) as a result of amplification treatment: Cortical auditory–evoked potential amplitudes to tone stimuli, auditory brainstem responses to speech stimuli, frequency discrimination, binaural temporal resolution, frequency pattern recognition, auditory working memory, core language, phonologic awareness, and speech perception in noise (spatial stream segregation). Amplification appears to treat a wide range of auditory skills simultaneously.

Clinically, children will often pass previously failed CAPD tests at a review 1 year following the fitting of amplification. Interestingly, they usually do not wish to relinquish their hearing aids at this point. If, as it seems, amplification facilitates positive neuroplastic change, children should be encouraged to wear amplification as much as possible and not just in school for the assistive benefits in the classroom. The use of amplification during AT and language therapy may also be beneficial, though this does not appear to have been investigated.

Most recent research on amplification for children with CAPD has used remote microphone hearing aids, in particular the Phonak EduLink, the predecessor to the iSense. The positive results are generally attributed to a belief that most children with CAPD have difficulty hearing in background noise and that this is ameliorated by the beneficial SNR generated by remote microphone hearing aids. However, classrooms are not always noisy when the teacher is speaking. In addition, clinical experience shows that more than half of children with CAPD can pass the LiSN-S test of spatial stream segregation, a test which simulates hearing a signal against competing speech from a different location. Most children with CAPD benefit from amplification (Johnston et al., 2009). A similar argument applies to speculation that the principal benefit of amplification is improved attention. Attention is certainly a precondition for successful learning, but not all children with CAPD have attention deficits. There may be an additional explanation.

SNR is not an independent parameter; it is a product of two other parameters, noise level and signal level. In theory the SNR can be improved by reducing the noise level (unmasking), by increasing the signal intensity, or by a combination of both. But with the open canal fittings necessary when peripheral hearing is normal, noise cannot be blocked from entering the ear canal. Thus, signal level is the only parameter to change significantly in remote microphone hearing aid fittings for children with CAPD. The neurophysiological response to an increase in signal intensity is not entirely the same as the response to unmasking. The effect of increasing signal intensity on evoked auditory responses is well known. As signal intensity increases more axons are recruited, more synapses connect, synchrony of firing improves, response amplitude increases, response latency decreases, and response morphology becomes more clearly defined and repeatable. The common factor in the research reported above is increased signal gain delivered (binaurally) to the ears of the experimental subjects. This significant parameter of stimulus amplitude may be an important contributing factor in the success of amplification in remediating CAPD and in neuroplastic change.

Clinical observations also suggest that SNR improvement cannot be the sole explanation for treatment success with amplification. Some children with CAPD immediately hear better in an audiology test room, a quiet environment, when speech is amplified through audiometer headphones or trial hearing aids. Some clinicians claim excellent results in treating CAPD in children with conventional hearing aids. Some children with CAPD who are home-schooled wear hearing aids. Adults with CAPD may wear conventional hearing aids as their primary form of assistance. Conventional hearing aids do not share the same degree of SNR advantage of remote microphone hearing aids, but they do share potentially similar gain levels. Although not discounting the importance of improved SNR, perhaps the almost universal positive effects of amplification for children with CAPD might be due, in large part, to increased synchrony of firing in the CANS as a result of increased signal intensity.

The benefits of amplification for children with CAPD are not confined to hearing, learning, and neuroplastic development. Children's self-confidence, self-esteem, and social behaviors improve and listening effort is reduced. Friederichs and Friederichs (2005) reported behavioral improvements and Johnston et al. (2009) reported multiple benefits across a broad range of psychosocial measures. When asked about benefits of amplification parents frequently report improved self-confidence and markedly reduced tiredness after school.

Although this chapter is focused on CAPD, there is a small but growing body of evidence suggesting remote microphone hearing aids may also be beneficial for children with dyslexia, autism spectrum, attentional, and language disorders.

Conventional Hearing Aids

Kuk et al. (2008) and Kuk (2011) reported on a 6-month trial of binaural mild-gain conventional hearing aids with open fittings on 14 children with CAPD and normal peripheral hearing. The aids were worn at home and school. Kuk used gain levels of approximately 10 dB for conversational speech. Anecdotally, some clinicians report positive results with conventional hearing aids, but with possibly higher gain levels. If conventional hearing aids are ultimately proven to be of benefit for CAPD, then they may be useful in cases where having to use a transmitter microphone is an impediment. If hearing aids without remote microphones are used, then the child should sit close to the teacher to optimize the input signal level.

Classroom Amplification Systems

Classroom amplification systems, also referred to as sound distribution or sound field systems, provide amplification of the teacher's voice through loudspeakers. Their efficacy is variable, depending in particular on the room acoustics. Classroom amplification systems typically improve SNR by 3 to 5 dB, but may worsen SNR in classrooms with very poor acoustics. Adaptive systems which increase the amplification as the noise level increases can achieve better than 5 dB. Portable desktop systems in which a small loudspeaker is placed on the desk of an individual child provide a slightly better SNR, perhaps 10 dB, for that child. Remote microphone hearing aids can provide at least 20 dB improvement in SNR. A meta-analysis by Schafer and Kleineck (2009) comparing speech discrimination in noise with various FM systems in trials involving cochlear implant users showed no significant improvement with sound field systems but 17% improvement with desktop systems and 38% improvement with personal direct auditory input FM systems.

Candidacy for Amplification

It is sometimes mistakenly assumed that only children with CAPD who complain of difficulty hearing in noise, or who score poorly on a speech-in-noise test, will benefit from remote microphone hearing aids. In fact, research results and clinical experience indicate that nearly all children with CAPD show classroom benefit from personal amplification as long as the classroom teacher is cooperative. Results range from children whose hearing ability in class is instantly transformed through to those in whom benefits are more subtle and slower to manifest. There is no known predictive test of degree of benefit to be derived from amplification (though the Hornickel et al. study reported above shows an interesting correlation between initial inconsistency of the brainstem response and subsequent benefit). However, recommendation of amplification only for children with abnormal scores on tests of hearing in noise undoubtedly denies potential benefit to many children.

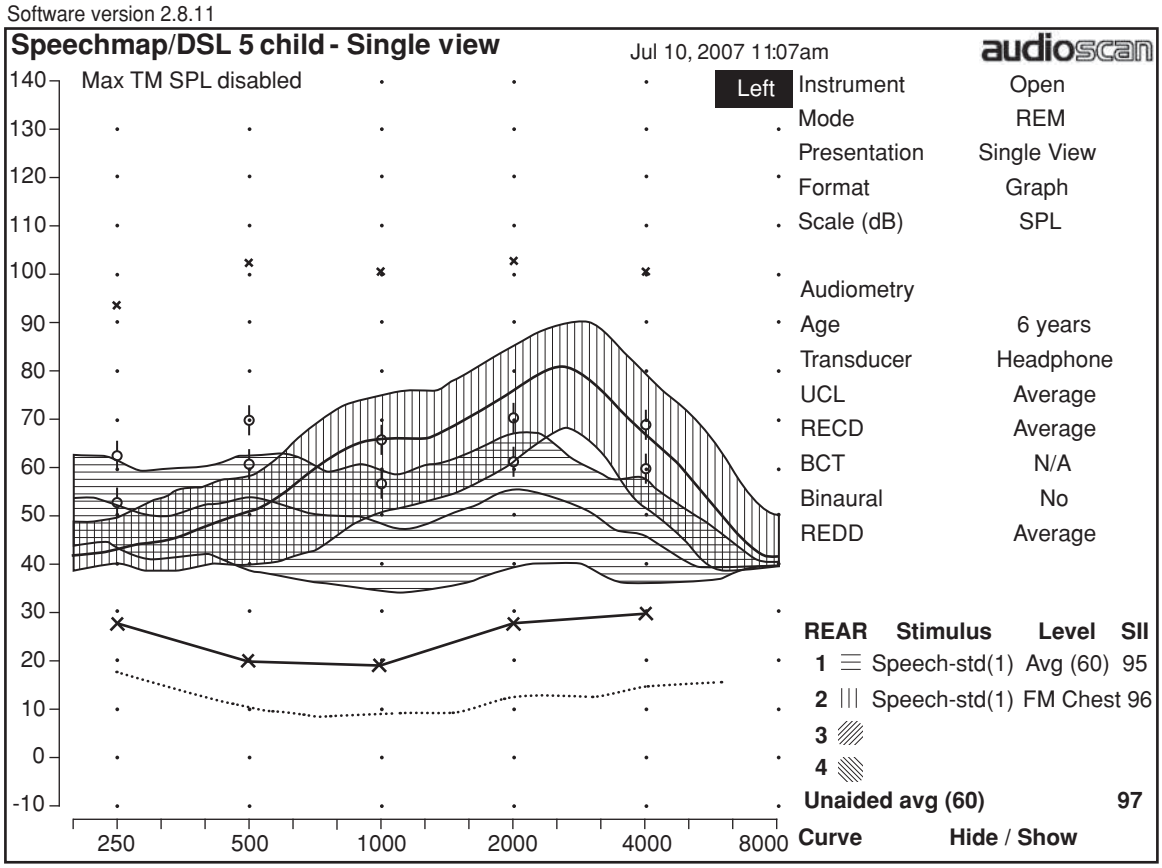


FIGURE 30.11 Example of electroacoustic verification of remote microphone hearing aid. *Upper curve* (*vertical hatches*) represents audibility of the amplified pathway. *Lower curve* (*horizontal hatches*) represents audibility of the unamplified pathway through the open ear canal. (From Eiten L. (2010) Assessing open-ear EduLink fittings. In: Achieving Clear Communication Employing Sound Solutions – 2008: Proceedings of the First International Virtual Conference on FM; pp 77–85.)

When to Fit Amplification

When a number of treatments may be beneficial it can be difficult to decide where to start. Amplification treats the underlying hearing disorder and it may be beneficial if amplification is worn during AT and subsequent top-down therapies. Amplification can address various auditory deficits simultaneously and it can, in many cases, provide immediate benefit in the learning environment.

Electroacoustic Verification of Amplification

Professionals prescribing amplification for CAPD should be familiar with the relevant section of the AAA (2011b) Guideline and see Eiten (2010). Two methods of electroacoustic verification are described. One utilizes targets based on audiometric thresholds, the other sets the system gain at unity for a 75-dB SPL speech-weighted input. One issue is the lack of research-based guidelines for target output levels for use with CAPD. However, it is recommended that the microphone is placed 1 to 6 inches from the speaker's mouth to enhance close speech input.

Figure 30.11 shows a typical real-ear output curve.

There is little if any research guidance on whether to fit amplification monaurally or binaurally in children with CAPD. Given all that is known about the effects of auditory deprivation and the benefits of binaural hearing, the safe course is to fit binaurally. Monaural amplification may actually cause deprivation effects in the neglected ear and initiate or increase interaural asymmetry.

The therapeutic benefit of amplification raises the question of how long hearing aids are needed for children with APDs and there is no research to provide us with an answer. Clinical experience suggests a wide range of individual differences, with significant numbers of children able to relinquish amplification after about 2 years, whereas a minority might require lifelong amplification. In the studies on treatment effects reported above, the greatest effects were seen in the longer studies of a 1-year period. Friederichs and Friederichs (2005) reported that they observed continuing improvement on various measures including cortical-evoked responses as the year progressed.

Behavioral Verification of Amplification

The AAA (2011c) Clinical Practice Guidelines (Supplement A) recommend behavioral (also referred to as functional) verification of HAT using speech-in-noise with speech at 50 dB HL (65 dB SPL) to represent the teacher's voice at a distance of 2 m, or speech at 40 dB HL to represent conversational level at 2 meters, with noise at 0 dB SNR. To simulate double the distance from the teacher (4 m) the signal level should be reduced by 6 dB. Materials and methods are outlined in the Guidelines and in Eiten (2010).

The Functional Listening Evaluation (FLE) (Johnson, 2004) provides a method for evaluating the effectiveness in the classroom. This is a procedure to test hearing with and without amplification at different distances in the classroom with and without background noise. Any speech material may be used and one of the test distances can be matched to the child's usual distance from the teacher. Consideration should be given to using materials that are more challenging than standard word lists for children with CAPD, for example, sentences or nonsense words.

Pre- and post-trial teacher, parent, and student observation questionnaires are also commonly used to evaluate amplification benefit in the classroom and home. Commonly used instruments are the Screening Instrument for Targeting Educational Risk (SIFTER), the Children's Auditory Performance Scale (CHAPS), and the Listening Inventories for Education (LIFE).

School and Teacher Guidance

One of the most critical factors affecting amplification success is the quality of the intervention with the school. Delegating school intervention to parents is also frequently unsuccessful. A very high acceptance rate can be obtained if a professional communicates with appropriate staff members including the teacher. Also advise and assist in the following areas: Explain the nature of CAPD and the child's difficulties, suggest management strategies, management of the amplification system, techniques to facilitate acceptance by the child and his/her peers, observe the child's auditory behavior and participation in class, manage an amplification trial, and where necessary assist in the preparation of individual education plans and applications for HAT funding. Educational audiologists or speech-language pathologists may perform this role. However, the more background and experience in special education, CAPD, and hearing aid management, the more likely the school will be to accept outside information and recommendations.

Teachers need advice on how to work with pupils with CAPD. First, their cooperation must be won by a collegial approach which acknowledges and emphasizes the importance of their role. Position in class is often emphasized as a first step in classroom management. Optimal audition is within about 2 m from the teacher but this is not an issue if the child is wearing remote microphone hearing aids. Clear speech, that is, speech at a slightly reduced rate and slightly raised intensity, is helpful for children (and adults) with CAPD. Instructions will be better understood if they are brief, clear, and simple. The teacher should verify that instructions have been understood. A hearing buddy beside the child with CAPD can assist. Complementary aids such as visual cues and written materials can support oral communication. Sometimes special accommodations for assessments and assignments will be necessary. More detailed advice on teacher guidance is available from CAPD texts.

SUMMARY

This chapter presented a number of effective approaches by experienced audiologists who have had wonderful results in helping those with CAPD. The consistent theme has been "if you do it, it will come." Taken together and with all of the research reported in Chapter 28, there should be no question, in the reader's mind, that CAPD is a treatable condition and that audiologists can contribute importantly to improving this prevalent disorder in a relatively short period of time in most cases.

FOOD FOR THOUGHT

You are an audiologist in a private practice. An important aspect of your work is evaluation of CAPD. Because of the great demand for services and the important contributions from therapy and management you would like to introduce these services. Based on what you have read and what you know about the topic, please answer the following questions:

1. Explain what services you would provide regarding individual and/classroom assistive devices. Please tell why and what procedures/devices you would employ and whether you would include orientation sessions and/or guidance for teachers, classmates, and/or families.
2. Do you think it would be advisable to employ bottom-up training/therapy approaches? Please explain why and, if so, what procedures would you include?
3. Do you think it would be advisable to employ top-down training/therapy approaches? Please explain why and if so what procedures would you include?

KEY REFERENCES

thePoint A full list of references for this chapter can be found at http://thePoint.lww.com/. Below are the key references for this chapter.

American Academy of Audiology. (2011a) Position Statement: Classroom Acoustics. Available online at: http://www.audiology.org/resources/documentlibrary/Documents/ClassroomAcousticsPosStatem.pdf.

American Academy of Audiology. (2011b) Clinical Practice Guidelines, Remote Microphone Hearing Assistance Technologies for Children and Youth from Birth to 21 Years.

American Academy of Audiology. (2011c) Clinical Practice Guidelines, Remote Microphone Hearing Assistance Technologies for Children and Youth from Birth to 21 Years (Supplement A).

Baran J. (2013) Metalinguistic skills, strategies, and approaches. In: Geffner D, Ross-Swain D, eds. *Auditory Processing Disorders: Assessment, Management, and Treatment.* 2nd ed. San Diego, CA: Plural Publishing; pp 469–494.

Bellis T. (2003) *Assessment and Management of Central Auditory Processing Disorders in the Educational Setting.* 2nd ed. San Diego, CA: Plural Publishing.

Bellis T, Ferre J. (1999) Multidimensional approach to differential diagnosis of central auditory processing disorders in children. *J Am Acad Audiol.* 10, 319–328.

Bellon-Harn M. (2011) Targeting prosody: a case study of an adolescent. *Commun Disord Q.* 32, 109–117.

Bennett K, Haggard MP, Silva P, Stewart I. (2001) Behaviour and developmental effects of otitis media with effusion into the teens. *Arch Dis Child.* 85, 91–95.

CCSSI. (2012) Common Core State Standards Initiative. Available online at: http://www.corestandards.org. Retrieved January, 2013.

Chermak GD. (1998) Metacognitive approaches to managing central auditory processing disorders. In: Masters MG, Stecker NA, Katz J, eds. *Central Auditory Processing Disorders: Mostly Management.* Boston, MA: Allyn & Bacon; pp 49–61.

Eiten L. (2010) Assessing open-ear EduLink fittings. In: Achieving Clear Communication Employing Sound Solutions – 2008: Proceedings of the First International Virtual Conference on FM; pp 77–85.

Ferre J. (1997) *Processing Power: A Guide to CAPD Assessment and Management.* San Antonio, TX: Psychological Corporation.

Ferre J. (2010) Aural rehabilitation & central auditory processing disorders (CAPD): outcome evidence among school-age children. *Educ Audiol Rev.* 27, 8–17.

Ferre JM. (1998) The M3 model for treating central auditory processing disorders. In: Masters MG, Stecker NA, Katz J, eds. *Central Auditory Processing: Mostly Management.* Boston, MA: Allyn & Bacon; pp 103–116.

Friederichs E, Friederichs P. (2005) Electrophysiologic and psychoacoustic findings following one-year application of a personal ear-level device in children with attention deficit and suspected central auditory processing disorder. *J Educ Audiol.* 12, 31–36.

Goldstein H. (1990) Assessing clinical significance. In: Olswang L, Thompson C, Warren S, Minghetti N, eds. *Treatment Efficacy Research in Communication Disorders.* Rockville, MD: ASHA; pp 91–98.

Hornickel J, Zecker S, Bradlow A, Kraus N. (2012) Assistive listening devices drive neuroplasticity in children with dyslexia. *Proc Natl Acad Sci.* 109 (41), 16731–16736.

Johnson C. (2004) The functional listening evaluation. Available online at: http://www.handsandvoices.org/pdf/func_eval.pdf. Retrieved May 15, 2013.

Johnston K, John A, Kreisman N, Hall J, Crandell C. (2009) Multiple benefits of personal FM system use by children with auditory processing disorder (CAPD). *Int J Audiol.* 48, 371–383.

Katz J. (1992) Classification of auditory processing disorders. In: Katz J, Stecker N, Henderson D, eds. *Central Auditory Processing: A Transdisciplinary View.* Chicago, IL: Mosby Yearbook; pp 81–92.

Katz J. (2009) *Therapy for Auditory Processing Disorders: Simple, Effective Procedures.* Denver, CO: Educational Audiology Association.

Katz J, Harmon C. (1981) Phonemic synthesis: Diagnostic and training program. In: Keith R, ed. *Central Auditory and Language Disorders in Children*. San Diego, CA: College Hill Park Press.

Katz J, Smith PS. (1991) A ten minute look at the CNS through the ears: using the SSW test. In: Zappulla R, LeFever FF, Jaeger J, Bildern R, eds. *Windows on the Brain: Neuropsychology's Technical Frontiers.* Vol 620. New York: Annals New York Academy of Sciences; pp 233–252.

Kuk F. (2011) Hearing aids for children with auditory processing disorders? *Semin Hear.* 32 (2), 189–195.

Kuk F, Jackson A, Keenan D, Lau C. (2008) Personal amplification for school-age children with auditory processing disorders. *J Am Acad Audiol.* 19, 465–480.

Luria A. (1970) *Traumatic Aphasia: Its Syndromes, Psychology and Treatment.* The Hague: Mouton; pp 18–19.

McAleer-Hamaguchi P. (2013) Metacognitive therapy approaches. In: Geffner D, Ross-Swain D, eds. *Auditory Processing Disorders: Assessment, Management, and Treatment.* 2nd ed. San Diego, CA: Plural Publishing; pp 431–446.

Moncrieff D, Wertz D. (2008) Auditory rehabilitation for interaural asymmetry: preliminary evidence of improved dichotic listening performance following intensive training. *Int J Audiol.* 47, 84–97.

Musiek F, Schochat E. (1998) Auditory training and central auditory processing disorders: a case study. *Semin Hear.* 19 (4), 357–366.

Russo N, Nicol T, Zecker S, Hayes E, Kraus N. (2005) Auditory training improves neural timing in the human brainstem. *Behav Brain Res.* 166, 95–103.

Schafer E, Kleineck M. (2009) Improvements in speech recognition using cochlear implants and three types of FM systems: a meta-analytic approach. *J Educ Audiol.* 15, 4–14.

Sharma M, Purdy S, Kelly A. (2012) A randomized control trial of interventions in school-aged children with auditory processing disorders. *Int J Audiol.* 51 (7), 506–518.

Skoe E, Kraus N. (2010) Hearing it again and again: on-line subcortical plasticity in humans. *PLoS One.* 5 (10), e13645.

Smart J, Purdy S, Kelly A. (2010) Personal FM systems for children with auditory processing disorder – successfully fitting this heterogeneous population. In: Achieving Clear Communication Employing Sound Solutions – 2008: Proceedings of the First International Virtual Conference on FM; pp 38–44.

Umat C, Mukari S, Ezan N, Din N. (2011) Changes in auditory memory performance following the use of frequency-modulated system in children with suspected auditory processing disorders. *Saudi Med J.* 32 (8), 818–824.

Yip F, Rickard N. (2011) Personal FM systems in children with a spatial stream segregation deficit. Poster (based on Master's thesis) New Zealand Audiological Society Conference 2011.

CHAPTER 31

Individuals with Multiple Disabilities

Anne Marie Tharpe and Samantha Gustafson

INTRODUCTION

Individuals with hearing loss and additional disabilities represent a widely diverse and complex group. They differ in the type and degree of their hearing loss, the type and degree of their accompanying disability, and their overall level of functioning. Approximately 25% to 50% of newborns who are deaf or hard of hearing have additional neurodevelopmental conditions, most often cognitive, behavioral-emotional, and motor problems (Chilosi et al., 2010; Fortnum et al., 2006). Similarly, the Gallaudet Research Institute (GRI, 2011) indicated that approximately 41% of deaf or hard-of-hearing school-age children have additional disabilities. As seen in Table 31.1, the most prevalent of these conditions were intellectual disabilities, followed by learning disabilities and vision deficits. It is also possible that some disabilities may not become apparent until well into childhood or adolescence, further increasing these numbers.

TABLE 31.1

Percentage of Disabilities that Occur in Children with Hearing Loss

Additional Disability	% Children with Hearing Loss
No additional disabilities	61.1
Vision impairment (including deaf-blindness)	5.5
Intellectual disability	8.3
Autism	1.7
Orthopedic disability (including cerebral palsy)	4.4
Specific learning disability	8
Attention-deficit disorder/ attention-deficit hyperactivity disorder	5.4
Emotional disability	1.8
Other	14.3

Note: Values were taken from Gallaudet Research Institute. (2011) *Regional and National Summary Report of Data from the 2009–2010 Annual Survey of Deaf and Hard of Hearing Children and Youth.* Washington, DC: Gallaudet Research Institute, Gallaudet University.

There is also some evidence to suggest that the number of people with hearing loss who have additional disabilities is on the rise (Synnes et al., 2012). Several reasons have been suggested to account for this increase including improved survival rates among very low (<1,500 g) and extremely low (<1,000 g) birth weight infants who have a high risk of disability (Cristobal and Oghalai, 2008). Once-extraordinary measures are now routinely used to save preterm infants who, even a decade ago, may not have survived. Most agree that those who do survive the traumas of birth are at higher risk of lifelong disorders than full-term infants (Robertson et al., 2007; Stoinska and Gadzinowski, 2011; Wilson-Costello et al., 2005). However, some studies suggest that the technology and intervention that have improved survival rates have also resulted in improved overall outcomes for premature babies (Jonsdottir et al., 2012; Washburn et al., 2007).

Genetic causes also contribute to the number of individuals with hearing loss and additional disabilities. Approximately one-third of those with multiple handicapping conditions have a syndromic cause of hereditary deafness (Picard, 2004). The most common of these include Down, Usher, Pierre Robin, Treacher Collins, and CHARGE syndromes. In underdeveloped countries where consanguinity is high and genetic forms of hearing loss are more prevalent than in the developed world, education and counseling about inherited forms of hearing loss might lead to a decrease in inheritable hearing loss (Smith et al., 2005). Maternal infection remains a contributing causative factor of hearing loss. Although the prevalence of maternal rubella infection is down worldwide, cases of cytomegalovirus (CMV) are on the rise. CMV is associated with hearing loss and motor and cognitive deficits. Additional risk factors for developmental delays include environmental teratogens (i.e., factors that have adverse effects on embryos or fetuses), maternal substance abuse, and environmental deprivation.

Clearly, the high prevalence of infants and children with hearing loss and additional disabilities serves to emphasize the need for audiologists to acquire knowledge and competence to meet the challenges posed by their complex needs into adulthood. This chapter reviews some of the general characteristics of children and adults with hearing loss and

additional disabilities. Basic principles for assessment and suggestions for management of these special populations are offered. In considering these suggestions, a few points should be kept in mind. First, it is likely that young patients with hearing loss and other disabilities will have some conditions that have not been identified at the time of the audiologic assessment. Therefore, audiologists should be mindful of the possibility that unknown conditions might influence the testing and management of some patients. This is especially true of more subtle conditions such as attention deficits and emotional problems. Second, the combined effects of some conditions may confuse or delay a diagnosis of hearing loss. For example, a child with autism and hearing loss might be nonresponsive to sound, in part, because of "tuning out" behavior and, in part, because he or she truly cannot hear some sounds. Third, a lack of training or experience might lead audiologists to think that some individuals with multiple disabilities are untestable by behavioral measures, which could result in a reliance on physiological measures alone. Certainly, physiological measures contribute valuable information about the integrity of the auditory system. However, we should keep in mind that behavioral tests provide an indication of how an individual *uses* his or her hearing, a very important factor when considering management needs. Collectively, age-appropriate behavioral and physiological test methods can result in an accurate assessment of hearing in most individuals with multiple disabilities and will result in an improved ability for audiologists to develop management strategies.

CUSTOMIZING THE HEARING ASSESSMENT

When evaluating individuals who have multiple disabilities, consideration must be given to any physical or cognitive limitations that could affect the assessment procedures. A thorough case history, review of prior evaluations, and keen observation can often identify the potential obstacles to assessment and may highlight individual strengths or interests that can be used to enhance the evaluation process. Obtaining as much information about the patient before the evaluation can help an audiologist prepare appropriately for the test session. For example, prior developmental testing or the use of developmental checklists will help audiologists determine an individual's ability to participate in behavioral tasks. Checklists are widely available and can be completed by parents or caregivers prior to their arrival at the clinic or while seated in the waiting room prior to the appointment. Likewise, when physical limitations exist (e.g., cerebral palsy (CP) or other gross motor deficits), modifications to any behavioral task requiring a motor response must be considered.

The widespread implementation of electronic medical records affords timely access to current medical histories and test results, thus avoiding repetitive tests and saving audiologists time in formalizing a profile of their patients. This is especially important when working with those who have multiple disabilities as they are likely to be receiving services from a number of professionals, thus providing a source of multidisciplinary information. More health systems today are moving toward an interdisciplinary model of care whereby several disciplines work together during a single consultation, assessment, or management session to provide an integrated plan of care. Interdisciplinary approaches to care can have an advantage over multidisciplinary care in that a patient's time is streamlined and communication among professionals should be enhanced. Another model of care is a transdisciplinary approach whereby representatives of several disciplines work together during the assessment and development of a care plan, but only a few members of the team provide the services. Regardless of the approach taken, communication among providers is of utmost importance when working with those who have multiple disabilities.

An initial observation without the patient's awareness can be helpful in determining typical behavior of the individual. Discretely observing the interactions between the patient and the caregiver in a waiting area can provide insight into the type, amount, and quality of communication or accommodation that may be effective (Dean and Harris, 2003). These initial observations aid in predicting how much cooperation can be expected and thus determining how to proceed with the assessment. For example, pretest observations of physical and cognitive engagement might reveal that an individual will not be able to participate in behavioral testing, and therefore, reliance on physiological measures will be necessary. Whether testing adults or children, individuals with multiple disabilities are more likely than typically developing individuals to require a heavy reliance on physiological measures over behavioral procedures. Observing the patient's behavior when his or her name is called in the waiting room can also provide some useful insight into the individual's level of functioning. Importance of the pretest interview cannot be overemphasized. Parents, care providers, therapists, and anyone who spends significant periods of time with the patient can provide valuable input about home and other environments, cognitive or physical limitations that might affect assessment or management, and potential compliance concerns.

Based on the review of case history information, previous evaluations, and observations of the patient, audiologists can prioritize the tests in the battery so that those likely to yield the most useful information and that are most easily obtained for the patient are conducted first. The order of the tests in the protocol might be quite different than that used with typically developing individuals. Audiologists should be mindful of the distinction between hearing sensitivity and responses of young children or those with developmental disabilities when interpreting the results of a behavioral test. Matkin (1977) coined the term *minimal response level* to describe the level at which a behavioral response to sound occurs, but also while recognizing that it might be elevated

as a result of nonsensory factors such as attention, motivation, or behavior.

CUSTOMIZING TECHNOLOGY MANAGEMENT

There is ample evidence to suggest that children with hearing loss and additional disabilities are likely to be fit with hearing technology (e.g., hearing aids, cochlear implants) later than otherwise typically developing children (Kaga et al., 2007; Oghalai et al., 2012). It is also reasonable to assume that adults with multiple disabilities receive hearing technology at a lower rate than adults with hearing loss who have no additional disabilities. There can be several explanations for this delay or lack of intervention including delayed confirmation of precise hearing levels, family/caretaker priorities on other health concerns, or concerns regarding one's ability to secure, care for, and safely wear technology. One way to assist in individualizing the hearing technology candidacy and selection process is the use of functional auditory assessments.

It is not uncommon when assessing the hearing of some individuals with multiple disabilities to obtain little in the way of formal behavioral test results during an initial visit because of difficulty gaining a necessary level of cooperation. However, even without the patient's cooperation, useful information can be acquired through the use of functional auditory assessment tools. These assessments evaluate listening behaviors in real-world settings—outside the confines of sound-treated booths where most formal audiologic testing takes place. The goal of functional assessments is to tell us not only *what* an individual hears, but more importantly, how the individual *uses* what is heard in everyday situations. In addition, information can be obtained about how listening behavior might change in different settings, under different conditions, or with different speakers. This information can then be used to guide more formal evaluation and management plans for these patients. Typically, this information can be obtained from self-assessment, parent, teacher, or caregiver questionnaires. Although these tools have primarily been designed for use with children, it is reasonable to adapt such questionnaires for information gathering purposes when assessing the needs of individuals of any age who have cognitive or behavioral disorders.

The following sections provide some limited guidance when considering hearing technology options for those with a variety of disabilities. Although expectations for benefit will naturally need to be adjusted relative to expectations of typically developing individuals, there is reason to believe that these patients can obtain significant benefit from various forms of hearing technology for daily living activities and in educational settings (Kaga et al., 2007; Oghalai et al., 2012). Counseling families regarding appropriate expectations for their child or family member receiving hearing technology, especially if receiving a cochlear implant, requires relaying a clear message that improvements in hearing might have little if any impact on nonhearing-related developmental concerns.

AUTISM SPECTRUM DISORDER

Autism spectrum disorder (ASD) is a developmental disorder characterized by symptoms appearing in early childhood and impairing day-to-day life function. These symptoms include qualitative impairments in social/communication interaction and repetitive and restricted behaviors, according to the *Diagnostic and Statistics Manual of Mental Disorders* (5th ed.) (DSM-5) (American Psychiatric Association, 2013). Under the umbrella of ASD, a patient's symptoms will fall on a continuum, with some showing mild symptoms and others, more severe. A diagnosis under the general diagnostic category of ASD is relatively new. Prior to the publication of DSM-5, there were five ASDs, each of which had a unique diagnosis: classic autism, pervasive developmental disorder (PDD), Asperger's disorder, Rett's syndrome, and childhood disintegrative disorder. With the exception of Rett's syndrome, these disorders are now subsumed into the diagnosis of ASD. Rett's syndrome is now its own entity and is no longer a part of the autism spectrum.

ASD is thought to have an early onset, with symptoms appearing before 24 months of age in most cases (Baghdadli et al., 2003; Ozonoff et al., 2010). Although a definitive diagnosis of autism is not generally made until the age of 3 years or later (Mandell et al., 2005), there are a growing number of reports of stable diagnoses following identification as young as 2 years (Chawarska et al., 2009). Prevalence estimates of ASD have increased steadily over time from reports of 1 to 5 children per 10,000 in the 1970s (Brask, 1972) to reports of 5 to 60 per 10,000 in the 1990s and early 2000s (Bertrand et al., 2001; Yeargin-Allsopp et al., 2003). Current numbers from the Centers for Disease Control and Prevention suggest a prevalence of 114 per 10,000 children (Baio, 2012; Rice, 2009). It remains to be seen whether there has been a true increase in prevalence of ASD over time or the reported changes in prevalence can be explained by changes in diagnostic criteria and increased awareness of the disorder by parents and professionals (Fombonne, 2003; Rutter, 2005). Boys are more likely to be affected with autism than girls, at a ratio of more than 3:1 (Van Bourgondien et al., 1987). About 50% to 70% of children with ASD also have an intellectual disability (LaMalfa et al., 2004).

There is no strong evidence to suggest that individuals with ASD have a greater risk of hearing loss than the general population. However, the presence of unusual sensory responses, including abnormal responses to sound, is considered an associated feature of ASD. For example, individuals with ASD might completely ignore sounds that would result in a reaction from typically developing individuals. Other times, they often appear to be overly sensitive to sound by covering their ears with their hands when loud or unexpected

sounds occur. In addition to these abnormal responses to sound, young children with ASD are known to lag behind on language milestones. Therefore, those with ASD will likely be referred to audiologists for hearing assessments as part of the developmental evaluation to rule out hearing loss as the cause of language delay. On average, behavioral responses to sound of children with ASD who have normal hearing are elevated and less reliable relative to those of typically developing children (Tharpe et al., 2006). Relatively little is known about higher order auditory abilities of individuals with ASD. However, altered temporal processing has been recorded in both adults (Samson et al., 2011) and children with ASD (Groen et al., 2009; Kwakye et al., 2011).

Special Testing Considerations

Children with ASD who have hearing loss are diagnosed, on average, almost 1 year later than those without hearing loss (Mandell et al., 2005). Therefore, it is reasonable for audiologists to be alert to the general behavioral characteristics of childhood ASD to facilitate referral for evaluation when indicated. Several screening tools are available that can be used by audiologists. These include, among others, the Modified Checklist for Autism in Toddlers (M-CHAT) (Robins et al., 2001) and the Pervasive Developmental Disorder Screening Test II (PDDST-II) (Siegel, 1996).

Understanding the general behavioral characteristics of those with ASD can also be helpful to audiologists as they consider modifications to the traditional test battery. Because the majority of those with ASD exhibit cognitive deficits, behavioral abnormalities, and hypersensitivity to sensory stimulation, audiologists should be prepared to address those issues during the test session. For instance, transitions are often difficult for individuals with ASD. When possible, audiologists should avoid travel from room to room with the patient, taking care to escort the patient to the testing area immediately rather than keeping him or her in the waiting area. Audiologists will want to minimize physical contact with those who have tactile sensitivities. This may require initial testing in sound field, because of the possibility of aversion to the tactile stimulation created by earphone placement. A quick inquiry with the parent or caregiver might alert audiologists to any sensitivity that could affect testing.

Regardless of the chronologic age of the individuals, audiologists will need to use behavioral test procedures that are appropriate for their patient's cognitive level. This may mean that procedures typically used with infants and young children (described in Chapter 24) such as visual reinforcement audiometry (VRA) or play audiometric techniques will be used with older children or even adults. If VRA is used, one should consider minimizing the impact of the reinforcement by turning off the animation (if a lighted, animated toy is used) or using a video reinforcement. Other testing options for patients functioning at a developmental level of 2.5 years or greater are conditioned play audiometry (CPA) and tangible-reinforcement operant conditioning audiometry (TROCA) (Lloyd et al., 1968). Although not commonly seen in audiology clinics, TROCA is often used in pediatric practices that specialize in serving those with multiple disabilities. TROCA requires the patient to press a bar or a button whenever a sound is heard, which is paired with the dispensing of a tangible reinforcement (e.g., small piece of food). TROCA is noted to be particularly effective with children having cognitive or behavioral (e.g., ASD) disorders. A significant number of children with ASD receive other clinical services (e.g., speech therapy). A thorough review of reports from other providers as well as a brief discussion with a caregiver can alert audiologists to reward techniques that work with an individual patient.

Patients with ASD are often resistant to earphones or probes used for individual ear testing. Audiologists can ask the parent or caregiver to practice listening activities with headphones with the patient prior to the appointment. If a patient with ASD will not allow the placement of earphones or probes, audiologists might have to resort to sedated procedures. This is certainly true if one plans to fit hearing aids. Individuals with ASD are known to be difficult to sedate with currently available pediatric sedating agents and are at risk for seizures while under sedation (Mehta et al., 2004). Therefore, consultation with the physician in charge of administering and monitoring the sedation process will need to include notification of the patient's diagnosis of ASD.

Special Management Considerations

For individuals with ASD, tactile sensitivities, and hearing loss, one can expect some resistance to wearing hearing technology. Therefore, maintaining consistent hearing aid or cochlear implant use might take longer to achieve with these individuals than with typically developing individuals. One technique for introducing amplification is to start by having the parent or caregiver gently massage the patient's ears several times a day until little or no resistance is offered. This may take anywhere from a few days to weeks. From there, one can introduce, to one ear only, a soft earmold without the device connected and build up wear time starting with a few minutes until the patient is willing to wear it for longer periods of time. Once the earmold is tolerated with little resistance, the device can be coupled to the earmold, and eventually, both devices can be introduced. Of course, this process will be slower or faster depending on the degree of tactile sensitivity and resistance offered. Hearing technology will need to be secured to the patient's clothing by use of retention devices designed specifically for that purpose. Such devices will leave the technology secured to the patient's clothing even if they are pulled from the ears. Once the individual becomes accustomed to wearing hearing technology, he or she may no longer need to use retention devices.

Loudness discomfort or hypersensitivity to sound has frequently been documented in children with ASD

(Tharpe et al., 2006). As such, it is essential that audiologists carefully adhere to prescriptive formulae for the selection and verification of hearing aid gain and output characteristics. Because it may be difficult or impossible to measure the patient's comfortable loudness levels, audiologists will often need to use age-appropriate normative targets provided by the prescriptive formulae. It is reasonable for audiologists to consider initially lowering the gain and output levels below those prescribed and gradually raising them as the patient becomes accustomed to the amplified sound. However, gain levels should always make speech audible for the patient.

PHYSICAL DISABILITIES

Persons who are deaf or hard of hearing should have similar motor development and skills as those with normal hearing unless vestibular function is affected. That is, deafness alone does not affect motor abilities or balance function. In fact, 93% of children with deafness have average to above average motor skills (Lieberman et al., 2004). Environmental factors such as emphasis on physical skills in the school curriculum, opportunities for practice and play, and parenting styles are believed to influence physical development of children with hearing loss. Audiologists should be aware of expected gross motor milestones in typically developing children. If a child with hearing loss is not walking by 15 months of age, a referral for further evaluation by a developmental psychologist or pediatrician is warranted.

Vestibular abnormalities that can result in gross motor problems include cochlear malformations such as Mondini's deformity and cochlear hypoplasia. Other congenital causes of gross motor deficits in children with hearing loss include syndromes such as CHARGE syndrome and Usher syndrome type I (described in a later section) and CP. CP is a disorder of neuromotor function. Approximately 3% of children with hearing loss also have been diagnosed with CP, which is characterized by an inability to control motor function as a result of damage to or an anomaly of the developing brain (GRI, 2011; Roush et al., 2004). This damage interferes with messages from the brain to the body and from the body to the brain. The effects of CP vary widely from individual to individual. There are three primary types of CP:

- Spastic—characterized by high muscle tone (hypertonia) producing stiff and difficult movement
- Athetoid—producing involuntary and uncontrolled movement
- Ataxic—characterized by low muscle tone (hypotonia) producing a disturbed sense of balance, disturbed position in space, and general uncoordinated movement

These three types of CP can coexist in the same individual. CP can also be characterized by the number of limbs affected:

- Quadriplegia—all four limbs are involved
- Diplegia—all four limbs are involved and both legs are more severely affected than the arms
- Hemiplegia—one side of the body is affected and the arm is usually more involved than the leg
- Triplegia—three limbs are involved, usually both arms and a leg
- Monoplegia—only one limb is affected, usually an arm

CP is not a progressive condition. The damage to the brain is a one-time event. However, the effects may change over time. For example, with physical therapy a child's gross and fine motor skills may improve with time. However, the aging process can be harder on bodies with abnormal posture or that have had little exercise, so the effects may result in a gradual decline in motoric ability. It is important to remember that the degree of physical disability experienced by a person with CP is not an indication of his or her level of intelligence.

The brain damage that caused CP may also lead to other conditions such as learning disabilities or developmental delays. Approximately 20% of children with CP will also experience hearing or language problems (Robinson, 1973). The hearing loss is typically sensory/neural in nature. In addition, between 40% and 75% of individuals with CP will also have some degree of vision deficit.

Special Testing Considerations

Individuals with motor delays may not respond behaviorally to auditory stimuli because their physical disabilities limit their ability to orient to sound (Moore, 1995). However, when testing children, VRA can still provide reliable information even for those with poor head and neck control. Modifications that might need to be made in the test arrangements for VRA include the use of an infant seat to provide additional head support. However, audiologists should ensure that head supports do not block the ears and impede sound field stimuli. If children with motor difficulties cannot make a head-turn response to sound, response modifications can be made. Modifications include alternative responses such as localizing to the sound stimuli with their eyes as opposed to head turns. CPA (see Chapter 24) might also require modifications. Response modifications might need to include options that do not require the use of fine motor skills. Examples of such modifications could include asking a child to drop a ball into a large bucket rather than having the child insert a peg in a pegboard, partial hand raising, or even just a head nod. Additionally, a variety of gross motor responses (e.g., hand motion) can be used to trigger an electronic switch that will, in turn, activate a computer screen programmed for appropriate visual reinforcement.

If the physical disability has a neuromotor component, such as with CP, physiological measures might be affected (Yilmaz et al., 2001). That is, abnormality in measures such as the auditory brainstem response (ABR) may be misinterpreted as indicative of hearing loss when, in fact, the

abnormality is in neurotransmission. Therefore, interpretation of the ABR must be made cautiously and in concert with the entire battery of auditory tests, behavioral and physiological. Sedation may be required when conducting ABR with individuals who have CP in an attempt to relax their head and neck or to reduce extraneous muscle movements, thus reducing myogenic artifact.

Special Management Considerations

When selecting and fitting hearing technology on someone with physical impairments, there are a number of factors that must be considered, including the types of activities in which the individual participates (e.g., physical therapy) and his or her fine and gross motor ability (e.g., use of a wheelchair with head supports). When fitting children, it is important that the audiologist consider input from the parents and other professionals working with the child when determining amplification options. Children who require amplification for their hearing loss are typically fit with behind-the-ear (BTE) hearing aids. However, use of this type of aid may be inappropriate for children or adults with physical handicaps if they have poor head control (Tharpe et al., 2001). The close proximity of head supports or the person's own shoulders, if the head is leaning to one side, may result in excessive feedback or discomfort from BTEs. Problems with feedback might be reduced by selecting a hearing aid with a feedback cancellation feature, although care must be taken to ensure audibility across the speech spectrum is maintained. Another feature that might be beneficial for those with poor head control is a remote control. This can provide easier manipulation of the controls (e.g., volume control) of the hearing aid by caretakers (Roush et al., 2004).

Body-worn hearing aids and cochlear implant speech processors, although rarely used today, provide another option and would eliminate many of the problems that BTEs pose for patients with poor head control. However, body-worn hearing aids also require special consideration when being used with patients who have physical disabilities. For example, for very young children and for those of any age with oral-motor difficulties, the microphone of the aid may be vulnerable to food and drink. Moreover, clothes may rub on the microphone port, resulting in extraneous noise, and wheelchair harnesses can rub or press against the aid, resulting in discomfort or damage. Although children are not typically fit with in-the-ear (ITE) hearing aids, they may be an appropriate solution for adults or children who spend part of their day in atypical positions or who use a wheelchair with headrests.

INTELLECTUAL DISABILITY

The term intellectual disability includes impairments of general mental abilities that impact adaptive functioning. Symptoms of intellectual disability first appear during the developmental period and diagnosis requires a comprehensive assessment of intelligence across conceptual, social, and practical domains (American Psychiatric Association, 2013). Adaptive skill areas include:

- Conceptual
  - Language
  - Reading
  - Writing
  - Math
  - Reasoning
  - Knowledge
  - Memory
- Social
  - Empathy
  - Social judgment
  - Interpersonal communication skills
  - Ability to make and retain friendships
- Practical/self-management
  - Personal care
  - Job responsibilities
  - Money management
  - Recreation
  - Organizing school and work tasks

As seen in Table 31.1, almost 10% of children with hearing loss also have intellectual disabilities (GRI, 2011). Those with an intellectual disability are at an increased risk for visual or hearing impairment or both (MacFarland, 2003). Detection and treatment of hearing loss in adults and children with intellectual disabilities is of utmost importance because hearing loss can exaggerate intellectual deficits by impeding the learning process (Roush et al., 2004).

Down syndrome, also referred to as trisomy 21, is the leading cause of hearing loss and intellectual disabilities and occurs in approximately 1 in 700 births in the United States (Parker et al., 2010). Audiologists are very likely to see a large number of children and adults with Down syndrome, a genetic disorder always associated with some degree of cognitive impairment. As individuals with Down syndrome age, there is a decline in intellectual ability. In fact, almost 100% of individuals with Down syndrome over 40 years of age demonstrate degenerative neuropathologic changes consistent with Alzheimer-type dementia (Zigman et al., 1995). Furthermore, some have speculated that the precocious aging of individuals with Down syndrome results in early presbycusis in this population (Dille, 2003). Hearing loss progresses more rapidly in adults with Down syndrome than those with other forms of intellectual disability or adults in the general population. Down syndrome is also frequently associated with conductive hearing loss and, less often, sensory/neural hearing loss. Although the majority of the conductive hearing losses in those with Down syndrome are secondary to middle ear effusion, some are the result of middle ear anomalies, such as ossicular malformations and damage to middle ear structures as a result of chronic

infection. In contrast to the typically developing population, the prevalence of middle ear effusion tends to remain high in individuals with Down syndrome regardless of age. Marcell and Cohen (1992) found that adolescents with Down syndrome have poorer hearing and greater incidence of conductive hearing loss than their peers with intellectual disability, but without Down syndrome. For a comprehensive review of hearing loss associated with Down syndrome, see Porter and Tharpe (2010).

Special Testing Considerations

Little has been published on hearing assessment of adults with intellectual disability. However, it is well documented that audiologists must use test techniques that will bridge the difference between the chronologic and developmental age of individuals with cognitive disabilities to obtain valid test results (Diefendorf, 2003; Roush et al., 2004). The patient's mental or developmental age, not their chronologic age, should be considered when selecting appropriate test procedures and materials. Several investigators have evaluated the effectiveness of VRA with children having intellectual disabilities, including those with Down syndrome (Greenberg et al., 1978; Thompson et al., 1979). With typically developing children and those with intellectual disabilities, VRA is effective with infants as young as 6 months cognitive developmental age. However, children with Down syndrome require a cognitive developmental age of 10 to 12 months to successfully participate in a VRA procedure. Furthermore, behavioral thresholds of infants with Down syndrome have been found to be 10 to 25 dB poorer than those of typically developing infants when all had normal hearing verified via ABR (Werner et al., 1996). This elevation of behavioral thresholds is presumed to be the result of more inattentive behavior on the part of the children with Down syndrome relative to their typically developing peers. Moreover, this inattentive behavior provides additional reason to utilize a test battery that includes physiological measures when testing children with Down syndrome.

Although it is recommended that audiologists attempt to elicit a spontaneous head-turn response during the VRA conditioning process (Tharpe and Ashmead, 1993), some children with intellectual disability may not have developed auditory localization ability. Recall that auditory localization is a higher order skill than detection, the required skill for VRA. In such cases, several administrations of paired conditioning trials (pairing the stimulus and the reinforcer) may be required. If the patient does not respond to the auditory stimuli, the audiologist may be left with the question, "Does the patient not hear the stimuli, or can she or he not perform the task?" One method that can answer this question is for the audiologist to place the bone vibrator either in the patient's hand or on the head and, using a low-frequency stimulus at approximately 50 to 60 dB hearing level (HL), determine if the patient can perform the task using this vibrotactile cue. In this way, the patient is able to feel the stimulus and, thus, is not required to hear to participate. If the patient is able to cooperate for the task under these vibrotactile conditions, then the audiologist should return to the auditory stimuli and continue testing with the knowledge that the patient understands the task.

If using a play audiometric technique, it is often appropriate for the audiologist to demonstrate the play task to the patient with intellectual disability rather than attempting to explain the instructions verbally. Because learning the desired response behaviors may take longer for children and adults with intellectual disability, it may be useful to have them practice the listening task at home before coming to the clinic. It is important to keep the task as similar as possible to what actually will be expected in the clinical setting. Another approach is for the audiologist to demonstrate the task engaging the patient's parent or caregiver as the one being tested. The patient can then observe the procedure being conducted and see what is required. If the patient has use of some language, the audiologist should keep verbal instructions short, simple, and accompanied by gestures. Nonverbal expressions of reinforcement can be used generously (e.g., smiles, clapping, thumbs up) to indicate to the patient that he or she is complying with the task. Audiologists should keep in mind that the reinforcement is provided to support the response behavior of the patient, not to indicate if the patient is correct or incorrect (i.e., can hear or not hear the stimulus). Additional time will likely be needed to complete the play task, and the audiologist should expect response delays as a result of additional time needed for the patient to process the instructions and formulate a response. It is not unusual for patients with intellectual disability to have to return for more than one visit to complete testing. However, the visits should not be so far apart in time as to result in a significant delay in diagnosis. It is important in these cases to keep the examiner and the test procedures the same so that a routine can be established with the patient. This differs from testing with typically developing children where the examiner often has to change the task to keep the child's attention.

Whether using VRA, CPA, or conventional test procedures, it is recommended that control trials (no sound trials) be included throughout the testing session. This is especially true if working with individuals who have Down syndrome, because they typically are eager to please others and this often results in a high number of false-positive responses. Control trials are inserted randomly into the testing procedure at times when the audiologist would otherwise present the auditory signal. If a response is noted during a control trial, it is evidence of a false-positive result and should not be reinforced. This lack of a reward for false responses should reduce their frequency.

Although important for complete evaluation of all patients, it is particularly important to monitor the middle ear status of those with intellectual disabilities, because they

are known to have a higher degree of abnormal tympanometry and conductive hearing loss than the general population (May and Kennedy, 2010). Those with Down syndrome have an even higher incidence of otitis media than others with intellectual disability, because of the anatomic anomalies of the head and neck including the cochlea, ossicles, Eustachian tube, and nasopharynx. Chronic ear infections afflict approximately 70% of children with Down syndrome (Mitchell et al., 2003). In addition, those with Down syndrome are highly susceptible to impacted cerumen, because of narrow or stenotic external ear canals. Therefore, all hearing test procedures (e.g., ABR, VRA, play or conventional audiometry) should include the use of bone-conduction testing when possible. A conductive component can mask the presence of sensory hearing loss, thus delaying the fitting of amplification.

There will likely be a heavy reliance on physiological measures during the hearing assessment of patients with intellectual disabilities. One should be mindful of the impact of abnormal middle ear function on otoacoustic emissions (OAE) and ABR. That is, OAEs will be absent in the presence of impacted cerumen or middle ear effusion. Therefore, immittance audiometry will be an important component of the test battery. In a review of ABR studies in persons with Down syndrome, Dille (2003) concluded that ABR testing should be interpreted with caution, because it is likely that those with Down syndrome demonstrate a neural developmental time course that is uniquely different than that of typically developing individuals. Thus, comparing latency-intensity functions to normative values might result in erroneous conclusions. Widen et al. (1987) suggested that the ABR interpretation be based on both threshold of the response and latency-intensity series.

Special Management Considerations

Because of the high incidence of middle ear disease in those with intellectual disability, especially those who are institutionalized or have Down syndrome, it is most efficient to have otologic examinations immediately prior to audiologic assessments. The otologic examinations can serve to ensure that the external canals are free of cerumen and that no active middle ear infection is present. Individuals with Down syndrome, regardless of age, should receive otologic and audiologic monitoring about every 3 months to manage cerumen and middle ear disease. By school age, between 45% and 93% of children with Down syndrome have had pressure-equalizing (PE) tubes (Mitchell et al., 2003; Shott et al., 2001). However, diligent audiologic and otologic monitoring is required because of the high failure and complication rates of PE tubes in those with Down syndrome (Iino et al., 1999).

For those requiring amplification, several issues must be considered. First, the implementation of prescriptive amplification fitting is recommended for all children and adults. Individual or age-appropriate ear acoustics should be taken into account in the hearing aid selection and fitting process. This is accomplished by measurement and application of the real-ear-to-coupler difference (RECD) (see Chapter 40). It is not uncommon for audiologists to use age-average RECD values as opposed to measuring them directly. However, one must consider the potential impact that any craniofacial anomaly (including Down syndrome) might have on this practice. Because of the typically smaller ear canals in individuals with Down syndrome, it is quite likely that an age-average RECD will result in an underestimation of ear canal sound pressure level, thus leading to overamplification.

Second, individuals with craniofacial anomalies or who have intellectual disabilities may have difficulty keeping hearing aids in place for a number of reasons. The use of wig tape or other hearing aid retention devices can help them stay in place behind the patient's ears.

Third, bone-conduction hearing aids may need to be considered for patients with chronic or recurrent middle ear disease or stenotic canals. Bone-anchored hearing aids have been used successfully in some children with Down syndrome (e.g., McDermott et al., 2008). In addition, for those with draining ears who use traditional air-conduction hearing aids, aids may need to be removed temporarily during times of active drainage.

Finally, the fitting of amplification may be delayed in individuals with intellectual disabilities because of other healthcare needs and concerns of the family. However, the earlier the amplification is introduced, the easier it may be to incorporate it into the patient's daily routine and the better the prognosis is for long-term acceptance. The parents or caretakers of patients with intellectual disabilities should receive careful and frequent instruction on the use and care of the amplification devices. Of course, to the extent possible, patients should be included in this educational process and encouraged to participate in the care of their devices.

VISUAL IMPAIRMENT

The combination of vision and hearing deficits may be congenital or acquired later in life. Although often referred to as "deaf-blindness," one should keep in mind that the term "deaf-blind" typically refers to persons with dual sensory impairments who have some residual hearing and usable vision (Miles, 2003). Possible etiologies include syndromes such as:

- CHARGE syndrome—A specific pattern of birth defects represented by the acronym CHARGE: "C" for coloboma, "H" for heart defects, "A" for atresia choanae, "R" for retardation of growth and development, "G" for genitourinary problems, and "E" for ear abnormalities.
- Usher syndrome—The most common condition that involves both hearing and vision problems; an autosomal recessive disorder with primary symptoms that include

hearing loss and progressive retinitis pigmentosa. The vision difficulties include the onset of night blindness, which might become apparent during a child's school years, followed by loss of peripheral vision typically leading to severe low vision or blindness.

- Bardet–Biedl syndrome—A complex disorder that affects many parts of the body including the retina. Individuals with this syndrome have a retinal degeneration similar to retinitis pigmentosa.
- Goldenhar syndrome—A congenital birth defect that involves deformities of the face. Characteristics include a partially formed or totally absent ear (acrotia or anotia) and one missing eye.

Other causative factors for vision and hearing deficits occurring together include congenital prenatal infections (e.g., rubella, toxoplasmosis, herpes, CMV). The rubella epidemic of 1963 to 1965 contributed to the birth of more than 2,500 children with deaf-blindness in the United States. By 2011, there were almost 10,000 children in the United States alone who were considered to be deaf-blind (Teaching Research Institute, 2012). There are also postnatal causes of vision and hearing deficits (e.g., meningitis, asphyxia, stroke). The majority of individuals who are deaf-blind have additional disabilities such as physical impairments, cognitive impairments, and behavior disorders. In fact, more than 60% of individuals who are deaf-blind have intellectual disabilities (National Consortium on Deaf-Blindness, 2007).

Children with hearing loss are two to three times more likely to develop ophthalmic abnormalities than their normal-hearing peers (Guy et al., 2003). The irony is that people with hearing loss have a greater reliance on their vision for communication and environmental monitoring than those with normal hearing. Therefore, audiologists should encourage families of patients with hearing loss to have their vision monitored on a regular basis.

Special Testing Considerations

One of the first things that an audiologist should determine is the patient's preferred sense (typically, it is tactile), and then the audiologist should let the patient explore the test environment for a short period of time or until the patient appears to be comfortable. In addition to the environment, the patient must be given time to "find the audiologist," rather than the audiologist imposing on the patient's space. It is important to remember that individuals who are deaf-blind may explore their environments tactilely, but many are also tactile-defensive, so they must be approached slowly. As the patient becomes more comfortable in the environment and with the test situation, the rules about space and touching may change (Mascia and Mascia, 2003).

During activities that require the audiologist to touch the patient (e.g., otoscopic examination, insertion of earphones), it is recommended that the patient be given as much involvement as possible. That is, the patient should be allowed to examine the equipment (e.g., otoscope, earphones) tactilely. Then, with the patient's hand still in contact, the otoscope, probe, or earphone can be slowly guided to the patient's ear. This process will require patience by the audiologist and may require more than one visit (Mascia and Mascia, 2003).

Auditory responsiveness of individuals who are deaf-blind may be compromised by their lack of curiosity. Thus, they may not turn toward the source of sound for a VRA procedure. As discussed in the section on individuals with intellectual disabilities, pairing the auditory stimuli with a vibrotactile stimulus may be necessary to condition the patient to the task (Mascia and Mascia, 2003). Once the patient has learned to respond consistently to the paired auditory and tactile stimulation, it can be assumed that the task is understood, and the tactile stimulation can be eliminated.

The selection of an appropriate reinforcement for behavioral tasks is critical. As previously mentioned, most individuals classified as deaf-blind have some residual vision. Therefore, even light perception can allow for successful implementation of visual reinforcement. This may require a slight dimming of the test suite lights to enhance the visual reinforcement for the patient. In some cases, a penlight positioned close to the patient and activated in response to a head turn or searching behavior can be implemented. If visual reinforcement is not possible, some patients may enjoy feeling specific textures, vibration, social praise, juice, food bits, or interesting toys. In any case, it will be important to consult with the patient's caregivers or teachers to assist in determining a desirable reinforcement.

It is also important when behaviorally assessing the hearing of a patient who is deaf-blind to determine an appropriate response to the stimulus. Parents, caregivers, and teachers may all be valuable resources in evaluating what kind of motor response can be expected from the patient in response to sound. Some possible responses include a head turn, reaching, arm raise, finger raise, or leg swing. Additionally, it may often be necessary to physically "show" the patient when and how to perform the response by manipulating the patient's hand, leg, or foot into place when the auditory stimulus is presented. This assistance can gradually be decreased using successive approximations until the child is able to respond with no cueing or assistance from the clinician.

Special Management Considerations

It is likely that individuals with dual hearing and vision impairments will welcome the use of amplification when indicated. After all, the majority of this population has some degree of residual hearing ability, and enhancement of hearing could serve as an important supplement to less-than-optimal visual input. A survey of clinical audiologists confirmed the belief that those with vision and hearing difficulties could potentially benefit more from amplification

than those with hearing loss alone (Tharpe et al., 2001). In addition, amplification for those with dual impairments has a role beyond that of only enhancing speech perception ability (Wiener and Lawson, 1997). That is, audiologists need to consider more than just enhancing speech perception and must also focus on the role hearing has in orientation and mobility, which is essential to the development of successful independent living skills (Tharpe et al., 2002).

Experts in the rehabilitation of visual impairment use the term "orientation and mobility" to refer to one's location relative to environmental features and moving safely through one's environment. Much research has been conducted on hearing aid specifications designed to enhance speech perception ability, but considerably less research exists on enhancing the detection of environmental auditory cues. It is unknown whether there is a combination of hearing aid characteristics that can be used to enhance speech perception and also improve detection of environmental cues or that can possibly affect one or the other adversely. The need for an integrated approach is apparent for individuals with dual sensory impairments who need to coordinate the aspects of guiding, route instruction, and verbal communication. Even the limited research that has been done on sound localization with hearing aids has not considered the specific spatial hearing needs of persons with visual impairments. Because speech recognition is based mostly on frequencies above 500 Hz, it is common for hearing aids to attenuate frequencies below a cutoff level in the range of 500 to 1,000 Hz. This low-frequency cutoff is designed to reduce background sounds that can interfere with speech perception. However, that frequency range contains critical information for orientation and mobility with respect to traffic sounds (Wiener and Lawson, 1997) and environmental surfaces, such as walls (Ashmead et al., 1998). A third important property of hearing aids is the flexibility to switch between different programs. That is, hearing aids that are programmable can be set to optimize listening in different environments. Assuming that different listening needs require different hearing aid settings for optimal perception, this flexibility will be important to consider in rehabilitation strategies for those with vision and hearing impairments.

Numerous investigators have found that directional microphones provide an advantage when listening to speech in noise under laboratory conditions. However, omnidirectional microphones appear to enhance localization ability under certain laboratory conditions and, perhaps, in real-world settings (Tharpe et al., 2002). A considerable amount of research is still needed to enhance our knowledge in this area. In the meantime, one should be cautious when selecting microphone options for use by individuals with significant vision and hearing deficits. It appears reasonable to offer a switchable directional/omnidirectional microphone option to those with significant visual impairments who must rely on their hearing for getting around their environments safely. Instruction regarding careful head positioning during communication, especially when using a directional microphone, appears warranted.

SUMMARY

The assessment and management of individuals with multiple disabilities is a great challenge for audiologists. However, with some knowledge of the characteristics of a number of disabilities, early planning for and adjustments to diagnostic procedures, and careful consideration of individual and family needs, one can obtain valid and reliable test results that lead to meaningful audiologic management.

Part of facing this challenge requires recognizing and admitting that no one can be an expert on all disabilities. With these patients, probably more than most, we must acknowledge that our expertise may be limited and that we must work with a multidisciplinary or, optimally, with an interdisciplinary team of professionals, the patient, and the patient's family in developing effective diagnostic and management strategies.

Finally, as with all patients, audiologists must consider the patient's and family's priorities as they relate to the hearing loss. For example, those with multiple disabilities may have other significant medical needs requiring substantial time and emotional energy. As such, the family may choose to defer the management of hearing loss until a time when they can more readily accept the challenge. Audiologists must be respectful of a family's decisions and be prepared to support and encourage families in their choices.

FOOD FOR THOUGHT

1. You are suspicious that a child you are evaluating in clinic might have ASD. What additional tests/screenings might you conduct in addition to your traditional audiologic testing and what referrals might you make to other professionals?
2. You see that there is a 28-year-old patient with Down syndrome on your schedule for next week. This patient has used hearing aids for a few years. What pre-visit recommendations would you have for this patient's caregiver to prepare for this appointment?
3. You are attending an interdisciplinary team meeting to discuss a 9-year-old child who has hearing loss and significant vision problems that are not correctable with glasses. What are the most critical pieces of information you need from other team members and what is the most critical information for you to share with the others?

ACKNOWLEDGMENT

The authors thank Jamie L. Watson for her assistance in the research and preparation of the original version of this chapter.

KEY REFERENCES

thePoint A full list of references for this chapter can be found at http://thePoint.lww.com. Below are the key references for this chapter.

American Psychiatric Association. (2013) *Diagnostic and Statistical Manual of Mental Disorders.* 5th ed. Arlington, VA: American Psychiatric Publishing.

Ashmead DH, Wall RS, Eaton SB, Ebinger KA, Snook-Hill MM, et al. (1998) Echolocation reconsidered: Using spatial variations in the ambient sound field to guide locomotion. *J Vis Impair Blind.* 92, 615–632.

Baghdadli A, Picot MC, Pascal C, Pry R, Aussilloux C. (2003) Relationship between age of recognition of first disturbances and severity in young children with autism. *Eur Child Adolesc Psychiatry.* 12 (3), 122–127.

Baio J. (2012) Prevalence of autism spectrum disorders: Autism and Developmental Disabilities Monitoring Network, 14 sites, United States, 2008. *MMWR Surveill Summ.* 61 (3), 1–19.

Bertrand J, Mars A, Boyle C, Bove F, Yeargin-Allsop M, Decoufle P. (2001) Prevalence of autism in a United States population: the Brick Township, New Jersey, investigation. *Pediatrics.* 108, 1155–1161.

Brask BH. (1972) A prevalence investigation of childhood psychoses. In: *Nordic Symposium on the Comprehensive Care of Psychotic Children.* Oslo: Barnepsykiatrist Forening; pp 145–153.

Chawarska K, Klin A, Paul R, Macari S, Volkmar F. (2009) A prospective study of toddlers with ASD: Short term diagnostic and cognitive outcomes. *J Child Psychol Psychiatry.* 50 (10), 1235–1245.

Chilosi AM, Comparini A, Scusa MF, Berrettini S, Forli F, Battini R, et al. (2010) Neurodevelopmental disorders in children with severe to profound sensorineural hearing loss: A clinical study. *Dev Med Child Neurol.* 52 (9), 856–862.

Cristobal R, Oghalai JS. (2008) Hearing loss in children with very low birth weight: Current review of epidemiology and pathophysiology. *Arch Dis Child Fetal Neonatal Ed.* 93 (6), F462–F468.

Dean J, Harris F. (2003) Adaptive hierarchical test procedures for developmentally delayed adults: Taking the "difficult" out of "difficult to test." *Semin Hear.* 24, 247–262.

Diefendorf AO. (2003) Behavioral hearing assessment: Considerations for the young child with developmental disabilities. *Semin Hear.* 24, 189–200.

Dille MF. (2003) Perspectives on the audiological evaluation of individuals with Down syndrome. *Semin Hear.* 24, 201–210.

Fombonne E. (2003) Epidemiological surveys of autism and other pervasive developmental disorders: An update. *J Autism Dev Disord.* 33 (4), 365–382.

Fortnum HM, Stacey PC, Summerfield AQ. (2006) An exploration of demographic bias in a questionnaire survey of hearing-impaired children: Implications for comparisons of children with and without cochlear implants. *Int J Pediatr Otorhinolaryngol.* 70 (12), 2043–2054.

Gallaudet Research Institute. (2011) *Regional and National Summary Report of Data from the 2009–2010 Annual Survey of Deaf and Hard of Hearing Children and Youth.* Washington, DC: Gallaudet Research Institute, Gallaudet University.

Greenberg DB, Wilson WR, Moore JM, Thompson G. (1978) Visual reinforcement audiometry (VRA) with young Down's syndrome children. *J Speech Hear Disord.* 43, 448–458.

Groen WB, van Orsouw L, ter Huurne N, Swinkels S, van der Gaag RJ, Buitelaar JK, et al. (2009) Intact spectral but abnormal temporal processing of auditory stimuli in autism. *J Autism Dev Disord.* 39 (5), 742–750.

Guy R, Nicholson J, Pannu SS, Holden R. (2003) A clinical evaluation of ophthalmic assessment in children with sensorineural deafness. *Child Care Health Dev.* 29, 377–384.

Iino Y, Imamura Y, Harigai S, Tanaka Y. (1999) Efficacy of tympanostomy tube insertion for otitis media with effusion in children with Down Syndrome. *Int J Pediatr Otorhinolaryngol.* 49, 143–149.

Jonsdottir GM, Georgsdottir I, Haraldsson A, Hardardottir H, Thorkelsson T, Dagbjartsson A. (2012) Survival and neurodevelopmental outcome of ELBW children at 5 years of age. *Acta Paediatr.* 101 (7), 714–718.

Kaga K, Shindo M, Tamai F, Tanaka Y. (2007) Changes in auditory behaviors of multiply handicapped children with deafness after hearing aid fitting. *Acta Oto Laryngol.* 127 (S559), 9–12.

Kwakye LD, Foss-Feig JH, Cascio CJ, Stone WL, Wallace MT. (2011) Altered auditory and multisensory temporal processing in autism spectrum disorders. *Front Integr Neurosci.* 4, 129.

La Malfa G, Lassi S, Bertelli M, Salvini R, Placidi GF. (2004) Autism and intellectual disability: A study of prevalence on a sample of the Italian population. *J Intellect Disabil Res.* 48 (3), 262–267.

Lieberman LJ, Volding L, Winnick JP. (2004) Comparing motor development of deaf children of deaf parents and deaf children. *Am Ann Deaf.* 149, 281–289.

Lloyd LL, Spradlin JE, Reid MJ. (1968) An operant audiometric procedure for difficult-to-test patients. *J Speech Hear Disord.* 33, 236–245.

MacFarland SZC. (2003) Current trends and issues in understanding adults with developmental disabilities. *Semin Hear.* 24, 171–178.

Mandell DS, Novak MM, Zubritsky CD. (2005) Factors associated with age of diagnosis among children with autism spectrum disorders. *Pediatrics.* 116, 1480–1486.

Marcell MM, Cohen S. (1992) Hearing abilities of Down syndrome and other mentally handicapped adolescents. *Res Dev Disabil.* 13, 533–551.

Mascia J, Mascia N. (2003) Methods and strategies for audiological assessment of individuals who are deaf-blind with developmental disabilities. *Semin Hear.* 24, 211–221.

Matkin N. (1977) Assessment of hearing sensitivity during the preschool years. In: Bess FH, ed. *Childhood Deafness.* New York, NY: Grune & Stratton.

May ME, Kennedy CH. (2010) Health and problem behavior among people with intellectual disabilities. *Behav Anal Pract.* 3 (2), 4–12.

McDermott AL, Williams J, Kuo MJ, Reid AP, Proops DW. (2008) The role of bone anchored hearing aids in children with Down syndrome. *Int J Pediatr Otorhinolaryngol.* 72 (6), 751–757.

Mehta UC, Patel I, Castello FV. (2004) EEG sedation for children with autism. *J Dev Behav Pediatr.* 25, 102–104.

Miles B. (2003) *Overview on Deaf-Blindness. DB-Link, The National Information Clearinghouse on Children Who Are Deaf-Blind.* Sands Point, NY: Helen Keller National Center.

Mitchell RB, Call E, Kelly J. (2003) Ear, nose, and throat disorders in children with Down syndrome. *Laryngoscope.* 113, 259–263.

Moore JM. (1995) Behavioral assessment procedures based on conditioned head-turn response for auditory detection and

discrimination with low-functioning children. *Scand Audiol.* 24 (suppl 41), 36–42.

National Consortium on Deaf-Blindness. (2007) Children who are deaf-blind. *Pract Perspect.* 2. Available online at: https://nationaldb.org/.

Oghalai JS, Caudle SE, Bentley B, Abaya H, Lin J, Baker D, et al. (2012) Cognitive outcomes and familial stress after cochlear implantation in deaf children with and without developmental delays. *Oto Neurotol.* 33 (6), 947–956.

Ozonoff S, Iosif AM, Baguio F, Cook IC, Hill MM, Hutman T, et al. (2010) A prospective study of the emergence of early behavioral signs of autism. *J Am Acad Child Adolesc Psychiatry.* 49 (3), 256–266.

Parker S, Mai C, Canfield M, Rickard R, Wang Y, Meyer R, et al. (2010) Updated national birth prevalence estimates for selected birth defects in the United States, 2004–2006. *Birth Defects Res A Clin Mol Teratol.* 88, 1008–1016.

Picard M. (2004) Children with permanent hearing loss and associated disabilities: Revisiting current epidemiological data and causes of deafness. *Volta Rev.* 104, 221–236.

Porter H, Tharpe AM. (2010) Hearing loss among persons with Down syndrome. In Urbano RC, ed. *International Review of Research in Mental Retardation: Health Issues in Down Syndrome.* New York, NY: Academic Press.

Rice C. (2009) Prevalence of autism spectrum disorders: Autism and Developmental Disabilities Monitoring Network, United States, 2006. *MMWR Surveill Summa.* 58 (10), 1–20.

Robertson CM, Watt MJ, Yasui Y. (2007) Changes in the prevalence of cerebral palsy for children born very prematurely within a population-based program over 30 years. *J Am Med Assoc.* 297 (24), 2733–2740.

Robins DL, Fein D, Barton ML, Green JA. (2001) The Modified Checklist for Autism in Toddlers: An initial study investigating the early detection of autism and pervasive developmental disorders. *J Autism Dev Disord.* 31, 131–144.

Robinson RO. (1973) The frequency of other handicaps in children with cerebral palsy. *Dev Med Child Neurol.* 15, 305–312.

Roush J, Holcomb MA, Roush PA, Escolar ML. (2004) When hearing loss occurs with multiple disabilities. *Semin Hear.* 25, 333–345.

Rutter M. (2005) Incidence of autism spectrum disorders: Changes over time and their meaning. *Acta Paediatr.* 94, 2–15.

Samson F, Hyde KL, Bertone A, Soulières I, Mendrek A, Ahad P, et al. (2011) Atypical processing of auditory temporal complexity in autistics. *Neuropsychologia.* 49 (3), 546–555.

Shott SR, Joseph A, Heithaus D. (2001) Hearing loss in children with Down syndrome. *Int J Pediatr Otorhinolaryngol.* 61 (3), 199–205.

Siegel B. (1996) *World of the Autistic Child: Understanding and Treating Autistic Spectrum Disorders.* New York, NY: Oxford University Press; p 107.

Smith RJH, Bale JF, White KR. (2005) Sensorineural hearing loss in children. *Lancet.* 365, 5–11.

Stoinska B, Gadzinowski J. (2011) Neurological and developmental disabilities in ELBW and VLBW: Follow-up at 2 years of age. *J Perinatol.* 31 (2), 137–142.

Synnes AR, Anson S, Baum J, Usher L. (2012) Incidence and pattern of hearing impairment in children with ≤800 g birthweight in British Columbia, Canada. *Acta Paediatr.* 101 (2), e48–e54.

Teaching Research Institute. (2012) National deaf-blind child count summary. Available online at: https://nationaldb.org/search/search/?sitesearch=national+child+count.

Tharpe AM, Ashmead DH. (1993) A computer simulation technique for assessing pediatric auditory test protocols. *J Am Acad Audiol.* 4, 2.

Tharpe AM, Ashmead DH, Ricketts TA, Rothpletz AM, Wall R. (2002) Optimization of amplification for deaf-blind children. In: Seewald RC, Gravel JS, eds. *A Sound Foundation through Early Amplification. 2001: Proceedings of the Second International Conference.* St. Edmundsbury, UK: St. Edmundsbury Press.

Tharpe AM, Bess FH, Sladen D, Schissel H, Couch S, Schery T. (2006) Auditory characteristics of children with autism. *Ear Hear.* 27, 430–431.

Tharpe AM, Fino-Szumski MS, Bess FH. (2001) Survey of hearing aid fitting practices for children with multiple impairments. *Am J Audiol.* 10, 1–9.

Thompson G, Wilson WR, Moore JM. (1979) Application of visual reinforcement audiometry (VRA) to low functioning children. *J Speech Hear Disord.* 44, 80–90.

Van Bourgondien ME, Mesibov GB, Dawson G. (1987) Pervasive developmental disorders: Autism. In: Wolraich ML, ed. *The Practical Assessment and Management of Children with Disorders of Development and Learning.* Chicago, IL: Yearbook Medical Publishers; pp 326–351.

Washburn LK, Dillard RG, Goldstein DJ, Klinepeter KL, O'Shea TM. (2007) Survival and major neurodevelopmental impairment in extremely low gestational age newborns born 1990–2000: A retrospective cohort study. *BMC Pediatr.* 7 (1), 20.

Werner LA, Mancl LR, Folsom RC. (1996) Preliminary observations on the development of auditory sensitivity in infants with Down syndrome. *Ear Hear.* 17, 455–468.

Widen, JE, Folsom, RC, Thompson, G, Wilson, WR. (1987) Auditory brainstem responses in young adults with Down syndrome. *Am J Ment Deficits.* 91 (5), 472–479.

Wiener WR, Lawson GD. (1997) Audition for the traveler who is visually impaired. In: Blasch BB, Wiener WR, Welsh RL, eds. *Foundations of Orientation and Mobility.* 2nd ed. New York, NY: AFB Press.

Wilson-Costello D, Friedman H, Minich N, Fanaroff AA, Hack M. (2005) Improved survival rates with increased neurodevelopmental disability for extremely low birth weight infants in the 1990s. *Pediatrics.* 115, 997–1003.

Yeargin-Allsopp M, Rice C, Karapurkan T, Doernberg N, Boyle C, Murphy C. (2003) Prevalence of autism in a US metropolitan area. *J Am Med Assoc.* 289, 49–55.

Yilmaz Y, Degirmenci S, Akdas F, Külekçi S, Çiprut A, Yüksel S, et al. (2001) Prognostic value of auditory brainstem response for neurologic outcome in patients with neonatal indirect hyperbilirubinemia. *J Child Neurol.* 16, 772–775.

Zigman W, Schupf N, Sersen E, Silverman W. (1995) Prevalence of dementia in adults with and without Down syndrome. *Am J Ment Retard.* 100, 401–412.

CHAPTER 32

Noise Exposure

Brian Fligor, Marshall Chasin, and Rick Neitzel

HISTORY OF THE STUDY OF NOISE EXPOSURE

Noise can be defined simply as "unwanted sound." Noise—or even desirable sound—above a given sound pressure level, and experienced over a sufficient duration of time, has the capacity to cause temporary or permanent changes in the structure and function of the auditory system. These changes result in the disruption of the normal function of the hearing apparatus, primarily through cochlear damage resulting in sensory/neural hearing loss; this is referred to as noise-induced hearing loss (NIHL). Unlike many other causes of sensory/neural hearing loss, NIHL is a fairly recent phenomenon in the history of human existence. Few naturally occurring sounds are of sufficient intensity to cause NIHL without also being a catastrophic threat to individuals—volcano eruptions, for instance. The human auditory system is amazingly refined to detect very soft sounds (such as a snap of a twig from a predator or the subtle acoustic difference between the /f/ and unvoiced /th/). A human voice can hit very high sound levels (e.g., shouting near an individual's ear produces levels of approximately 115 dBA), but it does not have the ability to sustain these levels continuously for long durations; one must breathe, and hoarseness ensues with continued vocal fold abuse. Thus, the human auditory system is designed to function over a wide dynamic range (from the threshold of hearing to roughly 75 dBA) without sustaining any noise-induced changes. However, our ability to produce intense levels of sound as a by-product of industrial, transportation, and economic activities (as well as from recreational pursuits) has outpaced our auditory system's evolution; NIHL is therefore almost entirely man made. Combating its ill-effects through prevention or mitigation should also be possible as a human endeavor.

There are accounts of hearing impairment in the Middle Ages in church bell ringers, miners, and blacksmiths (Berger et al., 2000). In the 19th century, medical literature made reference to "boiler-maker's deafness" and "blacksmith's deafness." NIHL began affecting a large proportion of the population with the onset of the Industrial Revolution (and the automated machines of industry), and later with surviving veterans of World War II returning home after being exposed to gunfire, explosions, and military machinery and aircraft noise. The prevalence of NIHL (particularly in the military) in the 1940s and 1950s was a major driving impetus to the massive expansion of the profession of audiology, which at the time was in its fledgling state.

MEASUREMENT OF NOISE EXPOSURE

Quantitative measurement of noise, conducted through use of sound level meters and noise dosimeters, is a relatively recent phenomenon and represents the foundation of all modern hearing conservation programs. Although sound level meters were in use for research purposes as early as the 1920s, they were not standardized until the following decade, and the first commercially available units were not introduced until the 1960s. Personal dosimeters, widely considered to be the gold standard for personal noise exposure assessment, did not become widely available until the 1970s. For evaluation of occupational noise exposures, sound level meters are typically used to make measurements of specific areas or short-term measurements on stationary workers whose noise levels are relatively steady over time. Dosimeters, which are small units designed to be worn by individuals for extended periods, may be used in these situations as well as in environments with time-varying levels and high worker mobility. Some dosimeters are also equipped with alarms that notify workers when their exposures exceed some predefined level—for example, one-half of their allowable daily noise dose—and other dosimeters are capable of making measurements inside workers' hearing protection.

Measurements with sound level meters are typically made as snapshots over short periods of time (from a few minutes to a fraction of an hour). Such measurements can be quite useful for determining whether or not hearing protection is needed in a particular area, as well as to develop a "noise map," that is, a facility map denoting noise levels in specific areas or in reference to specific machines or operations. Measurements made via dosimeter are typically made over an entire workshift and represent the gold standard, for example, the measured dose on a specific worker. Both types of instruments may be used to measure point-in-time

broadband sound pressure levels (in decibels), and dosimeters and integrating sound level meters may also be used to integrate broadband sound pressure levels into an average level over the examined time period. For 8-hour measurements, these integrated levels are referred to as time-weighted averages (TWAs). More sophisticated sound level meters have the additional capability of assessing frequency spectra in octave bands, 1/3 octave bands, and even narrow bands.

Regardless of the type of instrument used to measure noise, a single measurement is typically insufficient to adequately characterize noise exposure, and repeated measurements—under differing work conditions, if possible—are preferred. An integrated noise exposure assessment is the best approach to achieve a comprehensive understanding of noise exposures within a given facility or company; in other words, they collect area measurements, short-term measurements on workers, and full-shift measurements on workers. Measurements are becoming easier and less disruptive to administer because of continuing trends in miniaturization of dosimeters, and most modern dosimeters (and many sound level meters) also have powerful datalogging capabilities, which offer the ability to review and analyze minimum, average, and maximum noise levels with very high resolution (datalogging intervals of as little as 1 second).

HISTORICAL EVIDENCE OF AUDITORY DAMAGE FROM NOISE

One of the earliest studies of NIHL from workplace noise was a 1965 report of hearing thresholds of current and retired weavers who had a well-documented history of steady noise exposure for up to 40 years (Taylor et al., 1965). The pattern of hearing loss showed wide variability in hearing thresholds across individuals, but showed greatest hearing loss at 4,000 Hz (compared to lower and higher frequencies) with the most rapid onset of hearing loss occurring in the first 12 years, followed by continued (but slower) progression of hearing loss (Rosenhall et al., 1990). Numerous cross-sectional studies of noise-exposed workers have corroborated these classic findings, that hearing loss is greatest around 4,000 Hz and hearing loss progresses and includes lower and higher frequencies with increasing exposure.

The Environmental Protection Agency (EPA) estimated that 9.1 million workers in the United States were exposed to levels at or above 85 dBA (EPA, 1981) across a variety of industries, including manufacturing, mining, construction, agriculture, and transportation. From 1981 to 1983, the National Institute for Occupational Safety and Health (NIOSH) conducted noise surveys across a wide range of workplaces in the United States (excluding most mining operations) and estimated 16.9% of the workforce examined was exposed to levels at or above 85 dBA, although in some industries the fraction of potentially overexposed workers was much higher. For instance, follow-up surveys in the mining industry conducted from 1984 to 1989 estimated that 84.5% of miners were potentially exposed to noise in excess of 85 dBA (NIOSH, 1998).

CURRENT PERSPECTIVES ON THE PATHOPHYSIOLOGY OF NOISE-INDUCED HEARING LOSS

Sensory/neural shifts in puretone thresholds resulting from noise exposure are generally classified as noise-induced temporary threshold shift (NITTS or TTS) and noise-induced permanent threshold shift (NIPTS or PTS). Additional injuries associated with NIHL include tinnitus, hyperacusis, and abnormal pitch perception. The focus of this chapter will be on noise-induced threshold shift, as other chapters deal with these other injuries in great detail. This is not to minimize the significance of these other injuries. As will be described later in this chapter, there is a relationship between TTS and PTS, but decades of research attempting to use TTS to predict PTS have fallen far short of developing robust models for hearing loss risk estimation (Melnick, 1991). It is now better understood that the underlying mechanisms giving rise to TTS and PTS are markedly different, and these differences might account for limitations in predicting PTS from TTS.

Gradually developing cochlear hearing loss can result from long-term exposure to moderately intense noise ranging from roughly 75 to 78 dBA (EPA, 1974; Melnick, 1991; Prince et al., 1997) to 132 dB peak SPL (Price and Kalb, 1991). Those factors that influence the degree of hearing loss are the sound level of the exposure as well as the duration of the exposure. In addition, the spectrum of the offending sound does influence the relative risk for hearing loss. The primary site of lesion in gradually developing NIHL is the death of outer hair cells (OHCs) (Henderson et al., 2006), resulting in a 40- to 60-dB hearing loss (HL). The frequency region that is typically affected first is between 3,000 and 6,000 Hz (most often in adult males, 4,000 Hz has the poorest hearing threshold), with better hearing thresholds at 2,000 Hz and below, and at 8,000 Hz (and above). With further exposure, hearing loss extends to lower and higher frequencies, and degrees of hearing loss greater than 60 dB HL are observed as inner hair cells (IHCs) and auditory nerve fibers are damaged. See Figure 32.1 for an example of significant NIHL and stereotypical bilateral 4,000-Hz "noise notch" in a 42-year-old professional drummer who has 26 years of experience, 15 years of which were without hearing protection. He used combination hearing aid–tinnitus masker devices, as well as undergoing extensive tinnitus management, to cope with his hearing loss and tinnitus.

In most cases, gradually developing PTS develops insidiously, as the challenges associated with communication and changes in perception of music often are not obvious until the degree of hearing loss is quite significant. The degree of

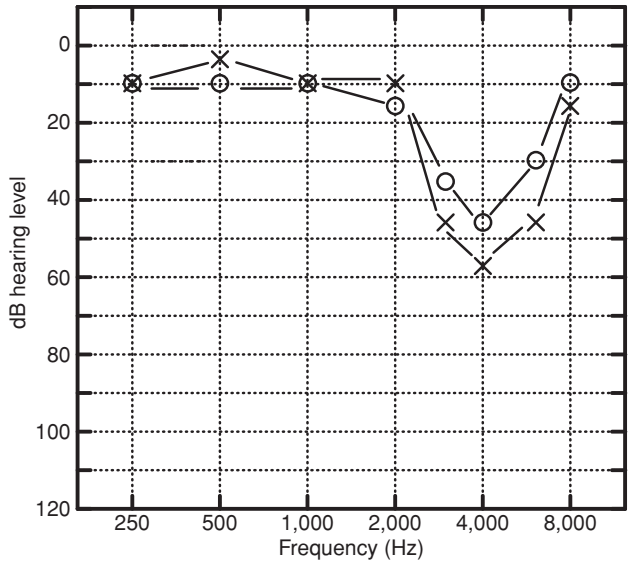


FIGURE 32.1 Audiogram of a 42-year-old drummer with significant noise exposure and noise-induced permanent threshold shift and tinnitus. [Courtesy of the Musicians' Clinics of Canada.]

hearing loss is cumulative throughout one's life, and by the time NIHL has direct impact on communication, considerable damage has been done that might have been avoided with earlier detection and mitigation of noise exposure.

In contrast, damage to the auditory system following a single, extremely intense, acoustic event may be marked by a large hearing loss that may not fully resolve to baseline. Such an event is commonly referred to as acoustic trauma and can affect a wide range of cochlear structures, such as OHCs and IHCs, supporting cells, the tectorial membrane, and the cochlear partition.

The mechanisms of cell death in both gradually developing PTS and acoustic trauma, the implications of these mechanisms for degree and range of frequencies affected by the hearing loss, and possible drug therapies for intervention will be covered in greater detail later in this chapter.

APPLICATION OF WEIGHTING FILTERS: dB SPL, dBA, AND dBC

Units of sound are typically expressed as a level that is a logarithm of the ratio between the sound pressure of interest (in Pascals) and the sound pressure at the threshold of human hearing at 1,000 Hz (i.e., 20 μPa). This ratio is expressed in decibels (dB) of sound pressure level (SPL). By definition, it is not weighted as a function of frequency. However, human hearing is most sensitive between 1,000 and 5,000 Hz and is less sensitive at lower and higher frequencies. Instrumentation used to measure sound exposures have built-in weighting filters that de-emphasize the relative contribution of sound energy at lower frequencies and give very slight emphasis to the frequency range that is most sensitive. The shape of the A-weighted filter is based on the 40-phon curve in the Fletcher–Munson equal loudness contours. Sound with the majority of energy between 1,000 and 5,000 Hz and very little energy below this range will have nearly the same level in both dB SPL and dBA. However, sound with the majority of energy in low frequencies (for instance, 200 Hz and below) will have a much higher level in dB SPL than in dBA. Those seminal cross-sectional studies that have provided the data relating degree of hearing loss with past noise exposure measured that exposure in dBA (with A-weighted filter applied). As well, the measures were made in the free field (or diffuse field), or at the shoulder of an individual, and so the measures of sound exposure do not account for individual differences, such as differences in ear canal acoustics, across individuals. Figure 32.2 shows a comparison of A- and C-weighting filters relative to unweighted dB SPL. The C-weighting filter (dBC) is quite similar to the unweighted dB SPL except in the very low frequency region.

There is a controversy whether the use of the A-weighted filter is appropriate in measuring noise levels that may pose a hazard. The A-weighted filter is based on the 40-phon equal loudness contour; 40 dB SPL is equal to 40 phons at 1,000 Hz, but roughly 60 dB SPL is needed to achieve the same loudness perception (40 phons) at 100 Hz (because of the nature of reduced perception of loudness to low-frequency sound energy at this moderately low sound level). By contrast, the C-weighted filter is based on the 100-phon equal loudness contour; 100 dB SPL is equal to 100 phons at 1,000 Hz, and 100 dB SPL is equal to 100 phons at 100 Hz. This is due to the fact that the Fletcher–Munson equal loudness contours flatten out across the frequency spectrum as the sound level increases. Those sound exposures that place hearing at risk are much closer to the 100-phon equal loudness contour (C-weighting) than the 40-phon level (A-weighting). Regardless, given that our current models of hearing loss risk as a function of

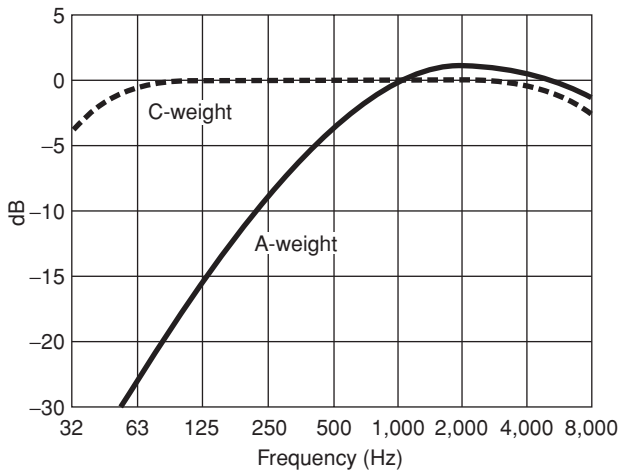


FIGURE 32.2 A-weight and C-weight filters that de-emphasize certain frequencies to which the human ear is less sensitive, relative to "0 dB" which is unweighted dB SPL.

sound exposure are based on exposures measured in dBA, and that we cannot ethically repeat studies of unprotected noise-exposed workers, dBA continues to be the required unit of measure of sound when considering risk for NIHL. The proponents of using dBC have advocated, with some success, the use of measures reported in dBC (or unweighted dB SPL) for very high level transient sounds (for instance, impact or impulse noise), so as to not de-emphasize the contribution of very intense low-frequency sound that would happen if high-level transients were reported in dBA.

WHY A NOTCH AT 4,000 Hz?

In humans, the frequency of maximum cochlear damage is one-half to one octave above the frequency of maximum stimulation (Royster, 1996). This phenomenon has to do with the angle of curvature of the human cochlea as well as less blood perfusion in the basal end of the cochlea compared to the apex. The human external ear (pinna and ear canal) influences the physical properties of sound outside the head (i.e., in the diffuse field) by resonating at frequencies between 2,000 and 4,000 Hz, depending on the volume and the length of the ear canal; for larger adult ears the maximum ear canal resonance, as measured with a probe microphone, is 2,600 to 3,000 Hz (Schotland, 1996). In children, with shorter ear canals with a smaller diameter, this ear canal resonance is higher in frequency. This resonance serves to amplify sound by 15 to 25 dB relative to the diffuse field (for instance, as measured at the shoulder) at the resonant frequency.

Acousticians and engineers have referred to this resonance as the transfer function of the open ear (TFOE) or the external ear transfer function and is known to audiologists as the real ear–unaided response (REUR). When fitting hearing aids, placement of an earmold results in disruption of this normal ear canal resonance, resulting in insertion loss. The real ear–aided response (REAR) must provide amplification to compensate for the insertion loss, just to get back to the sound level that would arrive at the eardrum without the earmold or hearing aid in place. For broadband sound, the result of the TFOE (REUG) is an overall level measured at the eardrum roughly 7 dB higher than measured at the shoulder. Given that most environmental sound is relatively broadband, the frequency range of maximum stimulation is roughly one-half to one octave below 4,000 Hz. This is another reason why the 4,000-Hz frequency region is the most susceptible to damage.

HISTORICAL DEVELOPMENT OF NOISE EXPOSURE MODELS

There are two types of studies typically performed to assess the potential effects of noise on the auditory system. These consist of (1) large-scale studies of populations of humans who were exposed to well-defined noise levels for well-defined durations and (2) laboratory-based animal studies. With actual humans, the most common type of experiment is to elicit a TTS in a subject's puretone thresholds after exposure to a significant level of noise or music and for a well-defined time period. As stated earlier, long-term studies of workers over many years can also provide evidence of the nature of permanent hearing loss (PTS). With laboratory-based animal studies, PTS can be studied in a more direct and controlled environment. A drawback of the large-scale studies, such as in industrial settings, is the possibility of contamination of the results by noncontrolled factors such as middle-ear disorders, smoking, and other forms of unreported (recreational) noise and music exposure; and a drawback of well-controlled laboratory studies is the limitations to generalizing results to human hearing loss.

Relationship between TTS and PTS

It would be tempting to be able to derive a relationship between TTS and PTS. If an individual worker were quite susceptible to TTS, then perhaps there is something that can be said about his or her future PTS. As far back as 1966, the Committee on Hearing and Bioacoustics (CHABA) attempted to establish a model that would define a relationship between TTS and PTS. In the words of CHABA, "If any single band exceeds the damage-risk contours specified, the noise can be considered as potentially unsafe" (Kryter et al., 1966).

For a number of reasons, CHABA made several assumptions to derive the damage-risk contours. One was that the recovery from TTS was only based on the magnitude—the larger the TTS, the longer it took to resolve. However, subsequent research demonstrated that recovery depended on both the duration and the sound level of the noise exposure (Melnick, 1991). Another erroneous assumption that was made is that an intermittent noise exposure with significant quiet periods was less damaging than a steady-state noise exposure. Again, for the purposes of the damage-risk contours, this assumption was shown to be false (Ward et al., 1976). It does appear that intermittent noise exposure does reduce hazard, but that is about all that can be said.

Some excellent research studies were performed in the 1980s and 1990s showing that there does not appear to be a high correlation between TTS and PTS. A person who demonstrates a large TTS after an exposure to noise or music is not any more nor any less susceptible to permanent hearing loss years later. TTS is simply not a predictor of PTS (Henderson et al., 1993). The association that can be made between TTS and PTS is that if a sound exposure is sufficient to cause a TTS (of any degree), then it is sufficient to cause a PTS (although not necessarily of the same magnitude as the TTS) (Melnick, 1991).

PTS and Some Historical Models

Between 1968 and 1973, there were a number of large-scale field studies on the relationship between noise exposure

TABLE 32.1

Summary of Five Studies on Predicted PTS (in dB) at 4,000 Hz for Three Exposure Levels

	Passchier-Vermeer	Robinson	Baughn	NIOSH	ISO R-1999
85 dBA	8	6	9	5	6
90 dBA	15	12	14	11	11
95 dBA	23	18	17	20	21

and PTS (Baughn, 1973; Lempert and Henderson, 1973; Passchier-Vermeer, 1968; Robinson, 1971). The Passchier-Vermeer, Robinson, and Baughn studies formed the basis of the 1973 US EPA's Criteria Document and noted very little (average) PTS for exposures below 85 dBA if exposed for 8 hours a day for 40 years. It should be pointed out though that this average PTS was calculated for 500, 1,000, and 2,000 Hz. There was poor correlation for hearing loss at 4,000 Hz, especially for higher exposure levels.

The Lempert and Henderson (1973) study formed the basis for the NIOSH model and is in good agreement with the previous studies at lower exposure levels, but tended to predict a greater PTS (average of 500, 1,000, and 2,000 Hz) at higher exposure levels.

A more recent model is based on the International Organization for Standardization (ISO) standard R-1999 (1990) which appears to be in good agreement with the previous models. Indeed, "models such as ISO R-1999 are sufficiently accurate to support the needs of most regulators, administrators, and others who need rough predictions on the effects of noise on groups of workers" (Johnson, 1991, p 174). There are some criticisms of ISO R-1999 and these mostly revolve around the interaction between noise exposure and presbycusis. Rosenhall et al. (1990) showed that by age 79, there was no longer a difference between those who have been exposed to noise and those who had not. That is, eventually presbycusis becomes a more dominant factor.

Table 32.1 summarizes the PTS at 4,000 Hz for a number of models for three exposure levels of 85, 90, and 95 dBA. There are limited data available in large-scale databases for exposure levels below 85 dBA.

PTS and Exchange Rates

The exposure–response or dose–response (also known as the damage-risk contours from the 1966 CHABA document) refers to contours or "equal risk" of PTS for a given average sound level (dBA) for a given duration (hours). That is, a relationship exists between the average exposure in dBA and the length of exposure in hours. This relationship is called the exchange rate (or time–intensity trading ratio). A 3-dB exchange rate means that there is an equal risk if the sound level is increased by 3 dB but for only half the amount of exposure time; in other words, for each increase in sound level of 3 dB, the damage-risk is doubled (and halved for an equivalent decrease of 3 dB). A 5-dB exchange rate implies that the risk is doubled for every 5-dB increase in exposure level. Table 32.2 shows the relationship for equivalent exposures for both a 3-dB exchange rate (NIOSH) and a 5-dB exchange rate (OSHA). Note that the 5-dB exchange rate is more "conservative" where there is a prediction of a lower risk at any given level and exposure time. When noise exposures are measured simultaneously using a 5-dB and 3-dB exchange rate, and all instrument settings are otherwise identical, the measurement made using the 3-dB exchange rate will always have an equal or higher average level than the measurement with the 5-dB exchange rate.

Equal Energy Hypothesis

The "equal energy hypothesis" is a concept that equal amounts of energy should produce equal amounts of hearing loss, regardless of the duration and variability of the exposure. The relationship of the PTS because of steady-state noise exposure with that of fluctuating noise exposure is complicated, but most researchers, basing their conclusions on the work of Martin (1976) and Robinson (1971), have argued that fluctuating noise can be equally hazardous as a steady-state noise

TABLE 32.2

Allowable Times Under NIOSH Recommendations and OSHA Regulations at Each Noise Level (in dBA)

	T (hours)	
Noise Level (dBA)	NIOSH	OSHA
85	8	16
86	6.4	13.9
87	5.0	12.1
88	4	10.6
89	3.17	9.2
90	2.5	8
91	2	6.9
92	1.6	6.01
93	1.0	5.3
94	0.9	4.6
95	0.8	4

of equal energy. Proponents of the equal energy hypothesis would advocate for a 3-dB exchange rate; an increase of 3 dB equals a doubling of sound energy, and a decrease of 3 dB equals halving the sound energy. However, some researchers working in the realm of TTS studies (e.g., Ward et al., 1976) have argued that noises that produce equal amounts of TTS are equally damaging. Proponents of this point of view would advocate for a 5-dB exchange rate, which assumes regularly spaced recovery periods throughout the workday.

Embleton (1994), in reporting on the results of an International Institute of Noise Control Engineering Working Party paper, concluded that "the scientific evidence is that 3 dB is probably the most reasonable exchange rate for daily noise exposure. Statistically it is also a good approximation for the results of many epidemiological studies relating to intermittent exposures even though these show considerable spread about any mean curve" (p 18). It should be emphasized that these exchange rates are meant only to summarize data and they are necessarily an oversimplification of a very complex relationship.

NOISE STANDARDS AND THEIR HISTORY

The earliest regulations designed to protect workers' hearing from NIHL were adopted by the US armed forces as a result of the tremendous amount of NIHL suffered by US service members in World War II (Gasaway, 1985). The first recommended exposure limit (REL) was issued by the US Air Force (USAF) in 1948, followed by the first enforceable hearing conservation regulation (also by the USAF) in 1956 (Suter, 1988). The 1956 USAF regulation identified five aspects of hearing conservation which still form the basis of modern standards:

- Noise reduction efforts
- Measurement of noise exposure
- Education of workers
- Use of hearing protection
- Audiometric surveillance

These requirements evolved from research and recommendations made by CHABA (Suter, 1988). After initial development by the armed forces, several groups, most notably the American Conference of Governmental Industrial Hygienists (ACGIH), established RELs for the civilian workforce. In 1969, ACGIH issued a voluntary threshold limit value (TLV) for noise that represented a greatly simplified version of the CHABA recommendations (Suter, 1988). In 1969 the TLV was adopted by Occupational Safety and Health Administration (OSHA) under the Walsh-Healey Public Contracts Act, which applied to large federal contracts, and separately under the Federal Coal Mine Health and Safety Act (Suter, 1988). Then, in 1971, following the establishment of the OSHA, the Walsh-Healey exposure requirements were promulgated as a permissible exposure limit (PEL) for noise in general industry and construction (Suter, 1988). This PEL remains in force today and specifies a TWA exposure limit (referred to as a criterion level, or L_C) of 90 dBA over an 8-hour workshift, with a 5-dB exchange rate (OSHA, 1981). The PEL requires that employers attempt to reduce noise exposures above 90-dBA TWA through noise controls, though subsequent OSHA policy interpretation effectively raised this level to 100 dBA. Workers exposed above the 90-dBA TWA limit must use hearing protection devices (HPDs), and hearing protectors are further required for exposures that exceed 115 dBA for 1 second or more. OSHA also recommends hearing protectors for exposures above 140 dB SPL regardless of duration. To provide further protection to noise-exposed workers, OSHA promulgated the Hearing Conservation Amendment in 1983, which requires employers to provide baseline and annual hearing conservation training to workers exposed above an action level of 85-dBA TWA, requires baseline and annual audiometric surveillance, and requires that workers exposed between 85 and 90 dBA be offered hearing protectors. OSHA's Hearing Conservation Amendment does not apply to workers in a number of industries, including agriculture, construction, oil and gas extraction, and offshore marine work. Miners are covered by an essentially equivalent PEL administered by the Mine Safety and Health Administration (MSHA), railroad workers fall under a similar regulation administered by the Federal Railroad Administration, and offshore workers fall under the jurisdiction of the US Coast Guard, which administers, though rarely enforces, a similar regulation.

The NIOSH, established in 1971, is tasked with conducting occupational health and safety research and recommending best practice exposure limits (as compared to OSHA, which uses public rulemaking to set mandatory exposure limits, and therefore must include factors such as economic feasibility in their rulemaking efforts). In 1972, NIOSH established a REL of 85-dBA TWA L_C with a 5-dB exchange rate (Suter, 1988). However, in 1998, NIOSH revised its REL to incorporate a 3-dB exchange rate, while retaining the 85-dBA exposure limit. This is consistent with the TLV for noise, which was updated to these same specifications in 1994. Both of these voluntary limits recommend that audiometry, noise controls, and use of hearing protection begin at TWA exposures of 85 dBA and may therefore be considered more protective than the OSHA regulation. The US Department of Defense, as well as the USAF, US Army, and US Navy, have all moved to exposure limits that are consistent with the current NIOSH REL and TLV. Individual states in the United States can opt to have state OSHA programs that administer regulations at least as protective as those promulgated by federal OSHA. None of the state OSHA programs have PELs or hearing conservation requirements that differ considerably from the federal OSHA programs, though several states, including Washington and Oregon, extend coverage to industries such as construction and agriculture.

Noise regulations around the globe are much simpler to describe. Virtually every high-income country in the world, and many medium- and low-income countries as well, has adopted exposure regulations that specify an 85-dBA TWA L_C and 3-dB exchange rate. For example, these limits are required in countries within the European Union. Outside of the United States, only a handful of countries—including Brazil and Israel—use regulations consistent with the OSHA PEL, or a mix of the OSHA PEL and NIOSH REL (e.g., an 85-dBA TWA exposure limit combined with a 5-dB exchange rate). A summary of US and worldwide noise standards and regulations can be found at http://sitemaker.umich.edu/neitzel/files/hearing_loss_references.pdf.

In addition to these regulatory and voluntary occupational exposure limits, limits have been recommended for the protection of public health. Specifically, both the US EPA (EPA, 1974) and World Health Organization (WHO, 1999) have recommended a 24-hour exposure limit of 70 dBA with a 3-dB exchange rate. This is equivalent to an 8-hour exposure at 75 dBA, with no noise exposure for the other 16 hours per day; note that this represents a strong and highly debatable assumption in modern societies. This 24-hour exposure limit is intended to protect against any hearing loss at 4,000 Hz among any exposed individual and can be considered truly "safe"—whereas many occupational exposure limits accept some level of excess risk of hearing loss (e.g., as many as one-third of workers with sound exposures at the OSHA PEL of 90-dBA TWA daily over a 40-year period are expected to sustain a material hearing impairment).

All noise regulations and standards specify—either implicitly or explicitly—methods to determine individual workers' noise exposures. Such determinations can be simple, as is the case when comparing a measured TWA exposure level for a worker to the relevant exposure limit. However, when noise measurements are made with a sound level meter and involve exposures to different noise levels for varying periods of time, it becomes necessary to convert these noise levels and durations into an accumulated personal noise dose. This is done by comparing the ratio of exposure time (C) at each given level to the allowable time (T) at that level, as shown in the equation below:

$$\text{Dose\%} = 100(C_1/T_1 + C_2/T_2 + \cdots + C_n/T_n)$$

Allowable times can be determined by referencing the relevant exposure standard: For compliance purposes, these times are located in Appendix A in the OSHA Noise Regulation (29 CFR 1910.95), whereas the NIOSH best practices recommendation can be found in Chapter 1 of the 1998 Criteria Document for Noise Exposure (DHHS/NIOSH report number 98-126, NIOSH, 1998). Example T values from each standard are based on the data in Table 32.2 presented earlier.

If a worker had an exposure of 4 hours at 95 dBA, 2 hours at 90 dBA, and 2 hours at 85 dBA over the course of a workshift, the OSHA dose would be computed as follows:

$$\begin{aligned}\text{Dose\%} &= 100(4/4 + 2/8 + 2/16)\\ &= 100(1.0 + 0.25 + 0.125)\\ &= 100(1.375)\\ &= 137.5\%\end{aligned}$$

For comparison purposes, the NIOSH dose for the same exposure would be 605%.

Allowable times that are not specifically listed in the relevant standard can be computed directly using the equation below:

$$T = 480 \text{ minutes}/2^{(L_p - L_c/\text{ER})}$$

where L_C is the criterion level, L_p is the measured sound pressure level in dBA, and ER is the exchange rate (in decibels).

The dose value resulting from a dosimeter measurement or computed using the equation above can be computed into a TWA value using the equation below:

$$\text{TWA} = (\text{ER}/\log 2) \times \log_{10}(D/100) + L_C$$

where ER is the exchange rate, D is the dose, and L_C is the criterion level. In the case of the worker described earlier, the OSHA TWA (using a 5-dB exchange rate and 90-dBA L_C) would be 92.3 dBA, whereas the NIOSH TWA (using a 3-dB exchange rate and 85-dBA L_C) would be 92.8 dBA.

Reduction of Occupational Noise Exposures

Exposures can be reduced at three points: (1) The source where the noise is generated; (2) along the pathway the noise travels; and (3) at the receiver (a worker, in this case) (Figure 32.3).

In occupational health practice, and in public health generally, there is a traditional approach to reducing the risk of health effects from any environmental hazard, including noise. This approach is referred to as the "hierarchy of controls" and is displayed in Figure 32.4.

Adherence to the hierarchy of controls, which stresses source control as primary, followed by pathway treatments, followed by a focus on worker behavior, is a requirement of most occupational noise regulations. When workers are overexposed to noise, the hierarchy of controls dictates that the first approach should entail elimination of the noise source

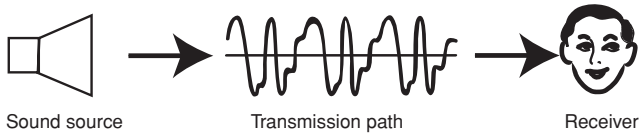


FIGURE 32.3 Generic noise exposure pathway illustrating the three points at which noise levels may be modified. [Used with permission. Courtesy of the Musicians' Clinics of Canada.]

Elimination	Most reliable and desirable
Substitution	↑
Engineering controls	
Administrative controls	↓
Personal protective equipment	Least reliable and desirable

FIGURE 32.4 Industrial hygiene hierarchy of exposure controls.

where possible—thereby eliminating the overexposure. Where elimination is not possible, substitution should be attempted—in other words, replacement of the high noise source with another lower noise source. Noise from sources that cannot be replaced should be minimized through the use of engineering controls, that is, changes to the source (e.g., addition of mufflers) or to the pathway between the sources and the exposed worker (e.g., addition of source or worker enclosures or addition of sound-absorbing coatings or materials to walls and ceiling). Where engineering controls are not sufficient to control exposures, administrative controls can be used to alter the timing or duration of the exposure. Examples of administrative controls include conducting noisy operations when and where few workers are nearby and establishing limits on exposure durations. Finally, when all of the above steps have been attempted, and excessive exposures persist, use of personal protective equipment is needed. Note that this is the last and least desirable approach to exposure control, because it relies on workers having access to protective equipment (HPDs, in the case of noise) as well as knowing when and where to use the equipment and how to use it properly. In the United States, many noisy workplaces do not follow the hierarchy of controls and instead depend on HPDs to reduce workers' exposures. This reliance on the least effective approach to exposure reduction is likely at least part of the reason that tens of thousands of US workers suffer hearing loss each year and why NIHL continues to be one of the leading occupational diseases in the United States (http://www.bls.gov/news.release/pdf/osh.pdf, accessed August 14, 2013).

NOISE AND ITS EFFECTS ON THE EAR: TTS AND PTS

TTS

TTS is understood to result from a variety of reversible physical changes in the cochlea, including broken tip links between OHC stereocilia, loss of contact between the stereocilia and the tectorial membrane, swelling of auditory nerve fibers because of overrelease of neurotransmitter from the IHC, and reduction in cochlear blood flow (Henderson et al., 2006). With adequate recovery time with sound exposure no higher than 76 to 78 dBA (Melnick, 1991), many of these physical changes reverse and hearing sensitivity returns to pre-exposure baseline. TTS is understood to behave in a fairly uniform manner, in that it grows to an asymptotic degree over the course of 8 to 10 hours, and the degree of TTS is a function of the level of the sound and the frequency spectrum. As well, the recovery from TTS follows a predictable pattern (an exponential decay in threshold shift over time), so long as the individual is in an environment less than 76 to 78 dBA ("effective quiet") and the degree of TTS is not greater than 30 dB (Melnick, 1991).

New perspectives on TTS have surfaced suggesting that this condition is not entirely benign. Kujawa and Liberman (2006) compared hearing thresholds in quiet-reared mice (control group) and mice that were exposed to noise sufficient to cause a large TTS, but were confirmed to have no PTS. The inbred mouse strain used in this experiment was chosen because they demonstrate considerably less variability in susceptibility to NIHL as compared to outbred mice and because they tend to maintain good hearing sensitivity into more advanced age. Those mice with early noise exposure (sufficient to cause 40 dB of TTS 2 weeks after the noise exposure, relative to the control group) showed markedly poorer hearing thresholds near the end of their lifespan compared to the quiet control group. The study concluded that the site of lesion of this accelerated age-related hearing loss was neural, rather than cochlear. The implications are that although hearing thresholds may recover to normal levels following a TTS, permanent loss of auditory nerve fibers may have occurred and the consequences will be seen later in life. It is important to note, however, the degree of TTS induced in these mice was just below the exposure necessary to cause a PTS (from acoustic trauma). It is not yet known how these results translate to humans, particularly when the TTS is of a much less significant degree.

PTS

The interested reader is referred to "The role of oxidative stress in noise-induced hearing loss" by Henderson et al. (2006) for a detailed review of the processes that direct cochlear hair cell death and resultant PTS.

As noted previously, PTS is the remaining hearing loss following incomplete recovery from TTS. When this occurs following long-term exposures to moderately intense sound, the primary site of lesion is the OHCs. It is thought that the OHCs are vulnerable because of their high metabolic activity associated with electromotility. Overexposure to noise results in a metabolic challenge to these cells, and the mitochondria of the OHC use large amounts of oxygen to keep up with the energy demands, and consequently a large amount of reactive oxygen species (ROS) by-product are produced. Unwanted ROS by-products, such as superoxide (O_2^-), are free radicals (molecules with an unpaired electron), which are highly reactive and scavenge for electrons from other molecules in the neighborhood. These free radicals

are capable of breaking down lipid and protein molecules in the cells and damaging the cell's DNA (Henderson et al., 2006).

When there is a massive acoustic or chemical insult, cells may die through a passive process of cell death termed necrosis. In necrotic hair cell death, cells swell and rupture, and the contents of the ruptured cell cause an inflammatory response in neighboring cells. The consequence tends to be widespread cell death that continues hours or even days after the resolution of the insult. This tends to be the process of cell death associated with acoustic trauma.

When an overexposure occurs that results in ROS production sufficient to do irreparable damage that will result in the cell's death, an organized process of the cell disassembling may be triggered. This organized process, known as apoptosis, is part of normal development and is important in vital aspects of life, such as maturation (e.g., pruning of redundant neuronal connections) and in the body's defense against cancer cells. Overabundance of ROS results in oxidative stress which triggers a cascade of intracellular events, including release of cytochrome *c* from mitochondria and activation of enzymes known as caspases, which cause fragmentation of the DNA in the cell's nucleus (Matsui and Cotanche, 2004). Throughout this process, the cell wall remains intact, and as the cell breaks apart, it is either ejected from the basilar membrane or engulfed by neighboring cells. The contents of the cell are not spilled, minimizing inflammatory responses of other cells, and damage is minimized.

When the hair cells die, they are replaced with supporting cells, which maintain the integrity of the basilar membrane, but do not contribute to the active process of electromotility. Humans are born with a full complement of roughly 12,000 OHCs and 4,000 IHCs in each cochlea, but once these cells die, they do not regenerate. A considerable number of OHCs tuned to a specific frequency may die without a change in the threshold of puretone detection (i.e., hearing threshold on the audiogram). Once enough OHCs in a specific frequency region have died, hearing sensitivity in that region decreases, permanently. With continued exposure, continued damage to OHCs progresses. It is believed that once exposure ceases (for instance, terminating employment at a noisy job), the degree of NIHL stabilizes. However, the loss of hair cells is cumulative throughout lifetime, and consequently, so is the degree of hearing loss.

NIHL is typically bilateral and symmetrical, because in most circumstances, the soundfield of exposure is typically diffuse. Only in rare situations, for instance, shooting a firearm braced against the shoulder or using a tool or instrument very close to only one ear, might a sound exposure be considerably higher on one side of the head compared to the other. In these rare situations, an asymmetric NIHL could occur. Unexplained asymmetries in presumed NIHL should trigger referral to appropriate medical professionals for evaluation of possible sinister causes, such as autoimmune disorder or eighth nerve tumor.

NIHL from Single Events: Acoustic Trauma

Very high level acoustic events, such as explosions, can result in immediate, permanent damage to the cochlea and to the middle ear. The acoustic parameters dictating the degree of hearing loss do not follow standard damage-risk or exposure-dose. There is not an established "dose" of high-level transient noise equating hearing loss risk with level over time. Single marked overdoses (such as 105 dBA for 6 hours, which is roughly 7,600% noise dose by the NIOSH REL) can result in immediate PTS (Henderson et al., 1993) and so do not follow the expected cycle of TTS growth to TTS recovery; there is incomplete recovery, leaving a PTS. Nonetheless, certain acoustic properties of very high level sound are understood to influence the type and degree of auditory damage.

When an acoustic trauma triggers necrotic cell death in the cochlea, the resulting audiogram may show a 4,000-Hz notch or may have a flat configuration or steeply sloping high-frequency hearing loss. Both OHC and IHC may be affected, as well as damage to supporting cells and the tectorial membrane as a result of the pressure wave traveling through the cochlea exceeding the tissue's elastic limit. The resulting mechanical damage could leave holes between the cochlear partition, allowing perilymph and endolymph to mix, causing widespread destruction. Tinnitus is a very common concomitant injury in acoustic trauma and is often more distressing than tinnitus that builds gradually with long-term progression of NIHL. This is particularly true if there is a psychologic component associated with the event, such as post-traumatic stress disorder (Fausti et al., 2009).

Although OSHA mandates that no single unprotected sound exposure should exceed 140 dB SPL (OSHA, 1983), there is evidence that suggests a much lower "critical level" of 132 dB SPL for threshold of acoustic trauma in highly susceptible individuals (Price and Kalb, 1991). Below this threshold of acoustic trauma, it is believed that NIHL risk is predicted by the established damage-risk criteria (DRC). Between the critical level for acoustic trauma and 170 to 180 dB peak SPL, the cochlea bears the greatest brunt of damage. For higher sound levels, the eardrum may rupture and/or the middle-ear ossicles are disarticulated. When damage occurs in the middle ear, significant energy is dissipated before it reaches the cochlea and considerably less cochlear damage may occur. Thus, peak sound pressure levels between 132 and 140 dB peak SPL and below 170 to 180 dB peak SPL result in greater cochlear damage, and levels above 170 to 180 dB peak SPL result in less cochlear damage, but do result in middle-ear damage (Fausti et al., 2009; Liang, 1992).

Otoprotectants and Reactive Oxygen Species

Antioxidants are molecules present in the body that have an extra electron which can be donated to the free radical. Donating this electron converts the free radical into a less dangerous molecule, thus neutralizing the potential for damage. Naturally occurring antioxidants provide a balance with ROS within the body's systems, unless a pathologic process causes a marked increase in the production of ROS (resulting in apoptosis, or worse, necrosis). Antioxidants can be increased endogenously, such as through exposure to moderate sound conditioning; exposure to sound that causes modest TTS can reduce the degree of future PTS from acoustic trauma (Henderson et al., 2006). The fact that antioxidants can be increased endogenously suggests that they can also be caused to be increased exogenously, such as with drug therapies.

There is now an aggressive search for otoprotectant compounds which can increase antioxidant production and prevent or disrupt the cascade of cell death from noise exposure. Several compounds have been identified as being effective in reducing or eliminating PTS in animal models, such as *n*-L-acetylcystine (LNAC) in concert with salicylate, D-methionine (DMET) (Henderson et al., 2006), and a combination of dietary supplements (beta-carotene, vitamin C, vitamin E, and magnesium; "ACEMg") (Le Prell et al., 2007). Clinical trials are currently underway to determine if any of these compounds are safe and efficacious in humans. In the not too-distant future, there may be pharmaceutical interventions that either reduce the degree of PTS following a noise event or eliminate PTS altogether.

NIHL FROM LONG-TERM EXPOSURES: NIPTS PREDICTION AND POPULATION FRACTILES

As noted previously in this chapter, there is wide variability in the degree of hearing loss sustained across a noise-exposed population; the observed differences are due to differences in individual susceptibility to NIHL, which are due to both exogenous and endogenous reasons (Henderson et al., 1993). Population fractiles (i.e., fractions of a population) are used to describe the variability in susceptibility across individuals in an exposed population. The median NIPTS risk is defined as fractile of 0.5 (i.e., one-half of individuals noise-exposed have greater hearing loss, and one-half have less hearing loss). The 0.1 fractile describes the population of highly susceptible individuals (only 10% of the exposed population has greater hearing loss; 90% has less hearing loss). The 0.9 fractile describes very low susceptibility (90% of the exposed population has greater hearing loss; 10% has less hearing loss). The American National Standards Institute (ANSI) and the ISO have mathematical models that allow one to calculate the predicted degree of NIPTS at each audiometric frequency for given exposure levels (dBA TWA) and given exposure duration (in years) for various fractiles of the population (ANSI S3.44-1996; ISO 1999:1990). These models were developed from the seminal studies of noise-exposed workers and observed degree of NIPTS (e.g., Lempert and Henderson, 1973).

From these mathematical models of predicted NIPTS, the percent of individuals whose hearing loss exceeds a level deemed a "hearing handicap" or "material hearing impairment" can be calculated. For this purpose, material hearing impairment is defined as the maximum acceptable hearing loss. This percent of an exposed population with a "material hearing impairment" or greater is the excess risk of NIHL from a given exposure. For instance, using a material hearing impairment defined as average hearing thresholds at 500, 1,000, and 2,000 Hz of >25 dB HL in both ears (i.e., the three-frequency puretone average hearing loss), NIOSH (1974) estimated that 40 years of 8-hour TWA to 80, 85, and 90 dBA would result in 3%, 15%, and 29% excess risk, respectively. In other words, 29% of workers exposed to 90 dBA TWA for 40 years would have three-frequency puretone hearing loss (500, 1,000, 2,000 Hz) greater than 25 dB HL. A reanalysis of risk estimates from the Lempert and Henderson (1973) data, defining material hearing impairment as a four-frequency average hearing loss >25 dB HL (frequencies 1,000, 2,000, 3,000, and 4,000 Hz weighted according to the articulation index), showed excess risk of 1.2%, 7.6%, and 22.3% for 40 year, 8-hour TWAs of 80, 85, and 90 dBA, respectively. In summary, NIOSH REL and ACGIH TLV of 85-dBA TWA, not to mention OSHA and MSHA PEL of 90-dBA TWA, are not *safe.* Some people with high individual susceptibility exposed to these "protected" levels will develop NIHL. Still, these recommendations (NIOSH, ACGIH) and regulations (OSHA, MSHA) are *safer* than no limits at all.

NONAUDITORY EFFECTS OF NOISE EXPOSURE

Nonauditory effects of noise exposure are those effects that do not cause hearing loss. Some of these are seen by changes in body functions, such as heart rate, and in learning/cognition in children, and sleep disturbances (Goines and Hagler, 2007). Nonauditory effects of noise exposure have been noted as far back as 1930 (Smith and Laird, 1930). In that specific study, nonauditory effects pertain to stomach contractions in healthy human beings when exposed to noise.

In the past 25 years, an increasing body of evidence has connected occupational and community noise exposures to a wide range of adverse health effects. These range from cardiovascular disease and sleep disturbance—effects which likely have a substantially larger public health impact than does NIHL—to stress, annoyance, and cognitive effects,

including learning disorders among children (van Kamp and Davies, 2013). A more complete overview of this area with the relevant historical references can be found in the appendix at the end of the book.

MUSIC AS NOISE

People recognize that the military and industry are high noise sources. In the military, there is equipment such as tanks, jet aircraft, and other heavy machinery, and personnel are exposed to rifle fire and explosions (in training and combat). Thus, noise exposure is an ongoing risk. In industrial settings, heavy equipment, machinery, printing presses, and so on, also create an environment in which individuals may be exposed to hazardous noise levels. However, it is more difficult for the average person to recognize that everyday noise may be a contributing factor in the development of NIHL.

Musical instruments also have the capability to generate high levels of sound both at the location of the musicians' ears and at the ears of their other musician colleagues who have the bad fortune to sit downwind. Unlike "noise" (the unwanted by-product of another activity), music is the purpose of the activity which is generating sound that may be potentially harmful. Table 32.3 is adapted from Chasin (2006) and shows a selection of sound levels measured on the horizontal plane and at a distance of 3 m. Also shown is the sound level (dBA) of the violin measured near the left ear. These data are based on over 1,000 measurements with the top and bottom 25th percentiles removed.

For nonmusicians (and musicians), a significant source of noise exposure outside of occupational and military environments is from "portable" music exposure. "Portable music" such as portable digital music players (MP3 players), as the name suggests, can be used in noisy environments such as city streets where ambient noise masks the music if earphones provide no sound isolation for the listener. The MP3 player user turns the volume control up to achieve a chosen (or preferred) listening level. In these situations, the most comfortable listening range is at a higher sound level than in quieter or in more controlled environments (Fligor and Ives, 2006; Portnuff et al., 2011).

Since the introduction of the first Walkman-style cassette player, numerous studies have reported potential for NIHL from using portable music inappropriately (Fligor and Cox, 2004; LePage and Murray, 1998; Portnuff et al., 2011; Rice et al., 1987; Turunen-Rise et al., 1991). Fligor and Cox (2004) reported that all evaluated commercially available portable CD players produced sound levels that could easily exceed DRC (with equivalent-continuous levels up to 121 dBA) if the user chose levels near maximum. As well, these levels depended on the earphone that was used. For example, with certain earphones, the outputs were 7 to 9 dB higher than the same CD player with the manufacturer-supplied earphone. Peak SPLs in the music (percussion transients) were found in excess of 130 dB SPL at the highest volume control setting. In general, Fligor and Cox (2004) found that greater outputs were produced when using physically smaller earphones. Fligor and Cox (2004, p 513) concluded that "based… on the noise dose model recommended by the National Institute for Occupational Safety and Health (NIOSH) for protecting the occupational worker, a maximum permissible noise dose would typically be reached within 1 hour of listening with the volume control set to 70% of maximum gain using supra-aural headphones." In the interest of providing a straightforward recommendation, they state, "reasonable guidelines would [be] to limit headphone use to 1 hour or less per day if using supra-aural style headphones at a gain control setting of 60% maximum" (p 513).

Similar results have been obtained with other music media such as MP3 players—the potential for music-induced hearing loss based on models of noise exposure is quite real, and the output is related not only to the setting of the volume control and duration of listening, but also to the nature of the earphone/earbud that is used. Portnuff et al. (2011) measured output levels of MP3 players and recommended a reasonable guideline for mitigating hearing loss risk from music listening on these devices was to limit use to 90 minutes per day with the volume control set to 80% of maximum or lower, using the earbud headphone that came with the device. Contrary to what one might expect, the output levels of MP3 players (Portnuff et al., 2011) were consistently lower than the output levels of CD players (Fligor and Cox, 2004). However, extended battery life and nearly infinite length of music playlists might give an MP3 player user the capacity to listen much longer than was convenient with a CD player. Level-over-time guidelines (such as "80 for 90") might provide users with an "acoustic speed limit" for using headphones with less risk than not having guidelines for safer listening.

TABLE 32.3

Average Sound Levels of a Number of Musical Instruments Measured from 3 Meters

Musical Instrument	dBA Ranges Measured from 3 m
Cello	80–104
Clarinet	68–82
Flute	92–105
Trombone	90–106
Violin	80–90
Violin (near left ear)	85–105
Trumpet	88–108

Also given is the sound level for the violin measured near the left ear of the players. Adapted from Chasin M. (2006) Music and hearing aids. *Hear Rev*. March, 34–41. Courtesy of the *Hearing Review*.

Fligor and Ives (2006) showed in adults, and Portnuff et al. (2011) showed in teenagers, that a small but significant percent of MP3 player users chose listening levels in quiet that exceeded 85 dBA, and that the chosen listening levels were the same for the individual subject regardless of the type of earphone (e.g., earbuds vs. over-the-ear vs. in-the-canal). As ambient noise increased, so did the percent of people who chose levels in excess of 85 dBA (as many as 80% of people chose levels in excess of 85 dBA in a simulated commercial airplane cabin when using both earbuds and over-the-ear headphones). This ambient noise masking problem was consistently managed when subjects used earphones engineered to attenuate ambient sound. They concluded that it is not the absolute level that earphones could produce at maximum volume control, but the ambient level that dictates riskier listening behavior, and sound-isolating earphones reduced the need for choosing levels that put hearing at risk.

Acoustic Trauma and the Musician

In addition to the overall long-term music exposure for musicians, exposure may include feedback squeals during sound checks, inappropriately set limiters, percussive blasts from cannons, and being stuck in front of a large stack of speakers for an extended performance. Whereas there is scant research in the literature on the effects of single-trauma impulses in music venues, hearing loss has been confirmed clinically where the source was a single or relatively short-duration blast. Reports of hearing loss because of long-term industrial noise exposure are numerous and, in some ways, better controlled because musical environments are often much more poorly defined. Most musicians serve as independent contractors and could not easily be tracked with baseline and annual audiometry in a hearing conservation program. In addition, unlike a worker in an industrial setting, musicians (and avid music consumers) may be subject to damaging levels of music exposure in their off-work hours.

HEARING PROTECTION DEVICES

History of HPD and How the NRR Evolved

The first commercially available HPD was the V-51R premolded hearing protector, introduced in 1945 (Humes et al., 2005). The HPD market was slow to develop, however, and following the introduction of this device, only a few types of HPDs were available through the 1960s (Gasaway, 1985). In the 1970s, a wider variety of earplugs and earmuffs became available, including the roll-down slow-recovery foam earplug and other devices made of newly introduced materials. HPDs continued to improve in the 1980s and 1990s with the introduction of minor technologic and cosmetic changes that increased comfort but did not result in appreciably improved performance. Since 2000, HPD technology has advanced substantially, with the introduction of passive as well as electronic "level-dependent" HPDs that provide variable levels of attenuation depending on the external exposure level (Humes et al., 2005). The market has continued to grow, as well, with more than 300 models of HPD available in the United States in 2003 (NIOSH, 2003).

The attenuation that earplugs provide users can be measured in a variety of ways. In the United States, all non-custom HPDs are required by law to be labeled with a noise reduction rating (NRR), a single-number rating (SNR, in decibels) of the amount of protection a trained user can expect to receive while wearing a specific HPD. The NRR was established by the US EPA in 1979 through a rule titled "Noise Labelling Standards for Hearing Protection Devices" (http://www.gpo.gov/fdsys/pkg/FR-2009-08-05/pdf/E9-18003.pdf, accessed August 24, 2013). Prior to this regulation, there had been no requirement for standardized testing procedures and labeling approaches for HPDs, and adoption of the rule created a powerful new tool by which users could compare attenuation across different HPDs. The NRR is a simplified interpretation of the expected performance of a given HPD across seven different frequencies between 125 and 8,000 Hz when the protector is fit on a trained user by an experimenter under laboratory test conditions. The NRR is computed from test data taken across multiple test subjects, and then subtracts twice the standard deviation around the mean attenuation at each frequency to account for individual user variability. This subtraction exercise is intended to result in an NRR that can be achieved by 98% of users of the HPD in question.

While the NRR labeling requirement benefits consumers, the NRR itself has been heavily criticized. A large body of research on attenuation achieved by actual users in work settings—as opposed to trained test subjects in a laboratory setting—is substantially lower than the NRR suggests (Berger, 2000) and often bears little relation to the labeled value. These differences stem from a variety of causes, most notably poor hearing protector fit among users in field studies. Differences between the NRR and field measurements of attenuation are generally smaller for earmuffs than for earplugs, as earmuffs are generally much easier to fit correctly. EPA has acknowledged the limitations with the current approach to measuring and labeling NRRs and has for some years been considering an update to the NRR regulation.

Computation of Attenuated Exposure

Whereas attenuation with a given HPD is known to vary widely across individuals, use of the NRR to estimate the attenuated (e.g., underneath the HPD) noise exposure for workers is nevertheless common. The nominal approach for computing attenuated noise exposures for workers whose

A-weighted TWA noise level is known is shown in the equation below:

$$\text{Nominal attenuated exposure (dBA)} = \text{TWA(dBA)} - (\text{NRR} - 7)$$

The 7-dB value in the equation is a spectral correction factor required to account for differences in the way noise is measured during the NRR test (using dBC) versus measurements made in the workplace (using dBA).

The equation above can be applied to TWA values measured according to either the OSHA PEL (using a 90-dBA L_C and 5-dB exchange rate) or the NIOSH REL (using an 85-dBA L_C and 3-dB exchange rate). However, this equation does not take into account variability in achieved attenuation, but rather assumes that all users of a hearing protector will achieve the labeled NRR. As described above, this is an unrealistic expectation. There are two approaches for accounting for expected differences between labeled and achieved attenuation. The first is recommended by OSHA and involves derating the labeled NRR of a HPD by 50%:

$$\text{OSHA attenuated exposure (dBA)} = \text{TWA (dBA)} - [(\text{NRR} - 7) \times 50\%]$$

The second approach is recommended by NIOSH and assumes patterns in achieved attenuation by the type of HPD used:

$$\text{NIOSH attenuated exposure (dBA)} = \text{TWA(dBA)} - (\text{NRR}_d - 7)$$

where NRR_d is the derated NRR for the type of earplug being considered. NIOSH's recommended deratings involve subtracting 25% of the NRR for earmuffs, 50% for foam earplugs, and 70% from all other earplugs. So, as an example, if a worker uses a foam earplug with an NRR of 30 dB, the NIOSH NRR_d would be $30 - (30 \times 70\%) = 9$ dB.

Workers with very high exposures (>100- or 105-dBA TWA) should be fitted with dual protection, that is, a pair of earmuffs over earplugs. The general rule of thumb for estimating attenuation for dual protection is to add 5 dB to the attenuation of the HPD with the higher NRR (NIOSH, 1998).

Whereas the NRR is the required standard for testing and labeling HPDs in the United States, there are other standards in use around the world. Common testing and labeling schemes include the SNR (used in the European Union) and the sound level conversion (SLC_{80}, used in Australia and New Zealand). There are several differences between the NRR, SNR, and SLC_{80}, including the fact that the NRR calculation subtracts 2 SD to account for user variability, whereas the other two schemes subtract only 1 SD, and that the test frequencies are somewhat different. The SNR rates protectors for specific types of noise environments, with different ratings for high-frequency (H), mid-frequency (M), and low-frequency (L) spectra. The SLC_{80} value is used to assign a classification to the tested HPD. For example, class 1 is valid for use up to 90 dBA, class 2 to 95 dBA, and so on (Williams, 2012).

Acoustics of HPD

The application of the laws of acoustics as applied to HPDs is similar to those found in the realm of hearing aid acoustics, classroom acoustics, music acoustics, or the larger area of architectural acoustics. For the purposes of HPD, these laws can be described as (1) wavelength phenomena, (2) Helmholtz/volume-related phenomena, and (3) mass and density characteristics. Wavelength-related characteristics can be seen in the degree of attenuation across the frequency spectrum whereas Helmholtz/volume-related characteristics tend to be relegated to a rather narrow spectral region, such as at a resonance. Like wavelength phenomena, mass and density characteristics can be observed over a wide range of frequencies.

All vibrations in air, whether noise, speech, or music, exhibit compressions and rarefactions of the air molecules. The degree of vibration of the movement of the air molecules is related to the amplitude of the vibration. And HPD serves to reduce the amplitude of the molecular vibrations. Because of the three acoustic features mentioned above, this sound reduction is not necessarily uniform across the frequency spectrum.

Wavelength-Associated Phenomena

With HPDs wavelength phenomena are related to the physical dimensions of the obstruction—longer low-frequency wavelengths do not acoustically "see" the obstruction as well as the shorter high-frequency wavelengths. A HPD, whether it is an earmuff, an earplug, or any other obstruction in the room or in the ear, will therefore provide less attenuation for the lower frequency sounds than for the higher frequency sounds. HPDs are inherently poor at attenuating the longer wavelength, low-frequency sounds and are inherently better at attenuating the shorter wavelength, high-frequency sounds.

This same line of reasoning explains why the attenuation characteristics of audiometric sound booths provide greater attenuation for higher frequency sounds than lower frequency ones. Because of the density and diameter of the sound booth walls, greater attenuations can be provided than head-worn HPDs which are relatively light. Figure 32.5 shows the attenuation characteristics of a well-fit earmuff-style HPD exhibiting the attenuation differences across the frequency range, as well as the attenuation provided by a commercially available audiometric sound booth.

Understandably, the attenuation characteristics of many HPDs that are the result of wavelength phenomena can be problematic. There can be significant low-frequency energy in an industrial noise spectrum, yet this is where HPDs provide the least amount of hearing protection. And with the greater degree of high-frequency sound attenuation many speech cues that contribute significantly to speech intelligibility can be lost. To a certain extent, it is understandable

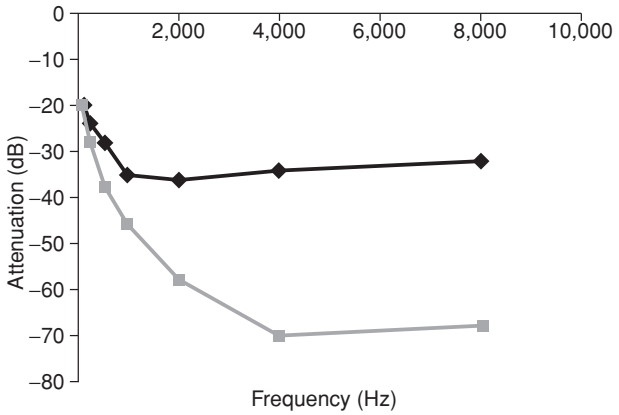


FIGURE 32.5 Attenuations of an earmuff-style HPD (*dark gray*) as well as the attenuation of a commercially available audiometric test booth (*light gray*) for comparison purposes.

that many industrial workers prefer to remove their HPDs when trying to communicate, thereby reducing their effectiveness. Unless specifically modified, HPDs can also have limited acceptability with listening to and playing of music: the lower frequency fundamental notes may be quite audible, but the higher frequency harmonic structure can be lost.

Helmholtz/Volume-Associated Phenomena

Unlike wavelength-associated phenomena which affect a wide range of frequencies, albeit greater in the upper range, Helmholtz/volume-associated phenomena tend to be restricted to a narrow range of frequencies. Resonant chambers can be created within a HPD that either offsets the attenuation (as a resonance) or adds to the attenuation (as a side branch resonator). These additional chambers, depending on their size and location, can significantly alter the attenuation characteristics of HPDs. As will be discussed in a later section, these resonances can be used to minimize the amount of attenuation in the higher frequency region, thereby creating a HPD with a more uniform (or frequency-independent) attenuation characteristic.

Mass and Density Characteristics

As discussed above, the greater the density and the mass of the HPD, the greater the overall attenuation. HPDs made of cotton will have less overall attenuation than those made of polymeric foam or silicon. From an ergonomic perspective, there is a natural upper limit to the mass of the HPD since this may cause discomfort over an extended wearing period.

Earmuffs and Earplugs

There are two major categories of HPDs: Earmuffs that fit over the ear and earplugs that fit into the ear canal. Both are similar in that they both provide obstruction of sound resulting in attenuation of the noise or music to a level that is less damaging. Earmuffs can have greater mass and density and therefore can provide more overall attenuation than earplugs, especially in the mid- to lower frequency range; nonetheless, well-fitted, deeply seated earplugs can still provide near-maximum amounts of attenuation.

Earmuff-style HPDs, because of their larger size, can incorporate a wider range of acoustic and electronic options such as two-way communication system. However, both earmuffs and earplugs can incorporate many acoustic modifications that can significantly alter their attenuation characteristics. Some of these will be discussed under the heading of uniform attenuation HPDs.

One essential difference between the two styles is that the earmuff-type HPDs do not destroy the natural ear canal resonance which occurs at approximately 2,700 Hz. The attenuation of this type of HPD is therefore given by the attenuation of the earmuff, offset by the natural 15- to 20-dB amplification caused by an unoccluded ear canal. In contrast, earplugs that are located in the ear canal do not have this offset. The ear canal resonance does not offset the attenuation because it has been interrupted by the insertion of an earplug, much like an occluding hearing aid can result in an insertion loss. A schematic of the ear canal is given in Figure 32.6 showing the quarter wavelength standing wave that corresponds to the 2,700-Hz resonance. If there is an obstruction in the lateral end (marked B) where the earplug HPD is situated then this natural resonance will be interrupted, thereby resulting in more relative attenuation at 2,700 Hz than with an earmuff HPD. Obstructions located near the medial end (marked A) will result in minimal attenuations.

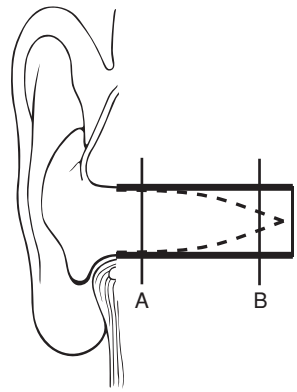


FIGURE 32.6 A schematic of the ear canal showing the quarter wavelength standing wave that corresponds to the 2,700-Hz resonance. If there is an obstruction in the lateral end (marked *A*) where the earplug HPD is situated then this natural resonance will be interrupted, thereby resulting in more relative attenuation at 2,700 Hz than with an earmuff HPD (which is positioned closer to position A). (Used with permission. Courtesy of the Musicians' Clinics of Canada.)

Subsequently, if one were to normalize the attenuation characteristics of an earmuff HPD and an earplug HPD, there would be less relative attenuation in the 2,700-Hz region with earmuffs versus earplugs.

Maximum Attenuations

As the air-conduction pathway is maximally disrupted by HPDs, the bone-conduction pathway sets a ceiling for maximum attenuation. This ceiling imposed by sound traveling via bone conduction is frequency specific; for instance, maximum attenuation at 2,000 Hz is roughly 40 dB (Berger et al., 2003). This has implications for the fitting and assessment of HPD and may influence the method of HPD evaluation.

Poor HPD Fittings

Like anything that is added to the human body, there may be a poor fit. This may be inherent in the design of the HPD (noncustom fit) or related to how it is worn by the worker or musician. Because of the acoustic characteristics of HPDs workers may partially lift, or loosen the HPD off, or from, their ear(s) in an attempt to improve communication with their worker colleagues. This will certainly improve the speech intelligibility since there will be improved audibility of the higher frequency consonant sounds.

The drawback of this removal is the creation of a slit leak or venting route in the HPD. This has the same effect as venting does for hearing aids: There will be less low-frequency attenuation for the HPD. Since many forms of occupational noise have significant energy in the lower frequency region, this partial removal may be quite detrimental to the long-term hearing of the worker. Low-frequency sound will enter the ear directly through the vent, bypassing the HPD. In addition, the existence of a vent (intentional or otherwise) may have deleterious effects on the other "tuned" acoustics in the HPD. One solution to the problem of a worker intentionally removing the HPD for improved communication would be the use of an electronic two-way communication system.

In a musical venue where there may only be intense high-frequency sound energy (such as being downwind of a cymbal), the intentional use of a vent in a HPD can be quite useful. These modified (and usually custom-made) vented/tuned earplug HPDs can be designed to provide minimal attenuation below 1,000 Hz (Chasin, 2006).

A common form of "unintentional" venting of earmuff HPDs is seen as the result of eye protection. Many protective goggles and other eye-wear can cause unintentional slit leaks where the arm of the eye-wear meets the cuff of the earmuff HPD. Protective eye-wear can be designed with very small, almost thread-like arms that will not significantly compromise the attenuation characteristics of the HPDs.

Uniform Frequency ("Flat") Attenuators

As noted previously, solid earplugs inserted into the ear canal disrupt the normal ear canal resonance. When maximum attenuation is necessary, such as in very high noise situations, this disruption is beneficial, as it results in the greatest attenuation in the frequency range where the ear canal would amplify the most dangerous sounds. However, maximum attenuation is often not a desired outcome of HPDs. When hearing auditory signals is important for safety (e.g., hearing a back-up alarm on a forklift), too much sound isolation increases risk for industrial accidents. When individuals need to communicate verbally, loss of ear canal resonance has a disproportionate impact on reduction in speech intelligibility; some of the most important speech cues are between 2,000 and 3,000 Hz. Mishearing a verbal command can also increase risk of accidents. These are some reasons earplugs are not inserted properly; workers may feel they need better situational awareness, and reducing low-frequency attenuation is in fact safer (not for their hearing, but for the rest of their body).

When the potentially hazardous sound is being produced purposely (such as music), rather than as an unwanted by-product of using machinery (such as drilling), earplugs that reduce normal acoustic cues make HPD unusable. Further, there are acoustic properties of music, such as the relative sound level between the first, second, and third harmonics of a musical note, that contribute significantly to positive attributes of the music, such as pitch and timbre. These properties are partly what make a piano sound like a piano, a clarinet sound like a clarinet, and a guitar sound like a guitar; the same musical note (for instance, A above middle C) played on different instruments is immediately distinguishable as being played by those different instruments. Musicians listen across the frequency range to monitor the quality of their performance, and introducing distortions (such as less attenuation in low frequencies and more attenuation in high frequencies, thus changing the relative level differences between a fundamental and harmonics) interferes with the musician's ability to perform. In the musician's competitive world, where consistent, perfect performance is a prerequisite for steady income, anything that interferes with performance is unacceptable.

One approach to improve acceptance of HPD in musicians are custom-fitted earplugs that are intended to provide a modest amount of uniform attenuation across a wide range of frequencies. The earliest forms of uniform attenuating earplug, intended for use by musicians, were Musicians Earplugs™ (MEPs) (Etymotic Research, Elk Grove Village, IL). Production of MEPs began when a need for improved acceptability of HPD was established with the Chicago Symphony Orchestra (Royster et al., 1991). These earplugs, by design, reduce sound levels by an intended 9,

15, or 25 dB (ER9, ER15, and ER25, respectively) and are composed of two components: The filter (which designates the amount of sound reduction—9, 15, or 25 dB down) and a "sleeve" which is custom fitted from an earmold impression and made of vinyl, acrylic, or silicone. MEP attenuations of 9 and 15 dB are modest, acknowledging that many musicians have significant sound exposures, but the levels and/or weekly duration may not necessitate higher levels of attenuation for the musician to receive less than a 100% noise dose (Fligor and Wartinger, 2011). Since the advent of the Musicians Earplugs™ in 1988, several other similar flat attenuation HPDs have come onto the marketplace.

Electronic HPD/Communication Systems

As HPDs have increased in sophistication, manufacturers have developed ways to incorporate two new technologies into some devices: Electronic amplification (also known as "level-dependent amplification") and communication capability. Electronic amplifying earmuffs have either one (monaural) or two (stereo) microphones on the outside of the earmuff cup(s), which amplify ambient sounds when the ambient noise levels are under a predefined threshold (typically around 82 dBA). Once ambient noise levels exceed this level, the electronic amplification circuitry is deactivated, and the earmuff reverts to being a passive device. When ambient levels drop below the threshold, the amplification circuitry reactivates. The benefit of this feature to the HPD user is that the earmuffs do not have to be removed to have a conversation with, or listen to instructions from, a coworker or supervisor. These earmuffs can also improve situational awareness by making it easier for workers to detect important sounds while still wearing their earmuffs. These devices can be especially useful for hearing-impaired workers. For such workers, conventional HPDs essentially represent an additional hearing handicap (e.g., the workers' hearing loss plus the additional attenuation provided by the HPD), whereas electronic amplifying muffs may actually allow for improved signal detection.

Some earmuffs also have an input jack that allows the user to connect a communications device, for example, a facility radio or mobile phone. These earmuffs give the user the advantage of being able to understand verbal communications clearly, via electronic means, without needing to remove their earmuffs. This can be a major benefit where frequent communications are required in a noisy work environment. Users may also have the added benefit of being able to listen to music through the same input jack. An additional advantage of these devices is that many have built-in volume limiters, meaning the employer can be confident that the exposure level inside the ear cup of the earmuff does not exceed a predefined threshold, providing assurance that no additional risk of NIHL is added through the use of the communications earmuff.

Verification of HPD

The effectiveness of a HPD depends partly on the physical properties of the device and partly on how they are used by the individual. An excellent example of the interplay is the difference between laboratory-measured NRR and the NRR actually achieved in the field of earplugs versus earmuffs. Laboratory NRR of earplugs (with perfect fit ensured by an experimenter) would suggest these HPDs provide better low-frequency attenuation than earmuffs do, but when accounting for real-world performance, earmuffs provide more low-frequency attenuation than earplugs. Whereas it would be expected that custom-fitted plugs (such as MEPs) would have ideal performance both in the lab or in the clinic and in the real world, this is dependent on the plugs being used correctly by the individual. Thus, verification of actual HPD performance is an important component of mitigating risk for NIHL. There are multiple approaches to verification of HPDs, although three methods are currently the most popular: real ear attenuation at threshold (REAT), microphone in real ear (MIRE), and acoustical test fixtures (ATF). Each method has its strengths and weaknesses, and some methods are more appropriate than others for specific types of HPDs (Berger, 2005).

Real Ear Attenuation at Threshold Method

The REAT method is the longest used method and most intuitive. Simply, it requires the user's hearing to be tested across a specified range of frequencies without earplugs in place, and again with the plugs in place. This method can be conducted via soundfield audiometry or under circumaural earphones (as long as these earphones are large enough that they do not distort the shape of the pinna). Often, 1-dB step sizes are used to narrow the standard deviation in the measure. As noted previously, the REAT method is used to derive the NRR. It is considered the gold-standard method for verifying HPD, although it has its drawbacks. For one, it requires a behavioral response (and so an introduction of subjectivity in the measure) which is a source of variability in the measure. NRR is measured only in adults, whereas REAT measures with sufficiently small variability using pediatric test techniques (conditioned play audiometry or visual reinforcement audiometry) have not been established. Additionally, it is relatively slow and requires appropriate test equipment and a professional capable of performing puretone audiometry; it requires more time than is typically allotted for fitting a person with earplugs or earmuffs and may not be possible (because of equipment limitations) in the field. Finally, level-dependent (e.g., active noise reduction and passive nonlinear) HPDs are designed to provide little to no attenuation at low input levels (such as used when measuring REAT) and so would purposely show a REAT value of zero (or near zero) dB attenuation.

Microphone in Real Ear Method

The MIRE technique uses a probe microphone placed near the eardrum and is equivalent to real ear measures (REM) in measuring hearing aid output with hearing aid verification equipment. The difference is that REM is intended to document increase in sound reaching the eardrum when the device is in place and functioning (to bring sound into the residual auditory area of the person with hearing loss) whereas the MIRE technique is intended to document the insertion loss (i.e., attenuation) between the diffuse field and the eardrum when the device is in place and functioning. Benefits over REAT include this measure being objective (no subject response required) and it is considerably faster. A principal challenge, however, is the placement of the microphone in the ear canal or through the HPD in a way that does not influence the performance of the HPD. A leak between the HPD and wall of the ear canal because of the presence of a probe tube introduces a measurement artifact that greatly reduces the amount of low-frequency attenuation. Commercially available MIRE systems have managed this challenge using different techniques, such as inserting microphones through a valve in the HPD. Using hearing aid verification equipment with standard probe tube microphone (outer diameter 0.95 mm), Fligor (in press) applied a water-based lubricant to the surface of the sleeve of MEPs to attempt to limit the influence of slit leak created by any gaps between the ear canal and the sleeve of the HPD. Examples of good-fit ER15 MEP (with relatively uniform "flat" attenuation across measured frequencies) and poorly fit ER15 MEP (with little attenuation below 1,000 Hz) are shown in Figure 32.7 in the right ear and left ear of the same patient. Although it is possible that there was slit-leak artifact in the left HPD measure, this result suggests the patient should

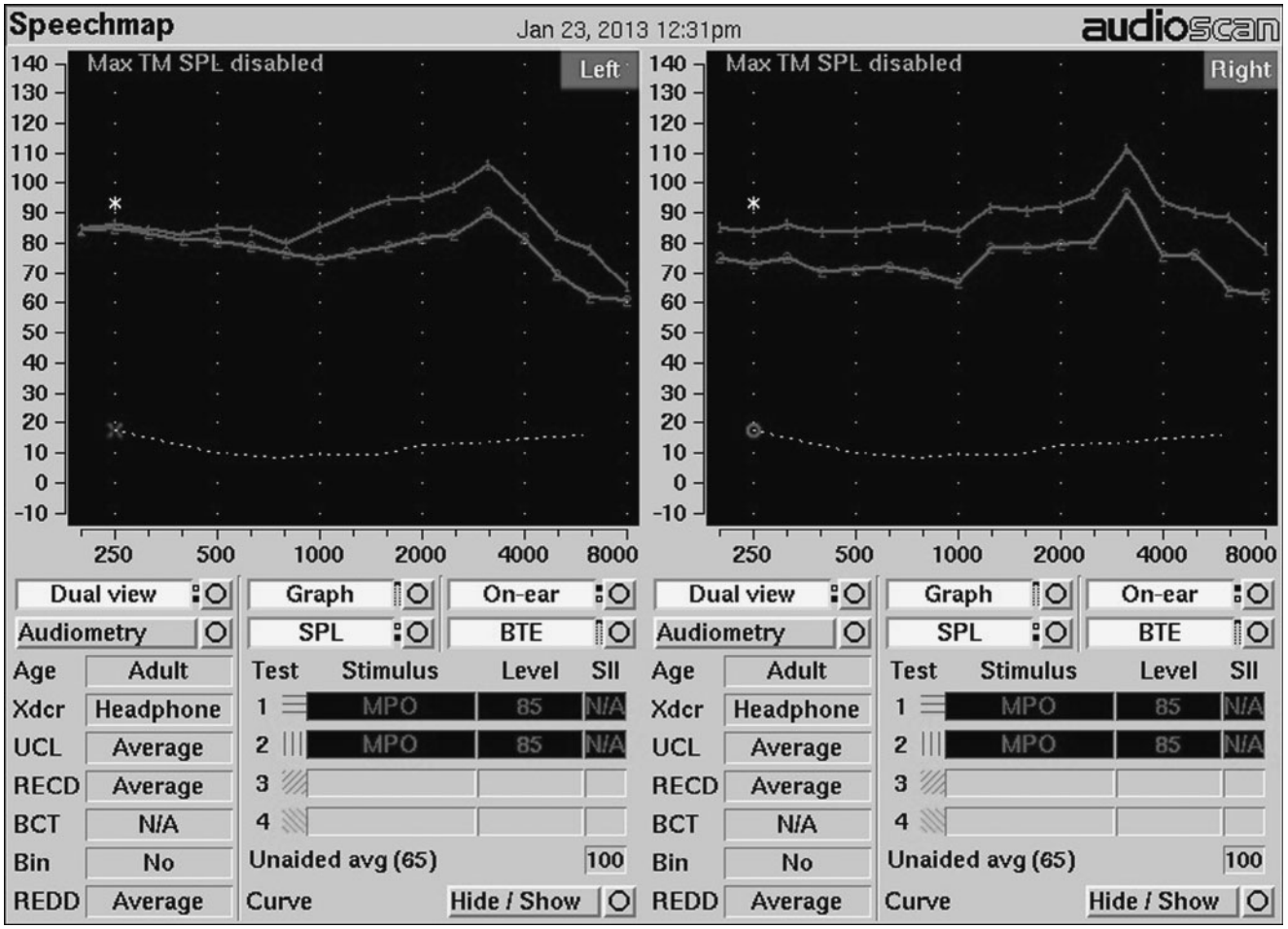


FIGURE 32.7 MIRE measures of right and left ears of patient fitted with ER15 MEPs. The line indicating test 1 (*top line*) shows the sound level as a function of frequency, as measured near the eardrum by a probe tube microphone to an 85-dB SPL swept tone in the unoccluded condition. Test 2 (*lower line*) shows the measure repeated with an 85-dB SPL swept tone, but with the left and right ear MEPs in place. There is less low frequency attenuation in the left ear with the MEP than the right ear. This may be due to a slit leak caused by the probe tube microphone.

have REAT measures of that left ER15 MEP and if this lack of low-frequency attenuation persists without the probe microphone in place, the MEP sleeve should be returned to the earmold laboratory and remade for poor fit.

Acoustical Test Fixture: ATF Method

The ATF method involves the use of a mannequin that acts as a surrogate for the human head and ears. The most readily recognized ATF for audiologists is likely Knowles Electronics Mannequin for Acoustic Research (KEMAR). To be an appropriate surrogate, the ear canals, pinnae, head, and so on should be equivalent to the size of the intended user and have characteristics (such as skin simulation) for testing the HPD of interest. A static model ATF lacks the wide variability in the dimensions of adults, and therefore gives data that have limited inference to the real world. An ATF can be used for applications that are unsuitable for use by human subjects, such as measuring nonlinear response of HPD to gunshots and explosions. More sophisticated ATFs can incorporate characteristics of sound transmission through bone conduction to avoid some of the errors with exceeding maximum possible attenuation.

Unresolved Issues with HPDs

Audiometry, Standard Threshold Shift, and Reportable Shifts

Arguably, a most effective way to mitigate NIHL risk is to engineer the environment to not exceed safe exposure limits (Figure 32.4). In fact, if noise levels are below 85-dBA TWA, the hearing conservation program need go no further than repeating the noise survey if the work environment changes. Once noise levels equal or exceed 85-dBA TWA, the alternative steps of either changing the worker's schedule so that the exposure time is reduced (administering out the noise) or using HPDs must be instituted. However, these options are problematic and, if not carefully monitored, can be less than effective. How well the alternate methods are working must be assessed. The most certain way to monitor the effectiveness of administrative controls and use of HPD is through periodic hearing testing. Additionally, as per the estimates of excess risk on which the OSHA regulations are based, it is anticipated that up to roughly 25% of workers exposed to 85- to 90-dBA TWA will still develop a handicapping hearing loss. Early identification of high susceptibility to PTS is an effective way for instituting steps in avoiding the development of material hearing loss. Therefore, OSHA requires that all workers with noise exposure at or above the action level have routine hearing tests. The hearing testing program includes baseline testing, annual retests (with appropriate analysis to monitor for changes in hearing), worker training, and follow-up procedures (OSHA, 1983).

A licensed professional must administer the audiometric testing program. Current regulations stipulate that the professional must be an audiologist, otolaryngologist, or other physician. The administrator does not need to be the person who actually conducts the testing. OSHA allows for trained technicians to do the testing, with an administrator who oversees the program, supervises the technicians, reviews employee audiograms, and makes appropriate recommendations and referrals when needed (OSHA, 1983). It must be noted, however, that while OSHA allows for the use of technicians, some states do not. Anyone responsible for a hearing conservation program that uses technicians should review the licensure laws and other applicable statutes of the states in which the hearing testing is being conducted. This is to ensure that they are adhering to state laws. OSHA does not require specific training for technicians but does recommend that the technician be certified by the Council for Accreditation of Occupational Hearing Conservationists (OSHA, 1983).

All hearing tests are puretone threshold procedures. They must include the following frequencies: 500, 1,000, 2,000, 3,000, 4,000, and 6,000 Hz, with each ear tested independently. The testing can be conducted manually or by using an automated audiometer calibrated to ANSI standard S3.6-2010. Steady-state or pulsed puretones may be used. Additionally, the testing must be conducted in a room meeting specified background levels as indicated in Table 32.4 (OSHA, 1983).

There are two basic types of audiograms that must be considered as part of a hearing conservation program. These are the baseline tests and the annual tests. The baseline test is the first test administered or the test results in which the most sensitive hearing thresholds have been obtained. Annual tests are conducted to monitor for changes in hearing. A standard requirement for all tests is that the preceding 14 hours be "noise free." Usually, this is the overnight period prior to the test. It is important that workers be cautioned to avoid noisy recreational activities (e.g., loud music, power

TABLE 32.4

Minimum Allowable Octave Band Sound Pressure Levels for Audiometric Test Rooms Used for Testing in Hearing Conservation Programs

Octave band center frequency (Hz)	500	1,000	2,000	4,000	8,000
Sound pressure level (dB)	40	40	47	57	62

Reprinted from Occupational Safety and Health Administration. (1983) Occupational noise exposure: Hearing Conservation Amendment: final rule. *Fed Reg.* 48, 9738–9785.

tool use, gunfire) during this noise-free period, and use of earplugs should not be a proxy for "quiet." If noise cannot be avoided, either at home or at work, then the employee should attempt to keep the exposure time to a minimum and must use effective hearing protection while exposed to levels equal to or in excess of 85 dBA (OSHA, 1983).

The initial baseline test must be obtained within 6 months of the employee beginning work in noise that is at or above the action level. An allowance is made for companies that have their hearing testing conducted by a mobile testing service. In those cases, the initial baseline may be obtained up to 1 year following the first exposure at or above the action level, provided the worker uses effective HPDs after the first 6 months in that setting (OSHA, 1983). Obviously, the closer the initial test is to the commencement of that individual being employed in the high-noise setting, the less likely it is that the data will be contaminated.

Standard Threshold Shift

All workers with exposure at or above the action level must be retested at least annually. The results of the annual test are compared to the baseline test. The frequencies used for the comparison are 2,000, 3,000, and 4,000 Hz, with each ear evaluated independently. If the average hearing sensitivity at these frequencies is 10 dB or worse than the average obtained on the baseline audiogram, then a standard threshold shift (STS) has occurred (OSHA, 1983). This apparently straightforward process is complicated slightly by OSHA's allowance of an age adjustment to account for predicted presbycusis. Therefore, a frequency-by-frequency adjustment must be applied to each of the three comparison frequencies based on the workers' age at the time of the baseline; adjustments are also made to the annual audiogram based on the current age. The averages are then calculated, and the comparison for STS is made (OSHA, 1983). As can be seen in Table 32.5, even though the raw data show a change of greater than 10 dB, the adjusted values may not constitute an STS.

The presence of an STS requires specific action be taken by the company, and it is in these actions that the process becomes rather complex. Under current regulations (29 CFR 1904.10, effective January 1, 2004), a shift needs to be recorded if the average age-corrected change in hearing at 2,000, 3,000, and 4,000 Hz is equal to or in excess of 10 dB *and* the total average hearing loss at those frequencies in the same ear equals or exceeds 25-dB hearing threshold level (HTL) (without age correction) (OSHA, 2006).

OSHA recognizes that some shifts in hearing may be temporary because of medical conditions, TTS, lapses in attention during the test, or simple day-to-day fluctuations in hearing. For a basic STS, there is an allowance that the shift needs to be confirmed before specific action is taken. The confirmation retest must take place within 30 days of the company becoming aware of the shift. Often, the company is aware of the shift on the day the audiogram is conducted, and the retest deadline is based on that date. However, there may be times when the determination of shift is delayed, such as when an outside agency conducts the test. Under those circumstances, the retest deadline may, in fact, be more than 30 days after the test that first showed the shift. If the retest confirms the shift, the employee's company must then notify the worker of the change in hearing. Notification must be in writing. If the company decides not to use the retest option, then the company must notify the employee of the shift within 21 days of the date on which the company first became aware of the shift (OSHA, 1983).

All STSs are presumed to be because of work-related noise exposure unless a physician or other licensed healthcare worker determines that the shift is due to causes other than occupational noise exposure or that work-related noise did not have a significant role in the shift (OSHA, 2006). Additional steps must be taken unless this shift is determined

TABLE 32.5

When Is a Significant Change in Hearing not a Standard Threshold Shift?[a]

		Frequency (Hz)			
		2,000	3,000	4,000	Average
Annual test	Hearing @ 58 years	30	35	40	35
	Presbycusis adjustment	12	22	31	
	Adjusted threshold	18	13	9	13.3
Baseline test	Hearing @ 23 years	5	10	10	8.3
	Presbycusis adjustment	3	4	6	
	Adjusted threshold	2	6	4	4

Actual change in hearing: 35 – 8.3 = 26.7 dB. Corrected change in hearing: 13.3 – 4 = 9.3 dB.
[a]Using the OSHA correction for presbycusis, this worker, who began work with normal hearing through 4,000 Hz and who is now developing a high-frequency hearing loss, would not be identified as having had a significant change in hearing.

medically to be nonwork-related or not to have been exacerbated by workplace noise. If the worker has exposure of less than 90-dBA TWA and is not currently wearing hearing protection, hearing protection must be fitted and the worker must be trained in the care and use of that protection. If the worker is currently using hearing protection, then he or she must be refitted with equipment providing additional attenuation, if needed, and retrained in the appropriate use and care of hearing protection. In either case, HPD use must be sufficient to reduce the exposure to less than 85-dBA TWA under the HPD. If there are signs that additional testing would be audiologically appropriate, then an audiologic referral is needed. If there are signs of a medical problem, either because of or made worse by HPD use, then otologic referral is required. Otologic referral is also required for suspected medical problems unrelated to hearing protection use (OSHA, 1983).

For workers whose exposures are less than 90-dBA TWA, if a subsequent hearing test shows that an STS is not persistent, then the employee must be notified of the new results, and hearing protection use may be discontinued (OSHA, 1983).

Reportable Shift

STSs equal to or greater than 25 dB require additional action. These reportable shifts must be recorded by the employer on the OSHA Illness/Injury Log as soon as they are first identified (OSHA, 1986). These shifts are recorded in the category of occupational illness. Similar to the basic STS, discussed earlier, the company has a retest option to confirm the 25-dB shift, but the rules are different than for a basic (10-dB) STS. When the shift equals or exceeds 25 dB, the retest to confirm or refute a reportable shift must be done within 30 days of the *date of the annual audiogram* on which the shift was noted. This is different than the retest for a basic STS, which has to occur within 30 days of the company *becoming aware* of the shift. If the retest fails to confirm the reportable shift or if a subsequent medical evaluation indicates the reportable shift is because of nonwork-related causes, then the Illness/Injury Log may be amended to reflect those findings. If the retest confirms the shift or if the retest option is not used, then the same follow-up procedures discussed earlier for a basic retest apply; that is, the worker must be notified in writing within 21 days, and the appropriate actions regarding hearing protection must be implemented.

Whenever an STS has occurred, the professional in charge of the hearing conservation program can revise the baseline audiogram to reflect current levels. This revised baseline audiogram serves as the basis for future comparisons to determine the occurrence of a new basic STS (OSHA, 1983). The rationale for this is as follows. If a worker shows a persistent STS, then each subsequent annual test can be expected to show the same STS relative to the initial test. By continually referring back to the initial test, future basic STSs may not be identified easily. Revision of the baseline also avoids overreferral for additional attention. However, annual comparisons for reportable shift (≥25-dB shift) should always be made relative to the *initial* test results. If comparison is not made to the initial results, a reportable shift could be missed. For example, worker Smith develops a 15-dB shift after 10 years of work. This basic STS is confirmed by retest. Worker Smith is notified of the change, and the baseline is revised. Ten years later, worker Smith develops another 15-dB basic STS (compared to the revised baseline). Unless comparison is made of the current test to the initial test, the 30-dB cumulative shift might be missed, and the company would be in violation of the OSHA regulations for failure to record the cumulative change.

Finally, if a worker shows a significant improvement in hearing, then revision of the baseline is also warranted (OSHA, 1983). For instance, some workers present with medical problems during their first test. Once those problems have been treated, hearing may improve significantly. Without a revision of the baseline following an improvement in hearing thresholds, any future noise-induced STS could go unnoticed, and initiation of (more) effective hearing protection could be delayed.

FOOD FOR THOUGHT

Given that our current models of hearing loss risk are based on exposures measured in dBA, and given that we cannot ethically repeat studies of unprotected noise-exposed workers with measurement made in dBC, what can potentially be done to resolve this historical possible measurement error? Is the use of the dBA scale actually an error?

What is the relationship between TTS and PTS? If there is no relationship between the two phenomena why do researchers use TTS as a measure and implicitly assume that this has some ramifications for PTS?

What would be the ramifications for industry and employers if OSHA chose to replace the existing PEL (established in 1972) with the NIOSH REL (established in 1998)?

What are some possible approaches to reducing the daily noise exposure of a flight deck technician on an aircraft carrier when engineering noise controls have already been implemented, the worker is wearing hearing protection, and exposures are still exceeding the PEL?

What is the relative societal noise burden from portable listening devices? Are headphones really a significant source of NIHL?

What exposure levels are most appropriate for application to young people? Estimates of excess risk are based on maximum exposure duration of 40 years. A teenager who is exposed recreationally will have many more than 40 years of potential exposure. Should exposures, then, not exceed 75-dBA TWA with 3-dB exchange rate?

How should an employer apply rules of a hearing conservation program to workers with hearing loss who use hearing aids? Such workers might need hearing aids for communication and situation awareness, but hearing aids (especially those with vents in the earmold/hearing aid shell) typically cannot provide attenuation of ambient sound and might amplify hazardous sound further. How can employers provide reasonable accommodation for these workers while still following regulatory hearing conservation program requirements?

ACKNOWLEDGMENTS

We acknowledge the many contributions by Dr. James Feuerstein from edition 6 of this chapter. We also acknowledge Homira Osman for her considerable editorial contributions in the preparation of this chapter.

KEY REFERENCES

thePoint For more information on the nonauditory effects of noise exposure go to the Point at http://thepoint.lww.com.

American National Standards Institute. (1996) *Determination of Occupational Noise Exposure and Estimation of Noise-Induced Hearing Impairment. ANSI S3.44-1996.* New York: Acoustical Society of America.

American National Standards Institute. (2010) *ANSI Specifications of Audiometers. A Report of the American National Standards Institute S3.6-2010.* New York: Acoustical Society of America.

Baughn WL. (1973) *Relation between Daily Noise Exposure and Hearing Loss Based on Evaluation of 6,835 Industrial Noise Exposure Cases.* Dayton, OH: Wright Patterson Air Force Base.

Berger EH. (2000) Hearing protection devices. In: Berger EH, Royster LH, Royster JD, Driscoll DP, Layne M, eds. *The Noise Manual.* 5th ed. Fairfax, VA: American Industrial Hygiene Association; pp 379–454.

Berger EH. (2005) Preferred methods for measuring hearing protector attenuation. *Proceedings of Inter-Noise 2005*, Rio de Janeiro, Brazil.

Berger EH, Kieper RW, Gauger D. (2003) Hearing protection: surpassing the limits to attenuation imposed by the bone-conduction pathways. *J Acoust Soc Am.* 114 (4), 1955–1967.

Berger EH, Royster LH, Royster JD, Driscoll DP, Layne M, eds. (2000) *The Noise Manual.* 5th ed. Fairfax, VA: American Industrial Hygiene Association.

Chasin M. (2006) Music and hearing aids. *Hear Rev.* March, 34–41.

Embleton TFW. (1994) Report by I-INCE Working Party on "Upper Noise Limits in the Workplace." *Proceedings of INTER-NOISE 94,* Yokohama, Japan.

Environmental Protection Agency. (1974) *Information on the Levels of Environmental Noise Requisite to Protect Public Health and Welfare with Adequate Margin of Safety: A Report of the Environmental Protection Agency (EPA).* Washington, DC: US Environmental Protection Agency.

Environmental Protection Agency. (1981) *Noise in America: The Extent of the Noise Problem.* EPA Report No. 550/9-81-101. Washington, DC: US Environmental Protection Agency.

Fausti SA, Wilmington DJ, Gallun FJ, Myers PJ, Henry JA. (2009) Auditory and vestibular dysfunction associated with blast-related traumatic brain injury. *J Rehabil Res Dev.* 46, 797–810.

Fligor BJ. (in press) Verification of flat attenuation characteristics of Musicians Earplugs™. *J Aud Eng Soc.* Suppl. Proceedings of the AES 47th International Conference, Chicago, USA, 2012 June 20–22.

Fligor BJ, Cox C. (2004) Output levels of commercially available portable compact disc players and the potential risk to hearing. *Ear Hear.* 25, 513–527.

Fligor BJ, Ives TE. (2006) Does headphone type affect risk for recreational noise-induced hearing loss? Paper presented at the Noise-Induced Hearing Loss in Children Meeting, Cincinnati, OH.

Fligor BJ, Wartinger F. (2011) Musicians' Hearing Program. *Audiol Today.* 23 (3), 30–39.

Gasaway DC. (1985) *Hearing Conservation: A Practical Manual and Guide.* Englewood Cliffs, NJ: Prentice-Hall.

Goines L, Hagler L. (2007) Noise pollution: a modern plague. *South Med J.* 100 (3), 287–294.

Henderson D, Bielefeld EC, Harris KC, Hu BH. (2006) The role of oxidative stress in noise-induced hearing loss. *Ear Hear.* 27, 1–19.

Henderson D, Subramaniam M, Boettcher FA. (1993) Individual susceptibility to noise-induced hearing loss: an old topic revisited. *Ear Hear.* 14 (3), 152–168.

Humes LE, Joellenbeck LM, Durch JS. (2005) *Noise and Military Service: Implications for Hearing Loss and Tinnitus.* Washington, DC: The National Academies Press.

International Organization for Standardization. (1990) *Acoustics—Determination of Occupational Noise Exposure and Estimation of Noise-Induced Hearing Impairment.* 2nd ed. Geneva, Switzerland: International Organization for Standardization.

Johnson D. (1991) Field studies: industrial exposure. *J Acoust Soc Am.* 90 (1), 170–174.

Kryter KD, Ward WD, Miller JD, Elridge DH. (1966) Hazardous exposure to intermittent and steady-state noise. *J Acoust Soc Am.* 30, 451–464.

Kujawa SG, Liberman MC. (2006) Acceleration of age-related hearing loss by early noise exposure: evidence of a misspent youth. *J Neurosci.* 26, 2115–2123.

Lempert BL, Henderson TL. (1973) *Occupational Noise and Hearing 1968 to 1972: A NIOSH Study.* Cincinnati, OH: US Department of Health, Education, and Welfare, Public Health Service, Center for Disease Control, National Institute for Occupational Safety and Health, Division of Laboratories and Criteria Development.

LePage EL, Murray NM. (1998) Latent cochlear damage in personal stereo users: a study based on click-evoked otoacoustic emissions. *Med J Aust.* 169, 588–592.

Le Prell CG, Hughes LF, Miller JM. (2007) Free radical scavengers vitamins A, C, and E plus magnesium reduce noise trauma. *Free Radic Biol Med.* 42 (9), 1454–1463.

Liang ZA. (1992) Parametric relation between impulse noise and auditory damage. In: Dancer A, Henderson D, Salvi RJ, eds. *Noise-Induced Hearing Loss.* Philadelphia, PA: B.C. Decker; pp 325–335.

Martin A. (1976) The equal energy concept applied to impulse noise. In: Henderson D, Hamernik RP, Dosanjh DS, Mills JH. *Effects of Noise on Hearing.* New York: Raven Press; pp 421–453.

Matsui JI, Cotanche DA. (2004) Sensory hair cell death and regeneration: two halves of the same equation. *Curr Opin Otolaryngol Head Neck Surg.* 12 (5), 418–425.

Melnick W. (1991) Human temporary threshold shift (TTS) and damage risk. *J Acoust Soc Am.* 90, 147–154.

National Institute for Occupational Safety and Health. (1974) *Occupational Noise and Hearing: 1968–1972: A Report of the National Institute for Occupational Safety and Health (NIOSH).* Cincinnati, OH: National Institute for Occupational Safety and Health.

National Institute for Occupational Safety and Health. (1998) *Criteria for a recommended standard: Occupational noise exposure, Revised Criteria 1998.* DHHS (NIOSH) Publication No. 98-126. Cincinnati, OH: National Institute for Occupational Safety and Health.

National Institute for Occupational Safety and Health. (2003) *The NIOSH Compendium of Hearing Protection Devices.* Updated version. Publication No. 95-105. Cincinnati, OH: US Department of Health and Human Services/Centers for Disease Control, National Institute for Occupational Safety and Health.

Occupational Safety and Health Administration. (1981) Occupational noise exposure: Hearing Conservation Amendment. *Fed Reg.* 46, 4078–4179.

Occupational Safety and Health Administration. (1983) Occupational noise exposure: Hearing Conservation Amendment: final rule. *Fed Reg.* 48, 9738–9785.

Occupational Safety and Health Administration. (1986) *Record Keeping Guidelines for Occupational Injuries and Illnesses: A Report of the US Department of Labor Statistics.* Washington, DC: United States Department of Labor.

Occupational Safety and Health Administration. (2006) Recording criteria for cases involving occupational hearing loss – 1904.10. Available online at: http://www.osha.gov/pls/oshaweb/owadisp.show_document?p_table=STANDARDS&p_id=9641.

Passchier-Vermeer W. (1968) *Hearing Loss due to Exposure to Steady-State Broadband Noise.* Report No. 35. Delft, The Netherlands: Institute for Public Health Engineering.

Portnuff CDF, Fligor BJ, Arehart KH. (2011) Teenage use of portable listening devices: a hazard to hearing? *J Am Acad Audiol.* 22, 663–677.

Price GR, Kalb JT. (1991) Insights into hazard from intense impulses from a mathematical model of the ear. *J Acoust Soc Am.* 90, 219–227.

Prince MM, Stayner LT, Smith RJ, Gilbert SJ. (1997) A re-examination of risk estimates from the NIOSH Occupational Noise and Hearing Survey (ONHS). *J Acoust Soc Am.* 101 (2), 950–963.

Rice CG, Breslin M, Roper RG. (1987) Sound levels from personal cassette players. *Br J Audiol.* 21, 273–278.

Robinson DW. (1971) Estimating the risk of hearing loss due to continuous noise. In: Robinson DW, ed. *Occupational Hearing Loss.* New York: Academic Press.

Rosenhall U, Pedersen K, Svanborg A. (1990) Presbycusis and noise-induced hearing loss. *Ear Hear.* 11 (4), 257–263.

Royster JD. (1996) Noise-induced hearing loss. In: Northern J, ed. *Hearing Disorders.* 3rd ed. Needham Heights, MA: Allyn and Bacon; pp 177–189.

Royster JD, Royster LH, Killion MC. (1991) Sound exposures and hearing thresholds of symphony orchestra musicians. *J Acoust Soc Am.* 89, 2793–2803.

Schotland LI. (1996) Dosimetry measurements using a probe tube microphone in the ear canal. *J Acoust Soc Am.* 99 (2), 979–984.

Smith EL, Laird DA. (1930) The loudness of auditory stimuli which affect stomach contractions in healthy human beings. *J Acoust Soc Am.* 15, 94–98.

Suter AH. (1988) The development of federal standards and damage risk criteria. In: Lipscomb DM, ed. *Hearing Conservation in Industry, Schools and the Military.* Boston, MA: College-Hill Press; pp 45–66.

Taylor W, Pearson J, Mair A, Burns W. (1965) Study of noise and hearing in jute weaving. *J Acoust Soc Am.* 38, 113–120.

Turunen-Rise I, Flottorp G, Tvete O. (1991) Personal cassette players ('Walkman'). Do they cause noise-induced hearing loss? *Scand Audiol.* 20, 239–244.

van Kamp I, Davies H. (2013) Noise and health in vulnerable groups: a review. *Noise Health.* 15 (64), 153–159.

Ward WD, Cushing EM, Burns EM. (1976) Effective quiet and moderate TTS: implications for noise exposure standards. *J Acoust Soc Am.* 59, 160–165.

Williams W. (2012) A proposal for a more refined single number rating system for hearing protector attenuation specification. *Noise Health.* 14 (59), 210–214.

WHO. (1999) *Guidelines for Community Noise.* Berglund B, Lindvall T, Schwela D, eds. Geneva: World Health Organization.

CHAPTER 33

Nonorganic Hearing Loss

Frederick N. Martin and John Greer Clark

INTRODUCTION

Not every patient seen in the audiology clinic is fully cooperative during the hearing evaluation. This lack of cooperation may be because the patient (a) does not understand the test procedure, (b) is poorly motivated, (c) is physically or emotionally incapable of appropriate responses, (d) wishes to conceal a handicap, (e) is deliberately feigning or exaggerating a hearing loss for personal gain or exemption, or (f) suffers from some degree of psychologic disturbance. This chapter will describe some of the concepts underlying false or exaggerated hearing test results and the motivation for this behavior. It will also present some audiometric and nonaudiometric procedures that aid in the detection of inaccuracies and in the determination of a patient's true hearing thresholds.

Many terms have been used to describe a hearing loss that appears greater than can be explained on the basis of pathology in the auditory system. The most popularly used terms in the literature today are "nonorganic hearing loss," "pseudohypacusis," and "functional hearing loss." Such terms as "psychogenic hearing loss" and "malingering" imply the motivation behind the behavior, may be oversimplifications, and certainly may not be describing the same phenomena. In as much as clinicians typically do not know whether an exaggerated auditory threshold is the result of conscious or unconscious motivation, it seems appropriate to use generic terms. The term "pseudohypacusis" is popular but often thought to be a "mouthful." An older term, "hysterical deafness," is based on a Freudian concept of conversion neurosis and is rarely used. Since the word "hysterical" derives from the Greek "hystera" (womb), the term is actually pejorative and its use is inappropriate for this reason as well. In this chapter, "nonorganic hearing loss" will be used to describe responses obtained on hearing-threshold examinations that are above the patient's true organic thresholds.

If one thinks of a hearing loss which is due to physical impairment in the auditory system as being "organic," then the term nonorganic is immediately clear. Many individuals with nonorganic hearing loss have nonorganic aspects superimposed on an organic hearing loss. Audiologists must remember that their primary function is to determine the extent of the organic component rather than to reveal the precise reason for spurious test results. As discussed later, a secondary responsibility when working with nonorganic patients, especially with pediatrics, is to help ensure that any psychologic underpinnings that may exist are addressed by the appropriate professionals.

NONORGANIC HEARING LOSS IN ADULTS

A number of factors may encourage some persons either to feign a hearing loss that does not exist or to exaggerate a true hearing loss. One of these factors is financial gain. Certainly, a significant amount of stress around the world is attributable to economics and the economic upheavals in more recent years have only exacerbated this. The very threat of the loss of income may drive some individuals to acts of "questionable honesty" that they might not otherwise consider.

Other factors that may contribute to nonorganic hearing loss are psychosocial and include the wish to avoid undesirable situations. There may be many other gains that the individual believes are afforded to hearing-disabled persons, including excuses for lack of success, advancement in position, poor marital situation, and so on (Peck, 2011).

The number of persons with nonorganic hearing loss may be increasing since the implementation of federal laws regarding hearing safety in the workplace. Some state laws regarding noise in industry are even more stringent than federal legislation. The promise of financial reward is bound to be a factor precipitating nonorganic hearing loss in workers who are in danger of incurring noise-induced hearing loss. Barelli and Ruder (1970) gathered data on 162 medicolegal patients and found that 24% of the 116 workers applying for compensation proved to have symptoms of nonorganic hearing loss. There is little reason to suspect the percentage may be significantly different today.

Studies suggest that those with nonorganic hearing loss have been found to score lower than those without hearing complaints on measures of socioeconomic status and verbal intelligence. They have also been shown to have a greater degree of clinically significant emotional disturbance, tendencies toward hypochondria, more frequent complaints of tinnitus, and a greater reliance on denial mechanisms. Such

patients appear to have a diminished sense of confidence in their abilities to meet the needs of everyday life and may feel a sense of gain by appearing to have a hearing loss.

There is disagreement over whether some nonorganic hearing loss may be psychogenic at all or whether all exaggerated hearing thresholds are deliberately and consciously manifested with an eye toward personal gain. There are certainly studies (i.e., Gleason, 1958) that suggest that those with nonorganic hearing loss might be influenced by psychodynamic factors but not necessarily psychiatrically ill. Some may be emotionally immature or neurotic and may express psychosomatic complaints or exhibit deviant social behavior. It is possible that in many cases of nonorganic hearing loss, the problem is on an unconscious level to gain a favored goal or to explain to society that the patient is blameless for inadequate social behavior. From this point of view, exaggerated hearing loss may be one symptom of a personality disturbance.

Katz (1980) cautions that certain neurologic problems can appear to be nonorganic in nature. For example, one patient who initially responded on puretone evaluation between 40 and 70 dB HL, and eventually at levels of 20 dB HL, responded immediately at 15 dB HL to spondees. This patient was neither a malingerer nor psychogenic. Rather he was a volunteer for a study because he was terminally ill with a tumor of the corpus callosum. He did not claim to have difficulty with hearing, nor did he exhibit any difficulty in communication. Peck (2011) similarly cautions that patients with what has been dubbed *obscure auditory dysfunction* (Saunders and Haggard, 1989) may be viewed by clinicians as exaggerating the extent of hearing difficulties reported. Certainly, the hearing complaints of these patients in the presence of normal audiometrics are a valid concern and are most likely tied to latent auditory processing difficulties.

The question arises, "Why hearing loss?" Why does the patient with nonorganic hearing loss select this disorder rather than back pain, headache, whiplash, or some other more conventional malady? It certainly is possible that some incident in the lives of these patients has focused their attention on hearing. The incident may have been an ear infection, physical trauma, noise exposure, or tinnitus or hearing loss in a relative or close friend. For whatever reason, this incident is the first step toward future nonorganic hearing loss.

Nonorganic Hearing Loss in Children

A number of case reports of nonorganic hearing loss in children appear in the literature dating back to 1959 when Dixon and Newby (1959) reported on 40 children between the ages of 6 and 18 years with nonorganic hearing loss. Despite claimed hearing losses, 39 of these children were able to follow normal conversational speech with little difficulty. Similar findings have subsequently been reported. Most experienced audiologists can report evaluating children with marked exaggeration of hearing thresholds for puretones in the presence of normal speech recognition thresholds (SRTs).

There are also cases of apparent malingering with psychologic undertones. For example, Bailey and Martin (1961) reported on a boy with normal hearing sensitivity who manifested a great many nonorganic symptoms. After the audiometric examination he admitted a deliberate attempt to create the impression that he had a hearing loss. He claimed he did this so that he could be admitted to the state residential school for the deaf where his parents taught and his sister and girlfriend were students. Investigation into this boy's background revealed that he was a poor student in a high school for normal-hearing students. Hallewell et al. (1966) described a 13-year-old boy with a severe bilateral hearing loss who revealed essentially normal hearing sensitivity under hypnosis.

Cases of presumed psychogenic hearing loss in children have also been reported. Lumio et al. (1969) described three sisters whose hearing losses all appeared to develop over a period of a few months. Two of the three girls also had complaints of visual problems and were fitted with eyeglasses. All three apparently had their hearing return to normal in 1 day during a visit with their aunt. When the hearing returned the visual disorders also disappeared. These authors reported that the nonorganic hearing loss was due to family conflicts. They believed that it was probable that the hearing loss of the youngest child was entirely unconscious, but the other two may have been deliberately simulated.

Investigators have reported, and clinicians frequently have witnessed, cases in which children with normal intelligence, but with a history of poor academic performance, have exhibited significant nonorganic hearing loss. It is likely in such cases that the attention paid to the children failing an initial school hearing test for whatever reason subsequently encouraged them, consciously or unconsciously, to feign a hearing loss on subsequent examinations. Certainly nonorganic behaviors must be detected as early as possible before children realize that there are secondary gains to be enjoyed from a hearing disorder.

Austen and Lynch (2004) point out the obvious, that a dichotomy between acts of deliberate falsification (malingering) and those of unconscious motivation is an oversimplification of what may be a complex human dynamic. They proposed another term, *factitious behavior,* and formulated a new nomenclature for the categorization of nonorganic hearing loss. Theirs is a detailed set of recommendations and the reader is referred to this publication for greater specificity than can be addressed in this chapter.

As noted, sometimes children who inadvertently fail school screening tests become the object of a great deal of attention. It is frequently recommended that professionals avoid recommendations for preferential seating, special classes, hearing therapy, and hearing aids until the extent

of the hearing problem is defined by proper audiologic diagnosis. Identification audiometry is a significant tool for discovering school children with hearing disorders. There is certainly some reason to fear that a child may fail a school test despite normal hearing because of such factors as noisy acoustic environment, improper testing technique, insufficient motivation, or poorly calibrated equipment. If attention is attracted to this inadvertent failure, the child may get the notion that a hearing loss provides a variety of secondary gains, such as excuse for poor school performance. The end result may be referral to an otologic or audiologic clinic for further evaluation of hearing. Ultimately, such cases may be more easily resolved if behaviors of nonorganic hearing loss were uncovered before referrals are made. Nonorganic hearing loss in children appears to occur with sufficient frequency to cause concern. Psarommatis et al. (2009) believe that nonorganic hearing loss is the most frequent underlying etiology of sudden hearing losses in children. Whether the notion of simulating a hearing loss comes out of a school screening failure or from some conscious or unconscious need, it must be recognized as early as possible by the audiologist to avoid a variety of unfortunate circumstances. Performance or supervision of hearing tests on young children by an audiologist, rather than a technician, may serve to avert what may later develop into serious psychologic or educational difficulties.

INDICATIONS OF NONORGANIC HEARING LOSS

The Nontest Situation

Frequently, the source of referral will suggest the possibility of nonorganic hearing loss. For example, when an individual is referred by an attorney after an accident that has resulted in a client's sudden loss of hearing, it is only natural to suspect that nonorganic behavior may play a role in test results. This is also true of veterans referred for hearing tests, the results of which decide the amount of their monthly pensions. Disability compensation to veterans with service-connected hearing losses constitutes a significant portion of the many millions of dollars paid annually to beneficiaries of the Department of Veterans Affairs (VA) in the United States in an effort to compensate them for their disability.

It must be emphasized that the majority of patients referred for such examinations are cooperative and well meaning; however, the VA population consists of a higher-risk group for nonorganic hearing loss than self-referred or physician-referred patients. Nonorganic hearing loss must be on the minds of clinical audiologists or they may miss some of the symptoms that indicate its presence.

A case history is always of value, but it is particularly useful in compensation cases. It is obviously beneficial for examining audiologists to take history statements themselves, so that they can observe not only the responses given to questions, but also the manner in which these responses are offered. The patient may claim an over-reliance on lipreading, may ask for inappropriate repetitions of words, or constantly readjust a hearing aid. It is usual for hard-of-hearing patients to be relatively taciturn about their hearing problems, whereas exaggerated or contradictory statements of difficulty or discomfort, vague descriptions of hearing problems, and the volunteering of unasked-for supplementary information may be symptomatic of nonorganic hearing loss.

In patients with nonorganic hearing loss, exaggerated actions and maneuvers to watch every movement of the speaker's lips, or turning toward the talker with hand cupped over the ear, ostensibly to amplify sound, is sometimes observed. As a rule, hard-of-hearing adults face the talker with whom they are conversing, but their attentive postures are not nearly so tortuous as described above. Not all patients who intend to exaggerate their hearing thresholds create such caricatures, and even patients who do should not be condemned as having nonorganic hearing loss on the basis of such evidence alone.

The Test Situation

During the hearing examination, the patient with nonorganic hearing loss is frequently inconsistent in test responses. A certain amount of variability is to be expected of any individual; however, when the magnitude of this variability exceeds 10 dB for any threshold measurement one must consider the possibility of nonorganic behavior. With the exception of some unusual conditions it can be expected that the cooperative patient will give consistent audiometric readings.

Two types of patient error are frequently seen in the clinical testing of puretone thresholds. These are false-positive and false-negative responses. When the subject does not respond at levels at or slightly above true thresholds, this constitutes a false-negative response. False-negative responses are characteristic of nonorganic hearing loss. Frequently, the highly responsive patient will give false-positive responses, signaling that a tone was heard when none was presented at or above threshold. False-positive responses, although sometimes annoying, are characteristic of a conscientious responder.

It has long been noted that the patient with nonorganic hearing loss does not offer false-positive responses during silent periods on puretone tests. Thus, one simple check for nonorganic behavior is simply to allow silent intervals of a minute or so from time to time. A false alarm is more likely to indicate that the patient is trying to cooperate and believes that a tone was introduced. In the absence of cognitive or physical impairment, extremely slow and deliberate responses may be indicative of a nonorganic problem because most patients with organic hearing losses respond relatively quickly to the signal, particularly at levels above threshold.

The Audiometric Configuration

A number of authors have suggested that an audiometric pattern emerges that is consistent with nonorganic hearing loss. Some have described this pattern as a relatively flat audiogram showing an equal amount of hearing loss across frequencies. Others have suggested that the "saucer-shaped audiogram" similar to a supraliminal equal loudness contour is the typical curve illustrating nonorganic behavior although these saucer-shaped audiograms can certainly occur in true organic hearing losses. This configuration may actually be fairly infrequent in nonorganic hearing loss and the saucer audiogram seems to have limited use in identifying nonorganic hearing loss.

Because the patient with nonorganic hearing loss may attempt to give responses that are of equal loudness at all frequencies, ignorance of the manner in which loudness grows with respect to intensity at different frequencies does suggest that the result should be a saucer-shaped audiogram. The logic of this is apparently not borne out in fact. It would appear that there is no typical puretone configuration associated with nonorganic hearing loss.

Test–Retest Reliability

One indication of nonorganic behavior is the lack of consistency on repeated measures. Counseling the patient about inaccuracies may encourage more accurate responses; however, if this counseling is done in a belligerent way it can hardly be expected to increase cooperation. Sometimes a brief explanation of the test discrepancies encourages improved patient cooperation. By withholding any allegations of guilt on the part of the patient the audiologist can assume personal responsibility for not having conveyed the instructions properly. This provides a graceful way out for many patients, even if they are highly committed to nonorganic loss. Clinicians have long recognized that some children can be coaxed into "listening harder," thereby improving results on puretone tests.

Although these suggestions are usually useful when working with civilian populations, exceptions exist when testing military personnel. When counseling and cajoling fail to eliminate symptoms of nonorganic hearing loss, direct confrontation has been made. Military patients have been told that exaggeration of thresholds is a violation of the Universal Code of Military Justice (in the United States) and therefore a court-martial offense. Personal communication with audiologists working in military installations reveals that such methods may be very effective indeed in altering patient behavior. Veterans with service-connected hearing losses may have their pensions interrupted until examining audiologists are satisfied with test results. It is not known, however, whether this kind of open confrontation may have serious psychologic effects on some patients. It can certainly be offensive if inappropriately used. A personal view is that the risk of psychologic trauma in even a very small percentage of cases should be considered carefully before such aggressive tactics are used. A more prudent approach may be more time-consuming but may also be safer.

The Shadow Curve

It may seem advantageous to a patient feigning a hearing loss to claim that loss in only one ear. Appearing to have one normal ear is convenient because individuals need not worry about being "tripped up" in conversation by responding to a sound that is below their admitted thresholds. In this way all hearing can appear to occur in the "good ear" and the claim can be made that hearing is nonexistent in the "bad ear." Normal activities can be carried on for the unilaterally hearing-impaired individual without any special speechreading abilities.

It is generally agreed that a patient with a profound hearing loss in one ear will hear a test tone in the opposite ear by bone conduction if the signal is raised to a sufficient level during a threshold test. For an air-conduction signal the levels required for contralateralization range from 40 to 70 dB when supra-aural earphones are used, depending on frequency and generally above 70 dB for all frequencies when insert receivers are used. The interaural attenuation, the loss of sound energy because of contralateralization, is much less for bone conduction than for air conduction. With the vibrator placed on the mastoid process there is virtually no interaural attenuation. Therefore, if a person truly has no hearing for air conduction or bone conduction in one ear, the audiogram taken from the bad ear would suggest a moderate unilateral conductive loss. Unless masking is applied to the better ear a "shadow curve" should be expected.

The naive patient with nonorganic hearing loss may give responses indicating no hearing in one ear and very good hearing in the other ear. The lack of contralateral response, especially by bone conduction, is a very clear symptom of unilateral nonorganic hearing loss and offers a good reason why all patients should be tested initially without masking, even if it appears obvious at the outset of testing that masking will be required later in the examination.

SRT and Puretone Average Disagreement

The SRT is generally expected to compare favorably with the average of the lowest two of the three thresholds obtained at 500, 1,000, and 2,000 Hz. Lack of agreement between the puretone average (PTA) and the SRT in the absence of explanations such as slope of the audiogram or poor word recognition is symptomatic of nonorganic hearing loss. Carhart (1952) was probably the first to report that in confirmed cases of nonorganic hearing loss the SRT is *lower* (better) than the PTA with this SRT/PTA discrepancy present in the majority of patients with nonorganic hearing loss. Ventry

and Chaiklin (1965) reported that the SRT–PTA discrepancy identified 70% of their patients with confirmed nonorganic hearing loss; in each case the SRT proved to be more than 10 dB lower than the PTA. The lack of SRT–PTA agreement is often the first major sign of nonorganic hearing loss.

It is impossible to know the precise strategies patients use if they wish to deceive examiners on hearing tests. For one thing, simply asking them their methods would only result in rebuke and, since nonorganic hearing loss is, in many cases, intrinsically deceitful behavior, an honest response would hardly be forthcoming. Martin et al. (2001) paid normal-hearing adults to feign hearing loss for puretones and motivated them by compensating them with more money as their actions became more convincing. Following a series of puretone tests they were simply asked to describe the strategies they used. Most said that they initially responded randomly and then set a sort of loudness metric in their minds and tried to repeat it during retesting, or at different frequencies. After they had taken the test for a short while and realized that a set procedure was being used they began to establish consistency by counting the number of tones, which were initially presented at 30 dB HL and then increased in 5-dB increments. Assuming that this methodology holds true for patients with actual nonorganic hearing loss an obvious procedure would be to vary from an established technique and present tones at random intensities.

In attempting to remember the loudness of a suprathreshold signal previously responded to, one might easily become confused between puretone and spondaic word levels. Very little research has been carried out to explain why the discrepancy generally favors the SRT. It might be that the loudness of speech is primarily associated with its low-frequency components. According to the equal loudness contours, the low frequencies grow more rapidly in loudness than tones in the speech frequencies. This speculation is supported by the work of McLennan and Martin (1976), who concluded that when puretones of different frequencies are compared in loudness against a speech signal, the difference between them is a function of the flattening of the loudness contours. Certainly one could theorize that the difference between the sensations of loudness for speech and puretones may be related to their different sound pressure level references but this theory has its limitations.

SPECIAL TESTS FOR NONORGANIC HEARING LOSS

One responsibility that audiologists bear is to determine the organic hearing thresholds for all of their patients, including those with nonorganic hearing loss although in some cases this may be more readily achieved through behavioral means after the potential underlying motivators for the exhibited nonorganic behavior have been addressed. It is not simply a matter of gathering evidence against the patient to prove nonorganic behavior. This is sometimes necessary, but the unmasking of nonorganic cases should not be an end in itself. Although it is easier to make a diagnosis on cooperative patients in terms of their hearing thresholds, a lack of cooperation does not justify disinterest in the patient's true hearing status. There are tests that qualify or prove the presence of nonorganic hearing loss, those that approximate the true threshold, and those that actually quantify the patient's threshold without voluntary cooperation.

QUALITATIVE TESTS FOR NONORGANIC HEARING LOSS

Acoustic Immittance Measurements

Among the many valuable contributions that immittance testing brings to our profession is the detection of nonorganic hearing loss. The acoustic reflex threshold is the immittance measurement that is of greatest value in the diagnosis of nonorganic hearing loss. The elicitation of this reflex at a low sensation level (SL) (60 dB or less above the voluntary threshold) has been construed to suggest the presence of a cochlear lesion. However, if the SL (the difference in decibels between the acoustic reflex threshold and the voluntary puretone threshold) is extremely low (5 dB or less) it is difficult to accept on the basis of organic pathology. There have even been reports of nonorganic patients who demonstrated acoustic reflexes that were better (lower) than voluntary thresholds. If the audiologist is certain that no artifact contaminates the readings, the suggestion that the acoustic reflex may be achieved by a tone that cannot be heard must be rejected, and a diagnosis of nonorganic hearing loss may be made.

More than merely identifying nonorganic hearing loss, acoustic reflex measurements may be useful in the actual estimation of thresholds. Jerger et al. (1974) describe a procedure in which the middle-ear muscle reflex thresholds for puretones are compared to those for wideband noise and low- and high-frequency filtered wideband noise. The procedure, which is referred to as SPAR (sensitivity prediction from the acoustic reflex), approximates the degree of hearing loss, if any, as well as the general audiometric configuration. This procedure has been shown to estimate thresholds in a large number of cases with a high degree of specificity. It certainly appears that this method may have use in estimating the thresholds of patients with nonorganic hearing loss.

There is no way to know how many patients with nonorganic hearing loss appear to give results reflective of a conductive hearing loss, although we have never seen this. Of course, the middle-ear muscle reflex measurement cannot be used in cases with nonorganic components overlying even mild conductive losses, since contralateral reflexes are absent in both ears when even one ear has a conductive disorder. Tympanometry is an objective method that may be used to suggest middle-ear disorders, in such cases.

The elaborateness of middle-ear measurements, including the instructions for the patient to be quiet and immobile, may have the effect of discouraging nonorganic behavior if this test is performed early in the diagnostic battery. It is often good practice to perform middle-ear measurements as the first test on adults and cooperative children. They are asked to sit quietly and are told that the measurements made will reveal a great deal about their hearing. We have no hesitancy in recommending this approach in general and believe it can be a useful deterrent to nonorganic hearing loss.

Stenger Test

Probably the best way to test for unilateral nonorganic hearing loss is by use of the Stenger test. The Stenger principle states that when two tones of the same frequency are introduced simultaneously into both ears, only the louder tone will be perceived.

Since its introduction as a tuning fork test over a century ago, the Stenger test has been modified many times. If unilateral nonorganic hearing loss is suspected, the Stenger test may be performed quickly as a screening procedure. This is most easily done by introducing a tone of a desired frequency into the better ear at a level 10 dB above the threshold and into the poorer ear at a level 10 dB below the admitted threshold. If the loss in the poor ear is genuine, the patient will be unaware of any signal in that ear and will respond to the tone in the good ear readily, because at 10 dB above threshold it should be easily heard. Such a response is termed a negative Stenger, indicating that the poorer ear threshold is probably correct.

If patients do not admit hearing in the bad ear, and are unaware of the tone in the good ear, they simply do not respond. This is a positive Stenger, which proves that the threshold for the "poorer" ear is better than the response given by the individual. A positive Stenger is the interpretation because the tone is actually above the true threshold in the "bad" ear and precludes hearing the tone in the good ear.

The screening procedure described above rapidly identifies the presence or absence of unilateral nonorganic hearing loss if there is a difference in admitted threshold between the ears of at least 20 dB. The test is most likely to be positive in nonorganic cases with large interaural differences (exceeding 40 dB) or large nonorganic components in the "poorer" ear.

A positive result on the Stenger test does not identify the true organic hearing threshold. To obtain threshold information the Stenger test can also be performed by seeking the *minimum contralateral interference levels* (MCIL). The procedure is as follows: The tone is presented to the good ear at 10 dB SL. There should be a response from the patient. A tone is then presented to the bad ear at 0 dB HL, simultaneously with the tone at 10 dB SL in the good ear. If a response is obtained the level is raised 5 dB in the *bad ear,* keeping the level the same in the good ear. The level is continuously raised in 5-dB steps until the subject fails to respond. Because the tone is still above threshold in the good ear the lack of response must mean that the tone has been heard loudly enough in the bad ear so that the patient experiences the Stenger effect and is no longer aware of a tone in the good ear. Being unwilling to react to tones in the bad ear, patients simply stop responding. The lowest hearing level of the tone in the bad ear producing this effect is the MCIL and should be within 20 dB of the true threshold. An alert patient feigning a hearing loss may "catch on" to what the clinician is doing unless, from time to time, the tone in the good ear is presented without competition from the bad ear.

The Stenger test is equally effective with either an ascending or descending approach with interference levels generally averaging around 14 dB. The result is a close approximation of hearing levels for the "poorer" ear in cases of unilateral nonorganic hearing loss. Monro and Martin (1977) found that the Stenger test, using the screening method, was virtually unbeatable on normal-hearing subjects feigning unilateral hearing losses. Martin and Shipp (1982), using a similar research method, found that as sophistication and practice with the Stenger test are increased, patients are less likely to be confused by low contralateral interference levels. Although the Stenger test, like most tests, has certain shortcomings, most clinicians regard it as an efficient test for quick identification of unilateral nonorganic hearing loss. The only equipment required for the test is a two-channel, puretone audiometer. To be sure, if the test is performed by an inexperienced clinician a series of patterns of tone introductions may betray the intentions of the test to an alert patient. The majority of respondents on a survey of audiometric practices (Martin et al., 1998a) named the Stenger as their most popular test for nonorganic hearing loss, which is difficult to understand since unilateral cases are far and away in the minority.

Modified Stenger Test

A modification of the puretone Stenger test allows completion of the test with spondaic words. The Stenger principle holds for speech stimuli if words, like spondees, are presented via both channels of a speech audiometer simultaneously. All of the criteria for application of the puretone Stenger test apply to the modified version, that is, there should be at least a 20-dB difference between the SRTs of the right and left ears, and the greater the interaural difference and the closer to normal one ear hears the better the test works. A two-channel audiometer is used with either monitored live voice or prerecorded presentation.

Subjects are instructed to simply repeat every spondee they hear. The words are presented 10 dB above the better ear SRT and 10 dB below the poorer ear SRT. If the patient continues to repeat the words the modified Stenger is

considered to be negative, providing no evidence of nonorganic hearing loss. If the patient does not repeat the spondees under these conditions, then the screening test has failed and the MCIL should be sought.

To determine the MCIL, the sensation level of 10 dB should be maintained in the better ear. The hearing level dial controlling the intensity at the poorer ear should be set to the lowest limit of the audiometer. Each time a spondee is presented and repeated by the patient, the level in the poorer ear should be raised 5 dB. The lowest hearing level dial setting in the poorer ear at which the patient stops repeating two or more spondees correctly is considered to be the MCIL and is above the threshold for that ear. The precise threshold cannot be known, but MCILs have been noted as low as 15 dB above the SRT of the poorer ear. If the MCIL is as low as 30 dB HL it may be assumed that hearing for speech is normal.

Experienced clinicians can manipulate the modified Stenger in a variety of ways. The speech itself can be less formal than spondaic words and may consist of a series of instructions or questions requiring verbal responses from the patient. The signal to the better ear may be randomly deleted on the chance that patients may be "on to" the test and may be repeating words they hear in their poorer ears, but will not admit to because they believe that words are also above threshold in their better ears even though they do not hear them. To paraphrase an old saw, "experience is the mother of invention."

Martin and Shipp (1982) found that sophistication with the speech Stenger test resulted in higher MCILs, which can lead the unsuspecting clinician to accept an exaggerated SRT as correct. Because there is no way to control for any knowledge about the modified Stenger that a patient brings to the examination, the alert clinician is wary of contamination of test results that such knowledge may cause.

Ascending–Descending (A-D) Methods

The use of both an ascending and descending approach to puretone threshold measurements has long been recommended as a rapid and simple procedure. A greater than 10-dB difference between these two measurements suggests a nonorganic problem because the two thresholds should be identical. Personal use of this procedure indicates that this difference is often as large as 30 dB for patients with nonorganic hearing loss. For these patients, the ascending method generally reveals lower (better) thresholds than the descending approach. The comparative ascending/descending threshold test is quick and easy to perform with the simplest puretone audiometer and serves as the basis for the BADGE test (Békésy ascending–descending gap evaluation: Hood et al., 1964). This test has been found to be an excellent screening tool for nonorganic hearing loss. Martin et al. (2000) used a combination of stimuli in the development of a screening procedure for nonorganic hearing loss. Using a standard diagnostic audiometer they developed a procedure very much like the BADGE but used standard instead of Békésy audiometry. They compared ascending and descending approaches using tones that were continuously on (CON), pulsing on and off with a standard off time (SOT) as past investigations had used, and pulsing with a lengthened off time (LOT). The CON-SOT-LOT test was described as being rapid and accurate in the detection of nonorganic hearing loss. Subsequent clinical use has borne this out.

The Swinging Story Test and the Varying Intensity Story Test

For some time, a procedure has been available to identify the presence of unilateral nonorganic hearing loss. The test requires the use of a two-channel speech audiometer. A story is read to a patient with portions directed above the threshold of the normal ear (e.g., 10 dB above the SRT) through one channel, other portions below the threshold of the "poorer ear" (e.g., 10 dB below the SRT), and portions through both channels simultaneously.

For this "swinging" test to work, the story must be presented rapidly, including rapid switching from channel 1 to channel 2 to both channels. Although this can be done using monitored live voice, an easier method is to use a prerecording. A calibration tone recorded on each channel allows for adjustment of the volume units (VU) meters before the test begins.

On completion of the recording the patient is simply asked to repeat the story. Repetition of information directed to (and presumably heard in) the good ear or both ears is to be expected. Any remarks from the *bad ear* column must have been heard below the patient's admitted threshold for speech and prove that the threshold for that ear has been exaggerated. All that can be determined from a positive result is that hearing is better in the poorer ear than what the patient has volunteered, providing evidence of nonorganic hearing loss.

One of the advantages of a modification of the swinging story test is that the theme changes when the *bad ear* column is included or excluded, adding to the complexity of the challenge to the patient with nonorganic hearing loss. Because the patient must concentrate on the story and commit it to memory, it is less likely that the individual will be able to remember which material was presented to the *bad* ear.

There is not much evidence that the swinging story test has been very popular. A major revision of this test is called the Varying Intensity Story Test (VIST) (Martin et al., 1998b). Two main advantages to the VIST are that it can be used in one or both ears (not limiting it to unilateral cases) and that it comes close, in many cases, to approximating the auditory threshold for speech.

TABLE 33.1

The Varying Intensity Story Test

Part I	Part II
Presented Above Threshold	**Presented Below Threshold**
China,	despite overpopulation,
is well known for its delicate beauty	and its rugged terrain.
Many popular styles of	cooking originating in
China exist today. Patterns	of beautiful gardens
of flowers and geometric	landscaping
designs are equally common	in many modern Chinese cities.
Hand-painted scenes	of the natural beauty of China
can be found	in many museums
if one knows where to look.	Books about
China owned by your grandmother probably	contain much misinformation because early 20th century China
is quite different from modern China. The computer age	has arrived and
changed the way complex designs are printed	on all types of textiles. A new age has dawned
on modern China.	

To perform the VIST, patients are advised that they will hear a story one time (see Table 33.1), following which they will be asked to respond to a series of 10 written questions. Part I of the story (see above) is presented at 10 dB SL and Part II is presented at 30 to 50 dB below the admitted SRT. The test is considered to be positive if questions resulting from information supplied only from Part II are answered correctly. The interpretation is that the SRT can be no poorer than the level used for Part II. The VIST was shown to work well on subjects simulating nonorganic hearing loss but it remains to be verified on true nonorganic cases.

Low-Level Speech Recognition Testing

Most audiologists tend to perform speech recognition testing at 30 to 40 dB above the SRT. A better approach is to ensure audibility of the speech signal through most of the frequency range to ensure that scores reflect maximum performance levels which often necessitate somewhat higher intensities. Some clinicians routinely do performance-intensity functions for speech recognition scores but these are usually reserved for special cases, such as for determination of site of lesion. Normally, low word recognition scores are expected at low sensation levels. The data in Table 33.2 are adapted from Hopkinson (1978) and suggest approximate word recognition scores that would be attained by normal-hearing individuals at given sensation levels.

It is frequently observed that unusually high word recognition scores can be obtained on patients with nonorganic hearing loss at levels slightly above their admitted thresholds. High scores certainly suggest normal hearing for speech.

Pulse-Count Methods

Some tests may be carried out by presenting a number of puretone pulses in rapid succession and asking the patient to count and recall the numbers of pulses that were heard. The intensity of the tones may be varied above and below the admitted threshold of the tone in one ear (Ross, 1964) or above the threshold in one ear and below the threshold in the other ear (Nagel, 1964). If the originally obtained thresholds are valid the patient should have no difficulty in counting the pulses. Inconsistency should occur only if

TABLE 33.2

Approximate Word Recognition Scores at Given Sensation Levels for Normal Hearing Individuals

Sensation Level (dB)	Word Recognition Score (%)
5	25
10	50
20	75
28	88
32	92
40	100

all the tone pulses are above threshold and the patient has to sort out the number of louder ones from the number of softer ones. This can be very difficult to do. A major advantage to this test is that it can be carried out quickly using any kind of puretone audiometer.

The Yes–No Test

Frank (1976) described a test for nonorganic hearing loss that would seem too simple to work; nevertheless, it often does. The test is intended for children but has occasionally been useful with naive adults. The patient is simply told to say "yes" when a tone is heard and "no" when a tone is not heard. The tone is presented at the lowest limit of the audiometer and increased in intensity in 5-dB steps. Some patients, in an attempt to convince the examiner of poor hearing, will say "no" to tones that are heard below the level selected to be "threshold." Of course, a "no" response that is time-locked with the introduction of a tone is clear evidence that the tone was heard, barring occasional false-positive responses.

QUANTITATIVE TESTS FOR NONORGANIC HEARING LOSS

Despite the considerable interest that has been generated and the appeal of the previously mentioned tests, none so far has provided the most sought after information. They lack the ability to provide the true threshold of audibility in patients who will not or cannot cooperate fully. For measures of more accurate estimates of the actual puretone thresholds, our profession has tended to turn to electrophysiological procedures.

Auditory-Evoked Potentials

Measurement of auditory-evoked potentials (AEP) has long been considered a "crucial test" in the diagnosis of nonorganic hearing loss as results obtained from this technique and from voluntary puretone testing generally agree within 10 dB. The early evoked potentials, the auditory brainstem response (ABR), have proven to be more reliable than the auditory middle latency responses or the auditory late responses in detecting nonorganic hearing loss. Hall (2007) has suggested that a current recommendation for frequency-specific estimation of hearing levels with nonorganic patients is the use of the auditory steady-state response (ASSR) combined with otoacoustic emissions (OAEs). Discrepancies between ASSR thresholds and both ABR and behavioral thresholds have been reported. As such, although ASSR is an important diagnostic tool for evaluating nonorganic hearing loss, it should be interpreted with caution.

Otoacoustic Emissions

Since their introduction into clinical audiologic practice, evoked otoacoustic emissions (EOAEs) have increased in popularity and they serve as an important tool in the diagnosis of auditory lesion site, as well as in estimating hearing sensitivity in noncooperative patients (Dhar and Hall, 2011). OAEs, especially transient-evoked otoacoustic emissions (TEOAEs), have been shown to be of value in cases of nonorganic hearing loss.

EOAEs may reveal that hearing is normal or near normal in patients feigning a hearing loss, but may be of little or no value for those with actual hearing levels greater than about 40 dB HL, who wish the audiologist to believe that hearing is poorer than that. The fact that the adult patient with nonorganic hearing loss is probably aware that some measures of hearing sensitivity are possible without patient cooperation may encourage individuals who arrive at the audiology clinic with plans to falsify test results to become more cooperative when they are prepared for EOAE or AEP tests. This deterrent may be of greater value than the test itself.

Puretone Delayed Auditory Feedback (DAF)

General dissatisfaction has been expressed with speech DAF because it does not reveal the true threshold of the patient with nonorganic hearing loss. Over 50 years ago a procedure was described that uses the delayed feedback notion with puretones and which can be administered to patients who appear unwilling or unable to give accurate readings on threshold audiometry (Ruhm and Cooper, 1964).

During puretone DAF testing, the patient is asked to tap out a continuous pattern, such as four taps, pause, two taps, pause, and so on. The electromagnetic key on which the patient taps is shielded from the individual's visual field. After the patient has demonstrated the ability to maintain the tapping pattern and rhythm, an audiometer circuit is added so that for each tap a tone pulse is introduced into an earphone worn by the patient. The tone has a duration of 50 ms at maximum amplitude but is delayed by 200 ms from the time the key is tapped. If the tone is audible, its presence causes the subject to vary tapping behavior in several ways, such as a loss of rate or rhythm, the number of taps, or an increase of finger pressure on the key.

It has been demonstrated that changes occur in tapping performance at sensation levels as low as 5 dB and are independent of test tone frequency and manual fatigue (Ruhm and Cooper, 1964). Once a subject has demonstrated key-tapping ability, any alterations seen after introduction of a delayed puretone must be interpreted as meaning that the tone was heard.

Not all researchers have found the 5-dB SL change in tapping performance using puretone DAF. Alberti (1970) found that tapping rhythms were disturbed in general at

5 to 15 dB above threshold, but has observed variations as great as 40 dB SL. He reported that some subjects are difficult to test with this procedure because they either cannot or will not establish a tapping rhythm. At times patients appear to fail to understand the instructions and at other times complain that their fingers are too stiff to tap the key.

Two studies (Martin and Shipp, 1982; Monro and Martin, 1977) show puretone DAF to be extremely resistant to effects of previous test knowledge and practice with tones at low sensation levels. The puretone DAF procedure is considerably less time-consuming than many of the electrophysiological methods and has been found to be accurate, reliable, and simple (Robinson and Kasden, 1973). Despite these advantages, puretone DAF is not generally used and commercial devices are not available for this procedure.

OBSOLETE PROCEDURES

There are several procedures that were developed for the diagnosis of nonorganic hearing loss whose days of popularity have come and gone. Some of these tests were more useful than others, but they have all, more or less, been replaced (Martin et al., 1998a). They are briefly discussed here because of their historical significance.

The *Doerfler–Stewart* test (1946) was designed to detect bilateral nonorganic hearing loss by presenting successive levels of noise and spondaic words through both channels of a speech audiometer. Although cooperative patients continue to repeat spondees even with noise levels slightly above threshold, patients who exaggerate their thresholds for both the speech and noise become confused. Probably because of its failure to produce quantitative date, the Doerfler–Stewart test is rarely used today.

Speakers monitor their vocal intensity primarily by means of auditory feedback. When masking is applied to their ears and their thresholds are raised, it is normal for people to speak more loudly in an effort to monitor their voices. This is called the Lombard voice reflex. Theoretically, there should be no change in vocal intensity unless the noise is well above the speaker's threshold, masking the normal auditory feedback. This was the principle of the *Lombard Test,* which has fallen into disfavor because it does not quantify the degree of nonorganic hearing loss present and many false-positive results were observed.

The phenomenon of *delayed speech feedback* has been known for some years. When a subject speaks into the microphone of a recording device and that signal is amplified and played back through earphones, the result is simultaneous auditory feedback and is not unlike what most of us experience as we monitor our voices auditorily. When the feedback is mechanically or electronically delayed by approximately 200 ms, the result is a change in vocal rate and intensity. The major problem with the delayed speech feedback test was that it does not come close to identifying auditory threshold.

In *Békésy audiometry* the locus of auditory lesion is determined by comparison of the threshold tracings obtained with continuous and periodically interrupted tones. Patients with nonorganic hearing loss were reported to manifest a distinct Békésy pattern (Jerger and Herer, 1961) with the tracings for interrupted tones showing poorer hearing than for continuous tones. Several modifications to this test, primarily tied to changes in the off time of the pulsed signal, strengthened its value. To add greater difficulty in Békésy tracings for patients with nonorganic hearing loss, Hood et al. (1964) developed a technique called BADGE. Comparisons are made of auditory thresholds that are approached from high- and low-intensity starting levels, thereby confusing patients who choose to exaggerate their thresholds. Békésy audiometry has not been practiced very much in recent years in large measure because of the extended time it takes for this procedure.

Once the most popular test for nonorganic hearing loss, and formerly required on virtually all veterans seeking compensation for hearing loss, is *electrodermal audiometry* (EDA). The abandonment of EDA is due, in part, to the use of noxious stimuli (electric shocks) as the unconditioned stimuli that were paired with puretones or speech as the conditioned stimuli. According to the model, once conditioning was established by pairing conditioned and unconditioned stimuli, the unconditioned response (drop in electrical skin resistance) to the unconditioned stimulus (shock) would be seen in addition to a conditioned response in reaction to the tone or speech alone. In part, because of the discomfort and possible liabilities involved with this procedure and the concern on the part of some audiologists regarding the validity of EDA, it has fallen into virtual disuse. There are also some tragic stories about misdiagnoses in children.

TEST SEQUENCE

During routine audiometry the precise order in which tests are done probably does not have a significant effect on results. However, patients with nonorganic hearing loss probably attempt to set a level above threshold as a reference for consistent suprathreshold responses. For this reason threshold tests should be performed before suprathreshold tests.

The following test order has proved useful in examining patients with suspected nonorganic hearing loss: (a) Immittance measures; (b) OAE; (c) SRT, including the modified Stenger test in unilateral cases; (d) air-conduction thresholds, including the Stenger test if indicated; (e) word recognition tests at low sensation levels; (f) bone-conduction thresholds; and (g) ABR or ASSR.

TINNITUS

Tinnitus, a term taken from the Latin "tinnire" meaning "to ring," has become a major health issue. Among all the claims

to the Department of Veterans Affairs (in the United States) for service-connected compensation the most common is for tinnitus. Certainly, the majority of these VA claims are honest and truthful. Nevertheless, for those whose aim is to gain or increase pension benefit, tinnitus may be a tempting way to acquire compensation by exaggerating or fabricating this symptom.

Most tinnitus sufferers also experience hearing loss, which is now the second-most common claim to the VA. The degree of hearing loss can be determined through audiometric testing. However, for purposes of recompense, a system for evaluation of tinnitus is necessary. At this time, there are no objective tests to substantiate tinnitus complaints (e.g., Byun et al., 2010). Therefore, assessment of tinnitus is mainly based on reports by the claimant. For members of the military and veterans, the degree of disability is determined by completion of a questionnaire. If the problem is determined to be recurrent, the usual compensation is a 10% disability. This percentage may be different in other countries.

Since tinnitus is a growing problem there is a great need for research to lead to better assessment measures. As surely as it is important to determine when tinnitus claims are feigned or intensified, it may be even more important to find ways to fairly compensate individuals who truly suffer from this vexing symptom.

COUNSELING NONORGANIC PATIENTS

Counseling sessions should be carried out after all audiologic evaluations. Counseling the individual with nonorganic hearing loss is naturally more difficult than counseling patients with an organic hearing disorder. Peck (2011) notes that nonorganic hearing loss should be viewed as a possible symptom of an underlying psychosocial problem. If the audiologist is able to get the patient to admit true organic thresholds the underlying problem may persist. Whereas adults more often present nonorganic hearing loss for financial gain, this is unlikely the motivation for children.

Children may be told only that their hearing appears to be normal (if this is believed to be the case) despite audiometric findings to the contrary. Parents should be cautioned not to discuss their children's difficulties in their presence or to provide any secondary rewards that may accompany a hearing loss. The audiologist should question the child and/or the parent on potential difficulties or disturbances that may have led the child to seek attention by feigning hearing loss. Raised concerns should be investigated further through referral to the school psychologist. It should be explained to the parent that consultation with the school counselor can help identify issues that may have led to the hearing test failure, so the child and the family can address these concerns together. The resultant supportive therapy from such referrals is often the most efficacious treatment for the child with nonorganic hearing loss (Andaz, Heyworth, & Rowe, 1995). Parents should be encouraged to see their children not as deceitful, but as resourceful. It is the clever child who can create circumstances that provide some psychologic support when confronting significant life stressors (Clark, 2002).

Adults with nonorganic hearing loss may simply have to be told that a diagnosis of the extent of the hearing disorder cannot be made because inconsistencies in response preclude accurate analysis. Peck (2011) suggests relaying to patients that some people coming in for a hearing test seem preoccupied with other concerns that seem to interfere with listening during the test. Asking the patient if that seems applicable may help to uncover underlying issues that should be addressed by a mental health professional. More in-depth questioning might be offered by asking if the patient is troubled by anything in particular with friends or at home or on the job. Guidelines for the audiologist broaching mental health referrals are given elsewhere (Clark and English, 2014). In general, if a referral for psychologic evaluation or guidance is indicated, a referral should be made with confidence stating that the audiologist believes it may be beneficial to talk with an expert in dealing with difficult life situations. It is at this juncture that audiology must be practiced as more of an art than a science.

DISCUSSION

In the vast majority of cases, the detection of nonorganic hearing loss is not a difficult task for the alert clinician. The more challenging responsibility of the audiologist is to determine the patient's organic thresholds of hearing, however the difficulty of this task increases as the cooperation of the patient decreases. Some patients with nonorganic hearing loss are overtly hostile and unwilling to modify their test behavior even after counseling.

It is not likely that a single approach to diagnosis and resolution of nonorganic hearing loss is forthcoming, although there are certain points on which we should all agree. For example, it is far better to discourage exaggeration of hearing thresholds at the outset of testing than to detect and try to correct these exaggerations later. Once nonorganic hearing loss is observed, the audiologist is faced with the responsibility of determining the true organic thresholds. Tests that may aid in deterring elevated responses include all the electrophysiological and electroacoustic procedures. In our opinion, acoustic immittance measurements and OAEs should be accomplished initially in all routine audiologic assessments, thereby discouraging some nonorganic hearing loss. The Stenger test is quick and easy to perform, where applicable, and like immittance testing and OAEs allows the patient to realize that the examiner has methods of determining puretone thresholds, even without patient cooperation.

Great care must be taken in writing reports about patients with suspected nonorganic hearing loss. It must be borne in mind that once individuals have been diagnosed as "malingering," "uncooperative," or "functional," their reputations and prestige may be damaged. To label a patient in such ways is a grave matter because it implies deliberate falsification. Such labels are difficult to expunge and may be tragically unjust. The only way an audiologist can be absolutely certain that a patient with nonorganic hearing loss is truly a malingerer is for the patient to admit to the intent, and most experienced audiologists would probably agree that such admissions are rare indeed. Value judgments are not within the purview of the audiologist and should be avoided.

CONTROVERSIAL ISSUES

Few subjects in the profession of audiology are as open to disagreement as the issue of nonorganic hearing loss. One of the most contentious is probably the terminology that should be used which was discussed earlier. Another would certainly be the believed causes of this condition.

Since the early days of the profession of audiology, there was a belief that the cause of individuals feigning or exaggerating hearing loss could be broken down into two major categories. First, there were those pretending to have a hearing loss (or other medical condition) for personal gain or freedom from an obligation. This deliberate action is called malingering (the prefix meaning bad, wrong, or improper). In other words, this is a hearing loss that is just made up in its entirety or exaggerated. It is a pejorative term that has doubtlessly caused much consternation and litigation over the many years of its use. It is the view of these authors that this is a term that should never be used verbally or in a report unless the patient admits to deliberate falsification, which, over our combined clinical experience of more than 80 years, has never been experienced.

Second, as far as etiology is concerned, the "other" cause was believed to be of a psychologic or psychiatric nature. It was believed by some that there are patients with normal or near-normal hearing who could not hear because unconsciously they did not want to hear. By its nature this would be a hearing loss with no discernible organic cause. There have been case studies of such unconscious behavior published over the years suggesting this to be true in some instances, but this is far from conclusive. In truth the whole matter of nonorganic hearing loss is complex and multivariate. Every case is different and such factors as education, age, occupation and life experience have complex influences that exist on a long continuum. Decisions made about the handling of patients with nonorganic hearing loss are very much influenced by the philosophy of the audiologist. There are still clinicians who believe that all those exhibiting nonorganic hearing loss are simply liars, and others who believe that the behavior is entirely unconscious.

FOOD FOR THOUGHT

1. Given that studies suggest that those with nonorganic hearing loss tend to have a greater degree of clinically significant emotional disturbance, tendencies toward hypochondria, and a greater reliance on denial mechanisms, should the audiologist refer these patients for mental health services following determination of hearing status? How might you determine who might benefit from a consultation with a professional counselor?
2. Do you agree with the authors' recommended test sequence for suspected nonorganic hearing loss and if not what would you do differently and why? Do you believe that the audiological test sequence should be standard for all patients?

3a. You have tested an eleven-year-old boy whose results are clearly indicative of nonorganic hearing loss. While your standard test results have not demonstrated frequency-specific normal hearing thresholds, you are confident that the hearing is within the normal range in both ears. What are the advantages to pursuing testing further to gain clear documentation of normal hearing? What are the disadvantages to doing this?

3b. In question 3a no specific tests for nonorganic hearing loss were utilized. Under what circumstances would you perform such tests, name those you would use and the order in which you would apply them.

3c. List the usual signs of nonorganic hearing loss. Include patient behaviors, anomalies on routine tests, history, and sources of referral.

3d. What might you say to a patient for whom you believe a professional counselor should be brought into the picture? Be prepared for lack of acceptance and even hostility from the patient and/or the significant others who might accompany him or her.

KEY REFERENCES

thePoint A full list of references for this chapter can be found at http://thePoint.lww.com. Below are the key references for this chapter.

Alberti P. (1970) New tool for old tricks. *Ann Otol Rhinol Laryngol.* 79, 900–907.

Andaz C, Heyworth T, Rowe S. (1995) Nonorganic hearing loss in children - A two year study. *Journal of Oto-Rhino-Laryngology and Its Related Specialties.* 57, 33–55.

Austen S, Lynch C. (2004) Non-organic hearing loss redefined. Understanding, categorizing and managing non-organic behavior. *Int J Audiol.* 45, 283–284.

Bailey HAT Jr, Martin FN. (1961) Nonorganic hearing loss: case report. *Laryngoscope.* 71, 209–210.

Barelli PA, Ruder L. (1970) Medico-legal evaluation of hearing problems. *Eye Ear Nose Throat Mon.* 49, 398–405.

Byun Y, Seung GY, Moon SP. (2010) Reliability of repeated tinnitogram as malingering test. *Otolaryngol Head Neck Surg.* 143 (2 suppl), 246.

Carhart R. (1952) Speech audiometry in clinical evaluation. *Acta Otolaryngol (Stockh)*. 41, 18–48.

Clark JG. (2002) If it's not hearing loss, then what? Confronting nonorganic hearing loss in children. *Audiol Online*. Available online at: http://www.audiologyonline.com/articles/search/all/term:nonorganic/.

Clark JG, English KM. (2014) *Counseling-Infused Audiologic Care.* Boston, MA: Pearson Education.

Dhar S, Hall JW. (2011) *Otoacoustic Emissions: Principles, Procedures and Protocols.* San Diego, CA: Plural Publishing.

Dixon RF, Newby HA. (1959) Children with nonorganic hearing problems. *AMA Arch Otolaryngol.* 70, 619–623.

Doerfler LG, Stewart K. (1946) Malingering and psychogenic deafness. *J Speech Disord.* 11, 181–186.

Frank T. (1976) Yes-no test for nonorganic hearing loss. *Arch Otolaryngol.* 102, 162–165.

Gleason WJ. (1958) Psychological characteristics of the audiologically inconsistent patient. *Arch Otolaryngol.* 68, 42–46.

Hall JW. (2007) *New Handbook of Auditory Evoked Responses.* Boston, MA: Allyn & Bacon.

HalleweII JD, Goetzinger CP, Allen ML, Proud GO. (1966) The use of hypnosis in audiologic assessment. *Acta Otolaryngol (Stockh)*. 61, 205–208.

Hood WH, Campbell RA, Hutton CL. (1964) An evaluation of the Békésy ascending descending gap. *J Speech Hear Res.* 7, 123–132.

Hopkinson NT. (1978) Speech tests for pseudohypacusis. In: Katz J, ed. *Handbook of Clinical Audiology.* Vol 25. 2nd ed. Baltimore, MD: Williams & Wilkins; pp 291–303.

Jerger J, Burney L, Mauldin L, Crump B. (1974) Predicting hearing loss from the acoustic reflex. *J Speech Hear Disord.* 39, 11–22.

Jerger J, Herer G. (1961) Unexpected dividend in Békésy audiometry. *J Speech Hear Disord.* 26, 390–391.

Katz J. (1980) Type A and functional loss. *SSW Newslett.* 2, 5.

Lumio JS, Jauhiainen T, Gelhar K. (1969) Three cases of functional deafness in the same family. *J Laryngol Otol.* 83, 299–304.

Martin FN, Champlin CA, Chambers, JA. (1998a) Seventh survey of audiometric practices in the United States. *J Am Acad Audiol*. 9, 95–104.

Martin FN, Champlin CA, Marchbanks T. (1998b) A Varying Intensity Story Test for simulated hearing loss. *Am J Audiol.* 7, 39–44.

Martin FN, Champlin CA, McCreery TM. (2001) Strategies used in feigning hearing loss. *J Am Acad Audiol.* 12, 59–63.

Martin FN, Monro DA. (1975) The effects of sophistication on Type V Békésy patterns in simulated hearing loss. *J Speech Hear Disord.* 40, 508–513.

Martin FN, Shipp DB. (1982) The effects of sophistication on three threshold tests for subjects with simulated hearing loss. *Ear Hear.* 3, 34–36.

Martin JS, Martin FN, Champlin CA. (2000) The CON-SOT-LOT test for nonorganic hearing loss. *J Am Acad Audiol.* 11, 46–51.

McLennan RO, Martin FN. (1976) *On the Discrepancy Between the Speech Reception Threshold and the Pure-Tone Average in Nonorganic Hearing Loss.* Houston, TX: Poster Session at the American Speech and Hearing Association Convention.

Monro DA, Martin FN. (1977) Effects of sophistication on four tests for nonorganic hearing loss. *J Speech Hear Disord.* 42, 528–534.

Nagel RF. (1964) RRLJ–a new technique for the noncooperative patient. *J Speech Hear Disord.* 29, 492–493.

Peck JE. (2011) *Pseudohypacusis: False and exagerated hearing loss.* San Diego, CA: Plural Publishing.

Psarommatis L, Kontorinis G, Kontrogiaannis A, Douniadakis D, Tsakanikos M. (2009) Pseudohypacusis: the most frequent etiology of sudden hearing loss in children. *Eur Arch Otorhinolaryngol.* 266, 1857–1861.

Robinson M, Kasden SD. (1973) Clinical application of pure tone delayed auditory feedback in pseudohypacusis. *Eye Ear Nose Throat Mon.* 52, 91–93.

Ross M. (1964) The variable intensity pulse count method (VIPCM) for the detection and measurement of the pure tone threshold of children with functional hearing losses. *J Speech Hear Disord.* 29, 477–482.

Ruhm HB, Cooper WA Jr. (1964) Delayed feedback audiometry. *J Speech Hear Disord.* 29, 448–455.

Saunders GH, Haggard MP. (1989) The clinical assessment of obscure auditory dysfunction. 1. Auditory and psychological factors. *Ear Hear.* 10, 200–208.

Ventry IM, Chaiklin JB. (1965) Evaluation of pure tone audiogram configurations used in identifying adults with functional hearing loss. *J Aud Res.* 5, 212–218.

CHAPTER 34

Hearing Loss in the Elderly: A New Look at an Old Problem

Barbara E. Weinstein

DEMOGRAPHICS OF AGING AND HEARING LOSS

We are living during historic times. The demographics of aging have changed dramatically with the coming of age of the "baby boomers" who began to turn 65 in 2011. Worldwide, the older population is growing at a dramatic rate; the world's 65-and-older population is projected to triple by midcentury, from 516 million in 2009 to 1.53 billion in 2050. The growth rate for the population of persons of 65 years of age is expected to outpace that for the total population rather dramatically, such that by 2040 it will be one in five. The older population is getting older with the most dramatic growth among those over 85 years of age, namely the "oldest-old." By 2030, people aged 85 or over, will nearly double the number in 2002. Although the number of people aged 75 and over who are employed is relatively small they had the most dramatic gain in employment—increasing by close to 170% from 1977 to 2007. Increasingly, job growth for older workers is most dramatic in the service sector, where audition is imperative for effective communication and transaction of business.

Hearing Loss and Comorbidities

It is notable that older adults with hearing loss have the increased burden of medical comorbidities relating to aging with more than 50% of older adults having three or more chronic diseases (i.e., multimorbidities). According to a recent report by Crews and Campbell (2004) individuals with hearing loss, vision loss, and dual sensory loss have an increased likelihood of presenting with comorbid conditions ranging from falls to hypertension to cardiovascular disease. Age is the most common risk factor for vision and hearing impairment in older adults and it follows that many older adults present with dual sensory impairments. Age-adjusted rates of hearing and visual impairment were significantly higher in men than women.

Using data from the National Health and Nutrition Examination Survey (NHANES), Bainbridge, Hoffman and Cowie (2008) compared hearing levels of adults with and without a diagnosis of diabetes. At all decades (1920s through 1960s), individuals with diabetes presented with poorer hearing threshold levels across frequencies than did those without diabetes and the difference appeared to be greatest in the higher frequencies. McMahon et al. (2008) found that smoking and diabetes were significantly associated with an increased odds of hearing loss confirming a probable link to age-related hearing loss (ARHL). Diabetes mellitus is associated with hearing impairment and cochlear microvascular dysfunction may be at the root of the association (Helzner et al., 2011).

A number of investigators have recently explored the link between memory, hearing, and cognitive function. Memory impairment and hearing impairment are considered common aspects of aging. In fact, using a sample from the Third NHANES, Li et al. (2006) found that self-reported functional hearing impairment and memory impairment were prevalent, but not comorbid in their sample of adults 65 years of age and older. According to newly emerging data, hearing loss is independently associated with an increased risk of cognitive decline over time with significant associations between greater hearing loss and poorer cognitive function (Lin et al., 2013). Using participants in the Baltimore Longitudinal Study, Lin et al. (2011) conducted a prospective study of 639 adults ranging in age from 36 to 90 years to determine the degree to which hearing impairment is associated with incident all-cause senile dementia and Alzheimer disease. All participants underwent audiometric testing and cognitive testing overtime. The risk of incident dementia increased with severity of hearing loss with adults who had moderate hearing loss were more likely than those with normal hearing to develop dementia and adults with severe hearing loss were at greatest risk.

Social isolation and loneliness, which are correlates of hearing loss and susceptibility to falls, may be possible factors mediating the link between hearing loss and cognitive decline (Cacioppo and Hawkley, 2009; Cacioppo et al., 2010; Lin et al., 2013). Weinstein and Ventry (1982) conducted one of the first audiologic studies on social isolation, demonstrating a stronger link between subjective social isolation, hearing handicap, and hearing loss than between objective social isolation and audiometric variables. We now know that there is also a link among social isolation, self-reported hearing difficulties, and depression. It is notable that individuals with visual impairment are susceptible to

falls and are at risk for activity limitations and consequent social isolation. Older adults with both vision and hearing loss were more likely than those without to have sustained a fall, and accordingly reported fewer social engagements and increased difficulty with activities of daily living. As impaired hearing is associated with greater risk for falls and possible attendant injuries, people with hearing impairment may have more mobility limitations than people without hearing impairment adding to the potential for decreased social engagement (Karpa et al., 2010; Viljanen et al., 2009). Viljanen et al. (2009) explored the relation between walking difficulty and hearing impairment in a female sample. In this study, participants underwent baseline hearing tests and tests of maximal walking speed and walking endurance. They were also asked to self-rate their walking difficulties. At the 3-year follow-up, participants were asked to once again self-rate their walking difficulties. It was of interest that at baseline women with hearing impairment had two times the risk of having major difficulties walking 2 km than those without hearing impairment. Interestingly, participants with hearing impairment were more likely than those without hearing impairment to develop walking difficulties at follow-up. Viljanen et al. (2009) speculated that hearing impairment correlates with mobility as a result of such factors as impaired postural balance and greater risk of falls. Alternatively, it may well be that impaired hearing places greater demand on attention sharing, thereby making mobility more of a challenge. It is important to note that in this sample, the overwhelming majority of people with hearing impairment had mild hearing loss and were not hearing aid users. The fact that people lacking in social connections and reporting frequent feelings of loneliness (i.e., persons with hearing impairment and persons prone to falls) tend to suffer higher rates of cognitive decline, depression, morbidity, and mortality may explain, in part, the link (Cornwell and Waite, 2009).

Helzner et al. (2011) explored the relationship between hearing impairment and cerebrovascular disease (CVD). It was of interest that risk factors for CVD, such as higher levels of triglycerides and history of smoking (in men), higher BMI (in women), and higher resting heart rate, tended to be associated with poorer puretone hearing levels, whereas clinical CVD did not seem to bear any relationship to hearing impairment. Helzner et al. (2011) speculated that insufficient cochlear blood supply may disrupt the chemical balance of endolymph, which in turn can affect the electrical activity of the hair cells and may compromise the activation of the auditory nerve. The authors concluded that "prevention of CVD and its contributing factors has the potential to slow the progression of age-related hearing loss" (Helzner et al., 2011, p. 978).

There are a number of modifiable and nonmodifiable risk factors for ARHL. The nonmodifiable factors include age, genetic predisposition, race, and gender (Yamasoba et al., 2013). The modifiable risk factors include noise, smoking, ototoxicity, and multiple health comorbidities including CVD, cardiovascular disease, diabetes, and cognitive decline. When present in older adults with chronic conditions ranging from CVD to diabetes and falls, hearing loss is likely to increase the burden of these conditions and at times can exacerbate or be exacerbated by these medical conditions. Hence, audiologists should work with primary care professionals to develop early intervention protocols to reduce the potential burden of hearing loss in persons with multimorbidity.

THE AGING AUDITORY MECHANISM

The field of "otogerontology" has made significant strides in documenting the anatomic, physiological, and audiologic changes within the peripheral and central auditory mechanisms. Current thinking is that the auditory system is an integrated one involving an interplay among its many components including the ear and the brain (Weinstein, 2013). More recent thinking is that poor output from the peripheral auditory system, due in part to age-related changes, reduces the quality of the input to the central auditory system and ultimately the communication challenges associated with ARHL. For some, the central locus may underlie the age-related declines in auditory temporal processing key to speech understanding, whereas for others, it may well be the cognitive changes associated with age-related changes in portions of the brain. The lack of uniformity may help to explain the individual differences in speech understanding in challenging acoustic environments which is the hallmark of ARHL. Presbycusis is the term traditionally used when referring to hearing loss in older people (Gates and Mills, 2005).

Although noteworthy, age-related changes in the periphery, including the outer and middle ears, have few implications for communication ability. There is a loss of elasticity and strength in the pinna and external auditory canal. The sebaceous and cerumen glands in the cartilaginous portion lose some of their secretory ability. The decrease in fat leads to significant changes in the skin lining the canal. Changes in the physical properties of the skin, including dryness and dehydration, make the canal prone to trauma and breakdown. Cerumen becomes more concentrated, hard, and impacted due, in part, to inadequate epithelial migration (Weinstein, 2013). The bony canal is especially susceptible to trauma from attempts at cerumen removal because the skin covering is very thin (0.2 mm in thickness). The shape and age-related changes within the ear canal may have implications when making earmold impressions for hearing aids located completely in the ear canal and will influence hearing aid fittings especially with some of the newer deep insertion hearing aids (Weinstein, 2013).

Cerumen impaction can occur in the outer ear because of increased activity of cerumen glands in the cartilaginous portion, physical obstruction because of a hearing aid,

frequent use of cotton-tipped swabs, or the production of drier and less viscous cerumen. Combined with the presence of thicker and longer hair follicles oriented toward the tympanic membrane, the latter condition leads to a higher rate of impaction among older adults. One of the most common reasons for physician visits is accumulation of cerumen because of failure of the self-cleaning mechanism. A common reason for a primary care visit is accumulation of excessive cerumen. Cerumen impaction is present in approximately one-third of older adults, with estimates being quite high among nursing home residents. Developmentally delayed adults, people with intellectual challenges and cognitive impairments, are prone to impacted cerumen (Roland et al., 2008).

The site of conversion of mechanical energy to an electrophysiological signal, the inner ear is composed of several functional components that are vulnerable to the effects of aging. These components are sensory, neural, vascular, metabolic, supporting, synaptic, and/or mechanical (Weinstein, 2013). The most critical risk factor for the auditory sense organ is age yet genetic susceptibility and noise exposure play a role, as well (Lin, Thorpe, Gordon-Salant, Ferrucci, 2011). Although the organ of Corti is most susceptible to age-related changes, structural and chemical changes occur throughout the peripheral and central auditory systems (Tremblay and Ross, 2007). Age-related atrophy ultimately interferes with the transduction process integral to the reception of sound. Knowledge of changes in the aging cochlea is based primarily on histopathologic studies of human temporal bones and more recently on animal models using a variety of animals (Frisina et al., 2009; Schuknecht, 1955; Schuknecht and Gacek, 1993).

The primary histopathologic changes in the organ of Corti include sensory cell degeneration along with loss of supporting cells including Deiters, pillars, and Hensen cells. In general, loss of hair cells begins in the extreme basal end where it is most severe with the outer hair cells degenerating first. Degeneration of the outer row of outer hair cells is often more severe than in the other rows. Decrease in hair cell population is greatest in persons over 70 years of age. It is important to note that outer and inner hair cells tend to degenerate independently. It is now well accepted that degeneration of outer hair cells may in fact be due in large part to noise trauma in addition to age. Loss of nerve fibers in one or more turns of the cochlea has been noted without severe hair cell loss. The loss of inner or outer hair cells is not a prerequisite for age-related pathology of ganglion cells; however, inner hair cell loss is almost always associated with ganglion cell loss. Hence, shrinkage of afferent nerve fibers and their cell bodies, even with inner hair cells present, is a classic finding associated with aging. There is a relationship between amount and location of ganglion cell loss and puretone thresholds. Hearing loss first occurs once the neural unit population falls below the number required for processing acoustic energy (Suzuka and Schuknecht, 1988). In contrast, speech recognition ability cannot be predicted from spiral ganglion cell population.

Recently, a number of neuronal changes have been documented with increasing age. Age-related changes have been noted to affect neural synchrony which is associated with reduced amplitude of the action potential, decreased neural inhibition (Caspary et al., 2005), and longer neural recovery time (Walton et al., 1998). In addition, there is a reduced number of neurons in the auditory nuclei, synaptic changes between inner hair cells and the auditory nerve, and changes in the level of inhibitory neurotransmitters (Caspary et al., 2005; Clinard et al., 2010; Weinstein, 2013). The neural representation of sounds is altered in the aged central auditory nervous system and there is an age-related loss of acoustic nerve activity with both contributing to the processing problems experienced by many older adults especially in the temporal domain (Frisina and Walton, 2006). It is now well accepted that the consequences of the changes in the peripheral auditory system are seen throughout the central auditory system including the cochlear nucleus, the inferior colliculus (IC), medial geniculate body, and the primary auditory cortex. Specifically, auditory deprivation in the periphery disrupts the tonotopic organization in the midbrain and cortex. There is central auditory reorganization because of neural plasticity in which intact regions of the tonotopic map adjacent to the impaired regions tend to become responsive (Tremblay and Kraus, 2002). Stated differently, reorganization of the auditory cortex and the central auditory system is widespread because of peripheral and central changes which take place with aging.

Although Schuknecht's 1950s histologic technique was crude by today's standards, his work did result in the classification of presbycusis into several distinct types including sensory, neural, metabolic, cochlear conductive, mixed, central, and indeterminate (Gates and Mills, 2005). Difficult to distinguish histologically and clinically from acoustic trauma, the audiometric pattern associated with sensory presbycusis is typical of noise-induced hearing loss. Based on data accumulated using distortion product otoacoustic emissions (OAEs) and audiograms, sensory presbycusis may not be as prevalent in older adults as once theorized (Gates et al., 2002). In fact, sensory presbycusis appears to have more to do with long-term exposure to environmental toxicities such as noise, than with age, per se.

A consistent pathologic change associated with neural presbycusis is degeneration of the population of neural units. Neuronal loss tends to diffuse, involving all three turns of the cochlea. Neuronal loss in the periphery, which may begin at any age, is often accompanied by loss of neurons in the ventral and dorsal cochlear nuclei. As the deleterious effects of aging are typically first seen in highly metabolic tissue in the body, it is not surprising that the most prominent feature of ARHL is atrophy of stria vascularis, an area very high in metabolic activity (Schmiedt, 2010). Current thinking is that age-related strial degeneration reduces the

endolymphatic potential (EP), which in turn is responsible for reduced activity in the inner hair cells. The latter changes translate into reduced activity in the auditory nerve and loss of neural synchrony (Ison et al., 2010; Tun et al., 2012). Specifically, according to data accumulated using animal models, there is a direct correlation between strial degeneration and EP voltage and when strial degeneration exceeds 50%, EP values drop rather substantially (Gates and Mills, 2005). The change in the EP with age has given rise to the dead battery theory of presbycusis. Low-frequency hearing loss is typical of persons with strial presbycusis and there is a hereditability factor associated with strial prebycusis, which appears to be more pronounced in women (Gates and Mills, 2005). Age-related degeneration of stria vascularis is the most common feature ARHL (Gates and Mills, 2005).

Cochlear conductive presbycusis is associated with changes in the physical properties of the cochlea such as loss of elasticity of the basilar membrane, which affects its mechanical response. Schmiedt (2010) suggested that mechanical presbycusis may merely be an extreme case of metabolic presbycusis, as from animal models we know that a very low EP is associated with a mild, flat audiogram with hearing loss greater in the low frequencies. Mixed presbycusis is characterized by the involvement of two or more of the four classic types of presbycusis. For example, the combination of sensory and strial presbycusis might present as an abrupt high-frequency hearing loss superimposed on a flat audiogram, whereas sensory and cochlear conductive presbycusis might emerge as an abrupt high-frequency loss superimposed on a descending puretone audiogram (Schuknecht and Gacek, 1993). Intermediate presbycusis, described by Schuknecht and Gacek (1993), is characterized by the presence of submicroscopic alterations in structures of the cochlea that control cell metabolism, a decrease in the number of synapses on hair cells, and chemical changes in endolymph. Audiograms associated with presbycusis of this variety are primarily flat or mildly descending, without consistent or distinct pathologic correlates. Thus, presbycusis has variable forms of clinical expression and is not necessarily represented by a single pattern. Despite the audiometric patterns often seen clinically, it is impossible to identify the type of presbycusis from the audiogram.

The aging process impacts the central nervous system in general and the central auditory system in particular. Neuronal age-related atrophy is characterized by an overall loss of neurons; a change in neuron size (i.e., shrinkage); a decrease in size of the cell body, nucleus, or nucleolus; and a decrease in dendritic arborization along with a diminution or disappearance of dendrites and a lengthening of dendrites (Shankar, 2010). Additional functional changes in the auditory nervous system include changes in dendritic morphology, alterations in neurotransmitter receptors and in electrophysiological properties, and glycine inhibition and loss of glycine receptors (Shankar, 2010). The frequency of spontaneous excitatory postsynaptic currents is reduced while there is interference with the electrical firing pattern characteristic of neurons involved in information processing (Shankar, 2010). The loss of auditory nerve function with age is evident in the changes in the action potential of the auditory nerve in which the input–output functions of the compound action potential are shallow in older animals as compared to younger animals (Gates and Mills, 2005).

According to Gates and Mills (2005), the asynchronous activity in the auditory nerve associated with aging may derive from a combination of factors including the nature of the synapse between the inner hair cells and individual auditory nerve fibers, primary degeneration of spiral ganglion cells, and a reduced EP. It appears that age-related changes in asynchronous activity of the auditory nerve combined with age-related changes in the central auditory nervous system explain the decline in temporal resolving abilities so prevalent in older adults (Gates and Mills, 2005).

According to functional and neurochemical studies using animal models, age-related changes in neural recovery may be attributable to an imbalance in inhibition and excitation critical for normal cellular function and slow peripheral deafferentation (i.e., incomplete afferent connections) may trigger decrements in inhibitory neurotransmission (Canlon et al., 2010; Caspary, Schatterman and Hughes, 2005; Eddins and Hall, 2010). The reduced amplitude of the action potential recording in aging ears is likely indicative of poorly synchronized neural activity in the auditory nerve which translates into abnormal function in the auditory brainstem as reflected in auditory brainstem studies (Gates and Mills, 2005). Alterations in synaptic processing, decline in inhibitory neurotransmitters such as GABA, and age-related disruptions in temporal processing associated with changes in the auditory nerve and central auditory pathways likely contribute to the speech understanding difficulties in background noise which are the hallmarks of ARHL (Frisina and Walton, 2005).

Using animal models, we now have an improved understanding of the nature of age-related changes in the central auditory nervous system. It appears that the primary aging changes in the dorsal cochlear nucleus are driven by the rapid loss of cochlear input (aka peripherally induced central effects) (Frisina and Walton, 2006). Further, there appear to be disruptions of synapses from ascending auditory nerve fibers in older animals as they make contact with cochlear nucleus neuron, along with a slight decrease in the number of nerve fibers within the lateral lemniscus and the IC (Frisina and Walton, 2006). It seems that there is an age-related downregulation of GABAergic inhibition throughout the auditory central nervous system, which may account for the age-related changes in the strength of central synapses (Caspary et al., 2008). According to Caspary et al. (2008) there is a selective loss of normal adult inhibitory neurotransmission with age which likely contributes to the loss of sensory function typical of older adults. Similarly, there are age-related declines in glycine receptors in the cochlear nucleus

which hampers glycinergic transmission critical to auditory processing (Canlon et al., 2010). Additionally, there are age-related changes in glutamate receptors which affect synaptic transmission in the cochlear nucleus, yet there does not appear to be age-related changes in the GABA receptors (i.e., a primary inhibitory neurotransmitter which decreases the neuron's action potential) in the cochlear nucleus (Canlon et al., 2010). There is a reduction in glycine levels in the cochlear nucleus with increasing age which alters the response properties of cells within the CN (Caspary et al., 2008). Interestingly, SOC studies in animals show age-related changes in potassium channels and calcium binding proteins in cells of origin in the descending pathway from the SOC to the cochlea (Zettel et al., 2007). According to Gleich et al. (2004) the size of glycine and GABA neurons in the high-frequency limb of the lateral superior olive is significantly reduced in older gerbils. The IC shows significant age-related changes in GABA neurotransmission (Caspary, Schatteman and Hughes, 2005). It appears that the degraded and decreased acoustic input associated with age-related changes in the auditory periphery is associated with a selective down regulation of normal adult inhibitory GABAergic function in the IC (Caspary et al., 2008). Notably, there is a decrease in the number of GABA-immunoreactive neurons, a decrease in GABA release, and a decreased concentration of GABA in the IC (Canlon et al., 2010). Further, decreased acoustic input from the auditory periphery is associated with significant changes in GABA neurotransmission in the normal adult IC (Caspary et al., 2008). In rats the IC shows significant age-related changes related to GABA neurotransmission and a loss of GABA-immunoreactive synaptic endings, as well (Turner and Caspary, 2005). The effects of age on the IC include reductions in the number of GABA-immunoreactive neurons, the concentration of GABA, GABA release, and GABA receptor binding (Leventhal et al., 2003). Similarly, there are deficiencies in glutamate function with age (Canlon et al., 2010). Reductions in the latter neurotransmitter have implications for neurotransmitter function in the IC likely affecting auditory processing. Finally, animal studies suggest that aging may be associated with a deficit in neural recovery at the level of the IC. According to Canlon et al. (2010), some of the above changes in the IC are typical of those seen in neural presbycusis and may explain deficits in intensity and temporal coding in older adults.

Brody (1955) was among the first investigators to identify age-related changes in the temporal lobe of the aging brain. Using a limited number of brains, Brody (1955) found that the magnitude of cell loss was greatest in the superior temporal gyrus. Notably, there was an almost one-to-one correlation between age and cell loss. He also noted a decrease in the thickness of the superior temporal gyrus with increasing age that was not apparent in other cortical regions. Subsequently, Scheibel et al. (1975) studied the superior temporal cortex and noted a loss of dendrites and cell death in older patients. According to animal studies, auditory deprivation from the periphery disrupts the tonotopic organization of the central auditory nervous system. Recent research confirms that the primary auditory cortex undergoes age-related plastic changes, similar to that observed at lower levels of the auditory pathway (Caspary et al., 2008). Age-related changes in the GABA enzyme levels have been found in the primary auditory cortex of rats and it is likely that in humans a loss of normal GABA transmission contributes to difficulty in temporal coding (Canlon et al., 2010). Age-related changes in the brain, including the prefrontal cortex, have profound implications for the communication challenges confronting older adults.

GERIATRIC SYNDROMES ASSOCIATED WITH HEARING LOSS IN OLDER ADULTS

In addition to age-related degeneration, a number of other factors explain the hearing loss experienced by older adults. These include excessive exposure to occupational or recreational noise, genetic factors, acoustic neuroma, trauma, metabolic disease such as diabetes, vascular disease, infections, autoimmune disease, and drug exposure. Because of polypharmacy in older adults, adverse reactions from ototoxic agents may result from drug–disease interactions or drug–drug interactions. The cochlea is especially susceptible to ototoxicity because medication is retained for a longer period and in a higher concentration in the inner ear fluids than in any other body tissue or fluid, especially in individuals with liver or kidney dysfunction. Older adults are at particular risk for aminoglycoside ototoxicity when they have an existing loss of auditory function associated with the aging process (Weinstein, 2013).

Cardiovascular disease (CVD) is widespread among older adults. Cardiovascular insufficiency compromises the blood supply to organs throughout the body. Helzner et al. (2005) found that individuals with CVD had a 56% higher risk of hearing loss. Low-frequency thresholds appear to be more closely correlated to CVD than high-frequency thresholds, suggesting a possible vascular or metabolic link (Gates et al., 1993; Helzner et al., 2005). Helzner et al. (2011) noted that such risk factors as higher levels of triglycerides and history of smoking in men, higher body mass index in women, and higher resting heart rate in men and women were related to poorer hearing. The likelihood of hearing loss and its progression is higher in older adults with a diagnosis of diabetes than in those without the diagnosis (Mitchell et al., 2009). Another condition rising in prevalence among older adults is cognitive decline and data on its association with hearing impairment are compelling.

Cognitive impairment is associated with lower hearing levels and with faster declines in peripheral hearing sensitivity (Kiely et al., 2012; Lin, 2011). Lin et al. (2013) found that persons with hearing loss at baseline demonstrate more dramatic decline in cognitive status than did

individuals with normal hearing. Further, over the 6-year follow-up period, individuals with hearing loss were initially at increased risk of developing cognitive impairment than were those with normal hearing, with persons with more significant hearing losses at greatest risk.

Depression is among the most prevalent affective disorders in older adults. MacDonald (2011) explored the relationship between hearing loss and depression in a sample of adults 65 years of age and older. Scores on the Hearing Handicap Inventory for the Elderly were significantly associated with scores on the depression scale on the Center for Epidemiological Studies-Depression (CES-D) Scale. Interestingly, the actual hearing loss scores accounted for a smaller proportion of the variance on the CES-D. Similarly, in a sample of older adults from Japan, Saito et al. (2010) found that scores on the HHIE-S were an independent predictor of scores on the Geriatric Depression Scale (GDS). Gopinath et al. (2012) reported that having a measured hearing impairment at baseline dramatically increased the likelihood of developing a self-reported hearing handicap over time (Weinstein, 1986). Furthermore, after adjusting for age, and other factors, older adults with a self-perceived hearing handicap had a greater chance of reporting depressive symptoms. Similarly, after adjusting for age, smoking, stroke, visual impairment, and walking disability as compared to persons without a self-reported hearing handicap, those with significant self-reported hearing handicap on the HHIE-S had significantly lower mean physical component and mental composite scores on the SF-36, a scale that quantifies dimensions of health and well-being.

Zuniga et al. (2012) explored the relation between hearing level and saccular, utricular, and semicircular canal function in a sample of older adults 70 years of age and older. High-frequency hearing loss was associated with reduced saccular function, yet not with utricular or semicircular canal function. Age and noise exposure were significantly associated with cochlear and saccular dysfunction. This finding may explain, in part, why older people with chronic dizziness or imbalance are two to three times more likely to fall in comparison with older people who do not experience these problems. Karpa et al. (2010) underscored the importance of the links between hearing loss and functional decline, physical decline, cognitive impairment, low self-rated health, and mortality. They reasoned that the association between hearing impairment and difficulty in walking may be attributable to fear of falling, impaired balance associated with decreased vestibular function in older adults, and/or decline in physical and social activity associated with hearing loss. Furthermore, hearing impairment may increase the odds of cardiovascular death perhaps because these individuals are socially isolated, do not visit their doctors regularly, and are less inclined to take preventive steps such as exercise and diet. Interestingly, a recent report using data from the 2005 to 2006 and 2009 to 2010 NHANES revealed that in fact, for adults age 70 years and over, hearing loss is independently associated with hospitalizations and poorer self-rated health (Genther, Frick, Betz et al., 2013). They too speculated that pathways through which hearing loss may increase the burden of disease may be through the relationship with social isolation and cognitive decline.

In conclusion, age-related changes within the auditory system are associated with many geriatric syndromes. The connections are linked either directly or indirectly to reduced independence, reductions in quality of life, increased mortality risk, social isolation, and disability in walking which are notable in selected individuals with hearing loss. Despite the fact that hearing loss is a chronic problem and is a leading cause of disease burden, it continues to go unrecognized and untreated in the majority of older adults.

AUDIOLOGIC FINDINGS

Puretone Thresholds

Age-related changes throughout the peripheral and central auditory systems are associated with decrements in hearing for puretones, speech understanding, and deficits in cognitive processing. Age and frequency effects emerge in cross-sectional and longitudinal studies of hearing loss with differences in prevalence estimates that may be associated with differing definitions of hearing impairment (e.g., three-frequency vs. four-frequency puretone average, 15 or 25 dB HL as cutoff) and lack of consistency in use of better or poorer ear to define hearing status. Despite the latter, there is general agreement regarding configuration and frequency specificity of hearing loss. Recent population-based studies on hearing loss in community-based older adults confirm that age-related hearing has several distinct features. Air- conduction thresholds became poorer with increasing frequency and puretone hearing sensitivity tends to decline with increasing age, with the greatest loss in the frequencies above 1,000 Hz. Further, the hearing loss tends to be bilateral, symmetrical, and sensory/neural in origin. The decline in high-frequency sensitivity appears to be greater in males, whereas the decline in low-frequency thresholds tends to be greatest in females of comparable age. Hearing loss configuration in the higher frequency tends to be sharply sloping in males and gradually sloping in females (Gordon-Salant, 2005). The average hearing loss in older males can be described as mild to moderately severe, bilateral, and sensory/neural with a sharply sloping configuration. Older women tend to present with a mild to moderate, gradually sloping, bilateral symmetrical, sensory/neural hearing loss. Among residents of nursing facilities, the sensory/neural hearing loss tends to be more severe than that of community-based individuals, in large part because of the older age of residents (Weinstein, 2013).

In addition to gender, race influences hearing status of older adults. (Lin, Thorpe, Gordon-Salant, Ferrucci, 2011) reported that the black race may be a protective factor

against developing hearing loss in that prevalence of hearing loss among black women and men over 70 years of age is dramatically lower (i.e., 45%) than in white (67%) participants. Prevalence of hearing loss is slightly higher among black men (43%) than among black women (40%), with white males having the highest prevalence (72%). Notable is that whites have a 63% greater likelihood than blacks of having a hearing loss. Finally, genetic factors have an impact on puretone hearing levels, as well. McMahon et al. (2008) explored the role of family history in hearing loss. Prevalence of hearing loss in their sample of 2,669 adults was higher in men (39%) than in women (29%). The majority (68%) of participants had mild hearing loss. Forty-seven percent of participants reported a family history of hearing loss. Most notable was that the majority (63%) of people reporting a family history of hearing loss were female. Severity of hearing loss was linked to familial history with 64% of participants who had moderate to severe hearing loss reporting a positive family history, as compared to 53% with mild hearing loss and 45% without hearing loss. Among those with moderate hearing loss, family history was on the mother's side. In addition, siblings in this cohort were more likely to have a hearing loss. This work suggests a strong association between presbycusis and family history with the relationship greater among females and their mothers than that found in males.

Speech Understanding

Older adults have more difficulty understanding speech in noisy environments, when people speak quickly, when the speaker has a foreign accent, in reverberant conditions, when there are multiple talkers, when the message is complex, and when there is reduced contextual information. Loss of peripheral hearing sensitivity, decline in cognitive abilities, age-related changes within the eighth nerve, auditory brainstem pathways, and auditory cortex are hallmarks of ARHL. Additionally, lack of neural inhibition and decrease in excitatory synchronization translate into degradation of the neural code throughout the central auditory nervous system (Tun et al., 2012).

There are large individual differences in speech understanding among those over 60 years of age with multiple probable etiologies. Results of a systematic review recently completed by Humes et al. (2012) shed additional light on the several hypotheses which have been suggested to explain the mechanisms underlying the central auditory processing (CAP) problems experienced by older adults, including the peripheral hypothesis, the central auditory hypothesis, and the cognitive hypothesis. In the peripheral hypothesis, the auditory periphery is implicated; in the central auditory hypothesis, the auditory portion of the central nervous system from the cochlear nucleus to the primary auditory cortex is implicated; and in the cognitive hypothesis, age-related changes in cognitive processing resources such as working memory, attention, and executive function appear to underlie the speech processing difficulties many older adults experience (Humes et al., 2012; Tun et al., 2012). It is abundantly clear that the CAP problems typical of persons with ARHL cannot be isolated nor can they be attributable solely to one of the above mechanisms. A brief overview of each is included below.

Peripheral (e.g., cochlear changes) hypothesis. This hypothesis holds that speech recognition difficulties are attributable to individual differences in the encoding of sound by the outer ear through the inner ear and eighth nerve (Humes et al., 2012). The peripheral component is reflected in the frequency-specific sensitivity loss revealed by the audiogram, most notable in the high frequencies. The peripheral hypothesis has been further subdivided into two versions. One version suggests that simple changes in audibility, in which sound energy falls below an individual's audible region, account for the speech understanding problems characterizing older adults. The other version suggests that reduced physiological processing associated with age-related changes in the cochlea creates distortions beyond loss of hearing sensitivity. Sources of distortion may be due to changes in peripheral encoding mechanisms including loss of spectral and temporal resolution and loss of intensity discrimination. Decreased frequency selectivity and reduced intensity discrimination are manifest by increased frequency difference limens, poor intensity resolution, and increased intensity difference limens.

Central auditory hypothesis. This hypothesis implicates age-related changes in auditory structures in the central nervous auditory system including the IC (Humes et al., 2012). There are two possible explanations for the central auditory hypothesis including the more direct, namely the central effect of biological aging (CEBA), or the indirect mechanism, namely the central effect of peripheral pathology (CEPP). In the case of the former, peripheral hearing is normal and the speech communication deficits are attributable to deficits in the central auditory mechanism from the cochlear nucleus through the auditory pathways (Humes et al., 2012). In contrast, in the case of CEPP, the speech understanding deficit is due to the fact that central auditory changes may be induced by the peripheral pathology (Humes et al., 2012). Speech-based measures are typically used to assess central auditory function; however, performance is undoubtedly influenced by cognitive function.

Cognitive hypothesis. This hypothesis implicates higher centers in the auditory pathways as a source of individual variations in cognitive abilities and declines in cognitive performance. Cortical functions subsumed under these areas include information processing, storage, and retrieval. These cortical processes underlie performance on speech understanding tasks, and it follows that individual

differences in speech understanding performance may be attributable to deficits in one or more of these areas. It is noteworthy that cognitive deficits are not confined to the auditory modality. Indeed, short-term memory deficits may emerge on tasks involving both auditory and visual presentations of stimuli. The most notable changes in cognitive performance that influence speech understanding include attentional deficits, age-related reductions in the speed of perceptual and mental processing, and deficits in working memory and in executive function. Executive control processes that include such concepts as inhibition, working memory, and attentional capacity are responsible for the ability to plan, assemble, coordinate, and sequence operations integral to speech understanding which is a highly complex task. According to Kalluri and Humes (2012) given the role of cognitive processing in auditory function, audiologist should consider using dichotic tests, for example, to screen for central/cognitive function as decisions regarding choice of technologies recommended to older adults (e.g., hearing aids, FM systems) must be informed by these changes.

The information above indicates that many older adults experience significant difficulty understanding speech in less than optimal listening situations and the etiology is likely multifactorial. That is, peripheral, central, and cognitive factors likely interact in a variety of ways to explain the auditory processing difficulties which are characteristic of ARHL. The large individual differences in processing problems underscore the importance of screening cognitive function, assessing speech understanding in noise or reverberant conditions, and using results from targeted testing to inform recommendations which in many cases must go beyond traditional hearing aids. In addition to incorporating objective tests which are influenced by peripheral, central, and cognitive factors, self-report measures of communication function should be included as responses reflect the quality of life implications of speech processing deficits. Similarly, given the fact that lifelong experiences of playing music and software-based brain fitness programs tend to have a positive effect on speech understanding in noise, audiologists should inquire about these experiences and should discuss the potential beneficial effects of cognitive and perceptual training. Finally, audiologists should work with older adults with ARHL to assist them in learning how to use top-down processing skills to supplement deficits in bottom-up processing of the auditory signal (Pichora-Fuller and Levitt, 2012).

PSYCHOSOCIAL CONSEQUENCES OF HEARING IMPAIRMENT

Literature that has emerged over the past 10 years has demonstrated conclusively that untreated hearing loss has detrimental effects on psychosocial well-being, communication, affect, cognitive status, and functional health status. In fact, the myth that hearing loss is harmless has been debunked, and it is becoming increasingly clear that, if untreated, hearing loss can be costly to the individual in terms of relations with family members, social engagement, mortality, and productivity at work. The increased listening effort and fatigue associated with communicating in noisy and reverberant rooms contributes to the burden of hearing loss and the desire on the part of older adults with ARHL to gradually withdraw from social activities. When hearing loss goes undetected, the burden on the individual and society is enormous. ARHL is associated with perceived social isolation, which in turn is a predictor of adverse physical and mental health outcomes (Cornwell and Waite, 2009; Weinstein and Ventry, 1982). It is notable that persons with more chronic conditions become more functionally impaired sooner than do persons with fewer chronic conditions.

To further understand its adverse effects, it is important to understand that hearing loss and attendant communication deficits occur in the context of other geriatric syndromes including cognitive impairment, falls, and depression, contributing to disability which is a growing public health concern (Rosso et al., 2013). To explain, disability is defined as difficulty in performing activities of daily living such as dressing or eating, because of an existing physical or mental health limitation. Interestingly, according to results from the Women's Health Initiative, an observational study of community-based women over 65 years of age, 75% of the participants with five or more geriatric syndromes at baseline suffered from dizziness, hearing impairment, visual impairment, or urinary incontinence (Rosso et al., 2013). Women with five or more geriatric syndromes were six times more likely to develop incident disability than were those with no geriatric syndromes at baseline. The fact that presence of geriatric syndromes such as hearing loss is predictive of developing disabilities is significant in that the negative effects of hearing loss are potentially preventable with early identification and targeted interventions.

Dalton et al. (2003) conducted a population-based study of the relationship between hearing impairment and selected quality of life variables in a large sample of adults between the ages of 53 and 97 years old. More than half of the subjects had a hearing impairment, which was mild in 28% and moderate to severe in 24% of subjects. The quality of life indicators associated with hearing loss were social functioning, mental health, and physical functional status. The adverse effects of untreated hearing impairment appear to be a global phenomenon. Wu et al. (2004) evaluated the psychosocial consequences of self-perceived handicap in a sample of 63 older adults ranging in age from 62 to 90 years attending a geriatric medicine clinic in Singapore. In their study of subjects with self-reported hearing difficulty and a failed puretone screening, 70% of respondents indicated that

they would be happier if their hearing were normal, 40% indicated that difficulty hearing made them feel frustrated, and 43% admitted to feeling sad because of their hearing handicap. Interestingly, the federal government in Australia is designing a comprehensive approach to managing ARHL because "as a cause of burden of disease, hearing impairment is the second highest disability for every Australian man" (Smith et al., 2005, p. 2).

Gerontologists have long studied social isolation and have identified a number of indicators, all associated with poorer health. The link between hearing impairment and subjective social isolation is typically ignored by gerontologists but given its link to depression and cognitive impairment it is likely that hearing loss may explain some of the variability across indicators of social isolation which include having a small social network, low participation in social activities, a perceived lack of social support, and feelings of loneliness (Cornwell and Waite, 2009). It is of interest that older adults who perceive high levels of social support tend to have better coping strategies, greater self-esteem, and sense of control, whereas those who are socially disconnected tend to have poorer physical and mental health outcomes (Cornwell and Waite, 2009). The costs of social disconnectedness to society, the individual, and family members are high. Audiologists could potentially play a role in reducing the burden of selected conditions such as cognitive decline and social isolation if we focus research and clinical interventions on demonstrating that use of hearing assistive technologies including hearing aids is effective treatment for older adults with age-related hearing difficulties who value social engagement and connectedness.

To summarize, poor hearing is associated with perceived difficulties across a wide variety of activities that relate to managing everyday life. ARHL is predictive of increased functional, physical, and psychosocial impairments, as well as poorer health-related quality of life. There is a high degree of individual variability in the reactions of older adults to hearing loss, ranging from complete acceptance and positive personal adjustment to feelings of displacement, anger, and withdrawal. For this reason, it is important to qualitatively and quantitatively assess each patient to determine how hearing impairment affects them, their activity level, and their relations, with friends and family.

A DIAGNOSTIC AND MANAGEMENT PROTOCOL

It is clear from research and clinical experience that older adults require a diagnostic and management protocol which is client centered and unique to their needs. The protocol proposed below is based on several premises taken from the geriatric literature on patient-centered care, coupled with the American Academy of Audiology Guideline for the Audiologic Management of Adult Hearing Impairment.

Premise 1. The objectives of the initial audiologic assessment with an elderly client should be to (1) understand the client's experience with hearing loss (aka the patient journey), that is, the communication difficulties the individual is having from their perspective and that of a communication partner; (2) gain an understanding of the hearing status and speech understanding under a variety of conditions and at differing signal-to-noise ratios (SNRs) using reliable and valid measures; (3) understand the client's stage of readiness and motivation to embrace some form of targeted intervention; and finally, (4) determine candidacy for the most appropriate hearing solution(s), be it personal sound amplifiers, hearing aids, hearing assistance technologies (HATs), speech communication training, and/or some form of counseling. Eliciting and understanding the patient's perspective or narrative—concerns, ideas, expectations, illness experience, needs, feelings, and functioning—is key to the assessment process (Epstein et al., 2005).

Premise 2. Clinical measures of hearing are inadequate predictors of the difficulties older adults face in carrying out routine activities in their natural environments, accordingly routine assessment protocols are limited in their ability to enable audiologists to understand the complex auditory processing difficulties that are hallmarks of ARHL. Stated differently, audiologists must incorporate measures into the test battery which tap into age-related declines in auditory temporal and binaural processing and aspects of cognitive processing including working memory and speed of processing. Objective tests should be designed to uncover the listening difficulties the individual is experiencing, such as difficulty in the presence of noise, and reverberant conditions and to determine listening strategies being used (e.g., does the individual take advantage of visual cues when communicating?). Self-report data should be incorporated and used as an adjunct to objective and multifactorial speech testing as responses which often reflect personality variables are predictive of candidacy for intervention and outcomes from intervention. Finally, the importance of a test battery approach cannot be overemphasized, especially since many older adults will have difficulty understanding speech in group situations, in noise, and in reverberant conditions despite relatively mild hearing loss.

Premise 3. We should rethink our conceptualization of interventions for the hearing impaired and insure that the "patient experience" drives our recommendations. We must keep in mind that cognitive function will influence choice of intervention and outcomes and we must remain cognizant of the interaction between peripheral sensory

function and cognition when designing intervention strategies. We can best serve the needs of our older clients by providing customized solutions to their various communication challenges. Using decision aids which present the many options available will engage the patient as a partner and can ensure that they are made aware of the numerous approaches to improving the communication difficulties and outcomes associated with options presented. Technologies which include personal sound amplifiers, hearing aids, remote microphone technology, situation-specific HAT, and/or computer-based auditory training to improve auditory, cognitive, and speech processing are some of the many options available. The philosophy behind the choice of intervention should be our desire to "foster the effective and efficient trading of bottom-up processing based on the signal and top-down processing based on knowledge" (Pichora-Fuller and Levitt, 2012, p. 355). Important intervention outcomes to be cited should include independence, safety, improved quality of life, stress-free and natural listening, improved relations, and reduced burden of illness. We should insure that every person who walks into our office leaves with some form of solution to the communication challenges being voiced especially when purchase of hearing aids has been postponed. To reiterate, solutions can range from listing and reviewing necessary communication strategies, advice on hearing protection to promote hearing health, use of mobile apps on smartphones, and discussions about the value of personal sound amplifier products (PSAPs) for selected uses to some form of computer-based auditory and speech comprehension training. Using the principles listed above, the audiologic evaluation for older adults should include the intake and the evaluation. More information about counseling regarding recommendations and follow-up can be found on the Point at http://thepoint.lww.com.

Step 1: The Intake and Needs Assessment

The purpose of the intake is to obtain a comprehensive history that encompasses medical and nonmedical aspects of the hearing loss. To best promote quality of care, the traditional case history should focus on the etiology and evolution of the hearing loss and geriatric syndromes that may be relevant especially those typically associated with hearing loss including diabetes, kidney disease, and CVD. Smoking history, family history, hearing aid use history, and history of noise exposure are important considerations, as well. A multifaceted needs assessment which provides a feel for the patient journey and the impact of the hearing loss on the individual and the family is an integral part of the intake. During the initial encounter keep in mind what is important: What does it mean to this person to have this impairment at this time in his or her life, with spouse and children, in the person's environment, and peer group? Table 34.1 lists some questionnaires to consider for a communication needs assessment. Responses will inform testing protocols and can serve as the basis for counseling and decision-making regarding next steps in the process including treatment options. Further as Tun et al. (2012) suggest, self-report measures yield information about lifestyle and quality of life, and at times you can get at ease of listening which is so relevant especially when communication partners are concerned. Table 34.2 includes a very recent modification of the Hearing Handicap Inventory, namely the Hearing Health Care Intervention Readiness Questionnaire (HHCIR), which is currently undergoing reliability and validity testing. This questionnaire includes questions on social engagement and readiness plus questions about activity limitations/participation restrictions. Given the link between social engagement and cognitive function and social isolation and hearing loss it is important to ask directly about the issue of social connectedness. People who consider themselves to be lonely and isolated are at risk for disability and should be referred to their physician and of course recommendations for hearing assistance if appropriate. Finally, at the intake, audiologists might consider routinely administering a screening form for mental status such as the Montreal Cognitive Assessment (MoCA) or Mini-Mental Status Evaluation (MMSE), for depression using the Patient Health Questionnaire (PHQ), and for vision using the Snellen Visual Acuity Eye Chart.

Information from these screening instruments is important given the interplay among hearing impairment, cognitive function, depression, and visual status. At times referral to a geriatric specialist or primary care physician may be appropriate as partnering with these professionals

TABLE 34.1

Communication Needs Assessment–Selected Instruments

1. Hearing Handicap Inventory (HHI) and spousal version
2. Client-Oriented Scale of Improvement (COSI)
3. Abbreviated Profile of Hearing Aid Benefit (APHAB)
4. International Outcomes Inventory for Hearing Aids (IOI-HA) and version for significant others
5. Speech, Spatial, and Qualities of Hearing Scale (SSQ)
6. Attitude Toward Loss of Hearing Questionnaire (HARQ)
7. SOFI (Screening for Otologic Functional Impairments)
8. HHCIR (Hearing Health Care Intervention Readiness) Questionnaire

TABLE 34.2

Hearing Health Care Intervention Readiness Questionnaire[a]

Hearing Health Care Intervention Readiness (HHCIR)

© **Barbara Weinstein, 2012**

Instructions: The purpose of this questionnaire is to identify any problems you are having communicating with others and to help determine your readiness to pursue a hearing healthcare intervention. Please circle either *the most appropriate response* to each question. If you hearing aids, please answer the way you hear when using the hearing aids, if you are not experiencing any hearing difficulties, please mark *NA* for each item.

Item		4	2	0	NA
H-1	Does a hearing problem cause you difficulty when listening to the television or to the radio?	Yes	Sometimes	No	
R-I	How important is it for you to have less difficulty when listening to the television or radio?	Very	Somewhat	Not very	
H-2	Does a hearing problem cause you difficulty when visiting with friends, relatives, or neighbors?	Yes	Sometimes	No	
R-2	How important is it for you to experience less difficulty when visiting with friends, relatives, or neighbors?	Very	Somewhat	Not very	
H-3	Does a hearing problem interfere with your ability to hear environmental sounds such as the telephone ringing or car horns honking?	Yes	Sometimes	No	
H-4	Does a hearing problem cause you to feel frustrated when communicating with friends, coworkers, or members of your family?	Yes	Sometimes	No	
R-3	How important is it for you to feel less frustrated when communicating with friends, coworkers, or members of your family?	Very	Somewhat	Not very	
SI-1	Do you experience feelings of loneliness or not belonging due to your hearing loss?	Yes	Sometimes	No	
SI-2	Do you perceive yourself to be isolated from friends and/or family members due to your hearing loss?	Yes	Sometimes	No	
SE-1	How confident are you that you would follow the recommendations of a hearing healthcare professional (e.g., undergo a hearing evaluation, use hearing aids, use a hearing assistance technology, participate in a communication program)?	Very	Somewhat	Not very	

[a]Montano, Preminger, Chisolm collaborated on early stages of development. © Barbara Weinstein, 2012.

can help achieve desired health outcomes. Our value as a doctoring profession by serving as an important partner in the healthcare maze may be elevated if physicians see how treatment of hearing loss may reduce some of the burden associated with treating persons with multimorbidity including reducing possible disability and some of the high cost of medical care associated with the prolonged life of older adults.

Step 2: The Evaluation

The purpose of the evaluation is to determine hearing status including type and severity of hearing loss and speech understanding/auditory processing ability in a variety of listening situations using material which are ecologically valid (e.g., sentence materials). Puretone air- and bone-conduction testing across octave and interoctave frequencies is the first part of the evaluation. Next, it is important to assess speech understanding ability using valid real-life listening materials, presentation levels, and situations. Given the individual variability inherent across persons with ARHL, speech understanding should be evaluated using open-set sentence materials presented under different conditions such as (1) with and without visual cues; (2) with and without competing noise at various SNRs; and (3) using degraded speech to uncover the temporal processing declines which are characteristic of ARHL (Tun et al., 2012). Adequate time at the end of the testing should remain during the session to discuss patient preferences relative to their journey and the

results and of course intervention options using decision aids.

HEARING SCREENING

Hearing screening has been a health promotion activity engaged in by audiologists to promote early identification of hearing loss and intervention with hearing aids. Historically, compliance with traditional screening programs conducted by audiologists has been quite low, because only a small proportion of individuals undergoing hearing screenings actually follow through with the recommendations to undergo hearing tests and then consider purchasing a hearing instrument. With the benefit of a series of epidemiologic studies we now know that the most successful screening programs are those which target and identify individuals at risk, who are interested in and motivated to change their behavior. These are the individuals who are most likely to benefit from intervention. Hearing screening is worthwhile if it leads to relief of distress or to improvement in the functions of daily living, and if it is highly acceptable to patients this improves compliance with therapeutic recommendations (Mitchell and Coyne, 2010).

Because hearing loss accompanies many geriatric syndromes and management of hearing loss could potentially reduce disability, primary care physicians, potentially the future gatekeepers of hearing health care for Medicare beneficiaries, should be involved in screening older adults for hearing difficulties. To explain, the primary care physician has considerable influence over their patient's actions pertaining to health matters. If a physician recommends a hearing test and possible treatment, the likelihood that the patient will comply is typically higher than when the referral is initiated by an audiologist. Screening of older adults with multimorbidity may be acceptable to physicians if they understand that uncovering and treating hearing loss may reduce some of the burden of geriatric syndromes which are associated with disability, mortality, increased hospitalizations, and social isolation. If physicians do not routinely engage in preventive activities, then they should consider a hearing screening under the following conditions: (1) If a family member reports a concern about hearing/understanding; (2) if selected chronic conditions place the patient at risk for hearing loss; (3) if the patient takes ototoxic medications; or (4) if the patient smokes or has a history of noise exposure. If the patient is known to be depressed or to have a cognitive impairment, it would behoove the physician to conduct a hearing screening, as it may be that untreated hearing loss is a contributing factor (Weinstein, 2013).

The target population for hearing screening programs should be individuals at risk for the health condition likely to benefit in terms of projected life-span, self-efficacy, outcome expectations, and cost–benefit considerations. For hearing health promotion activities to be successful they should be integrated and coordinated across providers and settings, and protocols should be functional in scope rather than disease based, including tailored and multicomponent interventions. Additional keys to success include a protocol that is brief, is easy to administer, is acceptable to both the healthcare provider and the patient, accurately discriminates across varying levels of function, and includes follow-up mechanisms and community resources that are in place to handle referrals and monitor adherence. One final key ingredient of successful programs is distribution of educational materials (Weinstein, 2013). Patient education materials are important because studies suggest a relationship between health literacy and rates of chronic health conditions. In short, individuals with inadequate health literacy have significantly higher rates of certain chronic conditions including arthritis and hypertension as compared with those with adequate literacy (Wolf, Gazmararian, and Baker, 2005). Hearing screening is a good target condition for screening as individuals are living longer and will have hearing loss as they live out their lives.

The physician screening program should be multicomponent including screening for hearing impairment and an otoscopic examination because of the high prevalence of impacted cerumen in older adults. The Welch Allyn Audioscope™, a handheld otoscope with a built-in audiometer, is well accepted as a reliable and valid alternative to a portable screening audiometer (Yueh et al., 2010). A self-report questionnaire, such as the Hearing Handicap Inventory, which is reliable and valid should be included as well, as scores are predictive of candidacy for and outcomes with intervention. Physicians conducting hearing screens should target older adults with multimorbidity. Multimorbidity complicates care and is associated with adverse consequences, including disability, high symptom burden, poorer quality of life, and higher rates of adverse effects of treatment or interventions (Tinetti et al., 2012). Finally, targeted referrals based on consideration of the patient's life expectancy and medical status are key elements. Therefore, in the case of a patient with compromised health status because of multiple comorbidities and a shortened life expectancy who failed the puretone screening, the physician might recommend communication strategies and use of a small personal amplifier such as the pocket-talker in small groups and when watching television. Use of this system will insure that the patient continues to communicate with family members and their physicians and importantly remains connected. In contrast, a person in good health with a life expectancy in excess of 10 years might be referred to an audiologist for a hearing test and consideration of hearing aids (Weinstein, 2013). Audiologists are essential to the success of physician screening initiatives, and part of their role is providing the physician with literature to be distributed to the patient that will promote a patient–clinician partnership, which is

critical to successful outcomes. The physician must know how to recognize patients who are hearing impaired, how to communicate with the hearing impaired, how to discuss options that are appropriate, and how to give advice that will guide the patient's actions. The physician must tell older patients that age is not a limiting factor for the hearing impaired to benefit from hearing aids and available HATs (Weinstein, 2013). It is incumbent on audiologists to educate doctors regarding the improvements in quality of care and life which could result from management of a geriatric syndrome, such as hearing loss, the effects of which are treatable with a variety of nonmedical interventions. Another point to emphasize is that older adults underreport or fail to report hearing deficits, and that hearing deficits are one of the few geriatric syndromes missed during the traditional medical exam (Weinstein, 2013).

It is important to underscore that the goal of a hearing screening is to identify those persons with hearing impairment who will pursue and benefit from intervention as hearing screening is only cost effective when compliance is high and outcomes are tangible and beneficial. It is important to emphasize that physicians are likely to screen for hearing loss if they are made aware that untreated hearing impairment can be detrimental placing some older adults at risk for disability which in the long term is costly to the individual and society. It is also imperative that physicians understand that the ability of the patient to understand during physician encounters is vital to achieving patient-centered care, which is a guiding principle of care of older adults with multiple geriatric syndromes (American Geriatrics Society (AGS) Expert Panel, 2012). In fact, an important conclusion from the AGS Expert Panel on the Care of Older Adults with Multimorbidity is as follows: "inadequate communication skills and educational materials are also barriers to the care of older adults with multimorbidity. Because conversations about prognosis and preferences can often be difficult for clinicians, training of all healthcare team members must address communication skills" (AGS, 2012, p. 20). Finally, audiologists should be aware of the isolating effects of hearing impairment given the link between social disconnectedness, mortality, and morbidity.

FUTURE TRENDS

Audiologists are well versed in all things related to hearing and are strong proponents for hearing aids which work for many but are not embraced by the majority of persons with communication difficulties despite the sophisticated signal processing available today. In my view, audiologists should adopt some of the guiding principles of the AGS which emphasize on patient-centered care, in which the healthcare professional elicits and incorporates the preferences of their patient into decision-making for older adults with multiple chronic conditions (AGS, 2012). Once the patient is informed of the benefits and harms of available treatment options using decision aids or decision trees, the patient's input and their preferences are given considerable weight. Importantly, clinical management decisions are framed within a broader context which includes life expectancy, likelihood of benefitting, functional status, resources, and quality of life (AGS, 2012). Perhaps, this change in philosophy will enable audiologists to truly partner with persons with hearing loss and healthcare professionals to help achieve mutually agreed upon outcomes.

SUMMARY

The aging of the Baby Boomer generation and increased longevity brings with it challenges to which audiologists should be armed to respond, namely a huge increase in the number of older adults in general and those with auditory processing and communication challenges in particular. These individuals will need and want to hear and understand family, friends, coworkers, physicians, and of course when at work or engaged in leisure time activities. In fact social engagement and ease of communication remain priorities in the golden years of one's life. Adults with ARHL must be encouraged to seek out audiology services early so that hearing loss does not interfere with the quality of their prolonged life. My goal in developing this chapter was to arm audiologists with information about hearing loss in older adults that will empower them to effectively identify, evaluate, and manage this growing and important population. Hearing healthcare services for older adults must be delivered with an understanding of the aging process and the biases older adults bring to their health care. A better understanding of aging in general and its impact on the hearing mechanism in particular will hopefully promote the delivery of patient-centered audiology combined with targeted interventions. Availability of HATs, digital hearing aids, cochlear implants, software to promote auditory and cognitive training, PSAPs, and a vast array of strategies to foster improved communication means that persons with hearing loss should leave the office of the audiologist armed with toolkits that will enable them to communicate more effectively.

FOOD FOR THOUGHT

1. Greater hearing loss is independently associated with self-reported falls such that a 25-dB hearing loss is associated with a nearly 3-fold increased odds of reporting a falls (Lin & Ferrucci, 2012). Is the relationship between hearing loss, mobility limitations, and self reported falls evidence enough to convince physicians to routinely screen hearing in older adults who have a history of falls?

2. Given the link between hearing loss and incident dementia, namely that cognitive impairment is associated with lower hearing levels and with faster declines in peripheral hearing sensitivity; should audiologists routinely administer a reliable and valid cognitive screening test (e.g, The Montreal Cognitive Assessment, MoCA) to older adults and make the appropriate referrals when indicated.
3. Hearing loss is independently associated with increased hospitalizations and poorer self-rated health. Does an untreated or unrecognized hearing loss affect transitions in care and adherence with physician recommendations? If so, what role could audiologists play if they served on the patient centered medical home (PCMH) which is a newly emerging team based health care deliver model?

KEY REFERENCES

thePoint A full list of references for this chapter can be found at http://thePoint.lww.com. Below are the key references for this chapter.

American Geriatrics Society. (2012) Guiding principles for the care of older adults with multimorbidity: an approach for clinicians. Available online at: http://www.americangeriatrics.org/files/documents/MCC.principles.pdf. Retrieved April 16, 2014.

Bainbridge K, Hoffman H, Cowie C. (2008) Diabetes and hearing impairment in the United States: Audiometric evidence from the National Health and Nutrition Examination Survey, 1999 to 2004. *Ann Intern Med*. 149, 1–10.

Brody H. (1955) Organization of the cerebral cortex: III. A study of aging in the human cerebral cortex. *J Compar Neurol.* 102, 511–556.

Cacioppo J, Hawkley L. (2009) Perceived social isolation and cognition. *Trends Cogn Sci.* 13, 447–454.

Cacioppo J, Hawkley L, Thisted R. (2010) Perceived social isolation makes me sad: a 5-year cross-lagged analyses of loneliness and depressive symptomatology in the Chicago Health, Aging, and Social Relations Study. *Psychol Aging.* 25, 453–463.

Canlon B, Illing R, Walton J. (2010) Cell biology and physiology of the aging central auditory pathway. In: Gordon-Salant S, Frisina R, Fay R, Popper A, et al. eds. *The Aging Auditory System. Springer Handbook of Auditory Research.* Vol 34. New York: Springer.

Caspary D, Ling L, Turner J, Hughes L. (2008) Inhibitory neurotransmission, plasticity and aging in the mammalian central auditory system. *The J Exp Bio.* 211, 1781–1791.

Caspary D, Schatterman T, Hughes L. (2005) Age-related changes in the inhibitory response properties of dorsal cochlear nucleus output neurons: Role of inhibitory inputs. *The J Neurosci.* 25, 10952–10959.

Clinard C, Tremblay K, Krishnan A. (2010) Aging alters the perception and physiological representation of frequency: evidence from human frequency-following response recordings. *Hear Res.* 264, 48–55.

Cornwell E, Waite L. (2009) Social disconnectedness, perceived isolation, and health among older adults. *J Health Soc Behav.* 50, 31–48. Available online at: http://hsb.sagepub.com/content/50/1/31.full.pdf+htmlr. Retrieved July 18, 2013.

Crews J, Campbell V. (2004) Vision impairment and hearing loss among community- dwelling older Americans: Implications for health and functioning. *Am J Public Health.* 94, 823–829.

Dalton D, Cruickshanks K, Klein B, Klein R, Wiley T, Nondahl D. (2003) The impact of hearing loss on quality of life in older adults. *Gerontologist*. 43, 661–668.

Eddins D, Hall J. (2010) Binaural processing and auditory asymmetries. In: Gordon-Salant S, Frisina R, Popper A, Fay R. eds. *The Aging Auditory System.* New York: Springer.

Epstein RM, Franks P, Fiscella K, Shields CG, Meldrum SC, Kravitz RL, et al. (2005) Measuring patient-centered communication in patient-physician consultations: Theoretical and practical issues. *Soc Sci Med.* 61, 1516–1528.

Frisina R, Walton J. (2006) Age-related structural and functional changes in the cochlear nucleus. *Hear Res.* 216–217, 216–223.

Frisina R, Zhu X, Souza P. (2009) Biological bases of age related hearing loss. Proceedings from the Phonak Hearing Care for Adults 2009: The Challenge of Aging, Chicago.

Gates G, Cobb J, D'Agostino R, Wolf P. (1993) The relation of hearing in the elderly to the presence of cardiovascular disease and cardiovascular risk factors. *Arch Otolaryngol Head Neck Surg.* 119, 156–161.

Gates G, Mills J. (2005) Presbycusis. *The Lancet.* 366, 1111–1120.

Gates G, Mills J, Nam B, D'Agostino R, Rubel E. (2002) Effects of age on the distortion product otoacoustic emission growth functions. *Hear Res.* 163, 53–60.

Genther D, Frick K, Chen D, Betz J, Lin F. (2013) Association of hearing loss with hospitalization and burden of disease in older adults. *JAMA.* 309, 2322–2324.

Gleich O, Weiss M, Strutz, J. (2004) Age-dependent changes in the lateral superior olive of the gerbil. *Hear Res.* 194, 47–59.

Gopinath B, Hickson L, Schneider J, McMahon C, Burlutsky G, Leeder S, et al. (2012) Hearing impaired adults are at increased risk of experiencing emotional distress and social engagement restrictions five years later. *Age Ageing.* 41, 618–623.

Gordon-Salant S. (2005) Hearing loss and aging: New research findings and clinical Implications. *J Rehabil Res Dev.* 42 (suppl 2), 9–24.

Helzner E, Cauley J, Pratt S, Wisniewski S, Zmuda J, Talbott E, et al. (2005) Race and sex differences in age-related hearing loss: The health, aging and body composition study. *J Am Geriatr Soc.* 53, 2119–2127.

Helzner E, Patel A, Pratt S, Sutton-Tyrrell K, Cauley J, Talbott E, et al. (2011) Hearing sensitivity in older adults: associations with cardiovascular risk factors in the health, aging and body composition study. *J Am Geriatr Soc.* 59, 972–979.

Humes L, Lister J, Wilson R, Cacace A, Cruickshanks K, Dubno J, et al. (2012) Central presbycusis: a review and evaluation of the evidence. *J Am Acad Audiol.* 23, 635–666.

Ison J, Tremblay K, Allen P. (2010) Closing the gap between neurobiology and human presbycusis: behavioral and evoked potential studies of age-related hearing loss in animal models and in humans. In: Gordon-Salant S, Frisina R, Fay R, Popper A, et al. eds. *The Aging Auditory System. Springer Handbook of Auditory Research.* Vol 34. New York: Springer.

Kalluri S, Humes L. (2012) Hearing technology and cognition. *Am J Audiol.* 21, 338–343.

Karpa M, Gopinath B, Beath K, Rochtchina R, Cumming RG, Wang JJ et al. (2010) Associations between hearing impairment and mortality risk in older persons: the Blue Mountains Hearing Study. *Ann Epidemiol.* 20, 452–459.

Kiely K, Gopinath B, Mitchell P, Luszcz M, Anstey K. (2012) Cognitive, health, and sociodemographic predictors of longitudinal decline in hearing acuity among older adults. *J Gerontol A Biol Sci Med Sci.* 67, 997–1003.

Leventhal A, Wang Y, Pu M, Zhou Y, Ma Y. (2003) GABA and its agonists improved visual cortical function in senescent monkeys. *Science.* 300, 812–815.

Li Y, Healy E, Dran J, Zhang J. (2006) Comorbidity between risk factors for severe hearing and memory impairment in older Americans. *Prev Med.* 43, 416–421.

Lin F. (2011) Hearing loss and cognition among older adults in the United States. *J Gerontol A Biol Sci Med Sci.* 66, 1131–1136.

Lin F, Metter E, O'Brien R, Resnick S, Zonderman A, Ferrucci L. (2011) Hearing loss and incident dementia. *Arch Neurol.* 68, 214–220.

Lin F, Thorpe R, Gordon-Salant S, Ferrucci L. (2011) Hearing loss prevalence and risk factors among older adults in the United States. *J Gerontol A Biol Sci Med Sci.* 66, 582–590.

Lin F, Yaffe K, Xia J, Xue Q, Harris T, Purchase-Helzner E, et al. (2013) Hearing loss and cognitive decline in older adults. *JAMA Intern Med.* 173, 293–299.

MacDonald M. (2011) The association between degree of hearing loss and depression in older adults. A thesis submitted in partial fulfillment of the requirement for the degree of Master of Science, University of British Columbia.

McMahon C, Kifley A, Rochtchina E, Newall P, Mitchell P. (2008) The contribution of family history to hearing loss in an older population. *Ear Hear.* 29, 578–584.

Mitchell A, Coyne J. (2010) *Screening for Depression in Clinical Practice: An Evidence-Based Guide*. New York: Oxford University Press.

Mitchell P, Gopinath B, McMahon C, Rochtina E, Wang J, Boyages S, et al. (2009) Relationship of type 2 diabetes to the prevalence, incidence and progression of age-related hearing loss. *Diabet Med.* 26, 483–488.

Pichora-Fuller K. Levitt H. (2012) Speech comprehension training and auditory and cognitive processing in older adults. *Am J Audiol.* 21, 351–357.

Roland P, Smith T, Schwartz S, Rosenfeld R, Ballachanda B, Earl L, et al. (2008) Clinical practice guidelines: Cerumen impaction. *Otolaryngology-Head and Neck Surgery*. 139, S1–S21.

Rosso A, Eaton C, Wallace R, Gold R, Stefanick M, Ockene J, et al. (2013) Geriatric syndromes and incident disability in older women: results from the women's health initiative observational study. *J Am Geriatr Soc.* 61, 371–379.

Saito H., Nishiwaki Y, Michikawa T, Kikuchi Y, Mizutari K, Takebayashi T, et al. (2010) Hearing handicap predicts the development of depressive symptoms after 3 years in older community-dwelling Japanese. *J Am Geriatr Soc.* 58, 93–97.

Scheibel M, Lindsay R, Tomiyasu U. (1975) Dendritic changes in aging human cortex. *Exp Neurol.* 47, 392–403.

Schmiedt R. (2010) The physiology of cochlear presbyacusis. In: Gordon-Salant S, Frisina R, Popper A, Fay R. eds. *The Aging Auditory System.* New York: Springer.

Schuknecht H. (1955) Presbycusis. *Laryngoscope.* 65, 402–419.

Schuknecht H, Gacek J. (1993) Cochlear pathology in presbycusis. *Ann Otol Rhinol Laryngol*. 102, 1–16.

Shankar S. (2010) Biology of aging brain. *Indian J Pathol Microbiol.* 53, 595–604.

Smith J, Mitchell P, Wang J, Leeder S. (2005) A health policy for hearing impairment in older Australians: what should it include? *Aust New Zealand Health Policy.* 2, 31.

Suzuka Y, Schuknecht H. (1988) Retrograde cochlear neuronal degeneration in human subjects. *Acta Otolaryngol Suppl.* 450, 1–20.

Tinetti M, Fried T, Boyd C. (2012) Designing health care for the most common chronic condition-multimorbidity. *J Am Med Assoc.* 307, 2493–2494.

Tremblay K, Kraus N. (2002) Beyond the ear: Central auditory plasticity. *Otoriolaringologica.* 52, 93–100.

Tremblay K, Ross B. (2007) Effects of age and age-related hearing loss on the brain. *J Commun Disord.* 40, 305–312.

Tun P, Williams V, Small B, Hafter E. (2012) The effects of aging on auditory processing and cognition. *Am J Audiol.* 21, 344–350.

Turner J, Caspary D. (2005) Comparison of two rat models of aging. In: Syka J, Merzenich MM, eds. *Plasticity and Signal Representation in the Auditory System.* New York: Springer.

Viljanen A, Kaprio J, Pyykko I, Sorri M, Koskenvuo M, Rantanen T. (2009) Hearing acuity as a predictor of walking difficulties in older women. *J Am Geriatr Soc.* 57, 2282–2286.

Walton J, Frisina R, O'Neill W. (1998) Age-related alteration in processing of temporal sound features in the auditory midbrain of the CBA mouse. *J Neurosci.* 18, 2764–2776.

Weinstein B. (2013) *Geriatric Audiology.* 2nd ed. New York: Thieme Medical Publishers.

Weinstein B, Amsel L. (1986). Hearing loss and senile dementia in the institutionalized elderly. *Clin Gerontol.* 4, 3–15.

Weinstein B, Ventry I. (1982) Hearing impairment and social isolation in the elderly. *J Speech Hear Res.* 25, 593–599.

Wolf M, Gazmararian J, Baker D. (2005) Health literacy and functional health status among older adults. *Arch Intern Med.* 165, 1946–1952.

Wu H, Chin J, Tong H. (2004) Screening for hearing impairment in a cohort of elderly patients attending a hospital geriatric medicine service. *Singapore Med J.* 45, 79–84.

Yamasoba T, Lin F, Someya S, Kashio A, Sakamoto T, Kondo K. (2013) Current concepts in age-related hearing loss: Epidemiology and mechanistic pathways. *Hear Res.* 303, 30–38. Available online at: http://www.sciencedirect.com/science/article/pii/S037859551300035X. Retrieved July 19, 2013.

Yueh B, Collins M, Souza P, Boyko E, Loovis C, Haegerty P, et al. (2010) Long-term effectiveness of screening for hearing loss: The screening for auditory impairment—which hearing assessment test (SAI-WHAT) randomized trial. *J Am Geriatr Soc.* 58, 427–434.

Zettel M, Zhu X, O'Neill W, Frisina R. (2007) Age-related decline in Kv3. 1b expression in the mouse auditory brainstem correlates with functional deficits in the medial olivocochlear efferent system. *JARO.* 8, 280–293.

Zuniga M, Dinkes R, Davalos-Bichara M, Carey J, Schubert M, King W, et al. (2012) Association between hearing loss and saccular dysfunction in older individuals. *Otol Neurotol.* 33, 1586–1592.

CHAPTER 35

Tinnitus and Hyperacusis

Richard S. Tyler, William Noble, Claudia Coelho,
Eveling Rojas Roncancio, and Hyung Jin Jun

INTRODUCTION

Tinnitus and hyperacusis are two challenging issues in audiology as patients can be desperate, and there are no cures. Nonetheless, several forms of treatment are available, and audiologists should possess a good foundation of hearing loss, hearing measurement, and rehabilitation to provide an accurate evaluation and effective management of tinnitus and hyperacusis. We advocate a flexible approach, as appropriate, that includes collaboration with informed psychologists and physicians.

Tinnitus can been defined as (1) a perception of sound (it must be heard), (2) involuntary (not produced intentionally), and (3) originating in the head (rather, it is not an externally produced sound), whereas hyperacusis does not have a widely accepted definition. Hyperacusis can involve loudness, annoyance, fear, and pain. We have noted that tinnitus is often accompanied by hyperacusis, and many current sound therapy protocols treat tinnitus and hyperacusis in parallel.

TINNITUS

Neurophysiological Causes, Mechanisms, and Models

Virtually anything that produces hearing loss can also produce tinnitus. The most common causes are noise exposure, aging, head injury, and medications. Sometimes, the causes are unknown. Estimates of prevalence vary, in part, because of differences in the definitions used in surveys (see Davis and Rafaie, 2000). The prevalence of tinnitus increases with age and hearing loss, but in particular is influenced by noise exposure. In our clinical experience, many workers in noisy situations report that the onset of tinnitus is gradual. Initially, tinnitus is heard only occasionally during the day or for brief periods after work. Subsequently, the duration of the tinnitus persists until it eventually becomes continuous. Typically (but not always), the onset of tinnitus occurs after the onset of hearing loss (sometimes years afterward). There are workers who report that tinnitus began after their exposure to noise had ended.

Tinnitus is classified as either sensory/neural or middle ear (Tyler and Babin, 1986). Middle-ear tinnitus is typically related to middle-ear vascular or muscular dysfunction. Sensory/neural tinnitus originates in the cochlear and/or neural auditory pathway. There are likely several different subgroups of tinnitus arising from different mechanisms (Dauman and Tyler, 1992; Tyler et al., 2008a). The mechanism responsible for coding tinnitus can originate in the cochlea, the brainstem, or the central nervous system (Figure 35.1). We believe that the auditory cortex must be active in tinnitus, since that is where sound is "perceived." This cortical activity could be associated with (1) an increase in spontaneous activity, (2) synchronous spontaneous activity across nerve fibers, and (3) more fibers tuned to the same best frequency (Salvi et al., 2000). As noted by Hallam (1989), other parts of the brain must be involved in patients who are anxious or have emotional reactions to their tinnitus. This includes the autonomic nervous system and the amygdala (Cacace, 2003).

It is curious that other parts of the nervous system can also influence tinnitus. For example, some patients report a change in their tinnitus with eye movements, light touch, or voluntary muscle contraction (Cacace, 2003; Levine, 2001). Others experience pressure around the head that can change tinnitus, or jaw clenching which can produce a high-pitch temporary tonal sound. It is not completely understood how the stimulation of systems outside the auditory pathway changes tinnitus. It is important to distinguish between muscle contraction that changes tinnitus by contracting middle-ear muscles and effects mediated by nonauditory neural pathways.

Some patients with "normal" hearing also report tinnitus. It should be remembered that "normal" hearing is arbitrary. Someone could have an audiometric "notch" of 20 dB hearing level (HL) at 4,000 Hz with 0-dB HL thresholds elsewhere. This likely represents an auditory pathology. Additionally, hearing thresholds are traditionally measured at octave frequencies from 250 to 8,000 Hz, leaving large regions of the cochlea unexamined, including frequencies above 8,000 Hz (see Kujawa and Liberman, 2009).

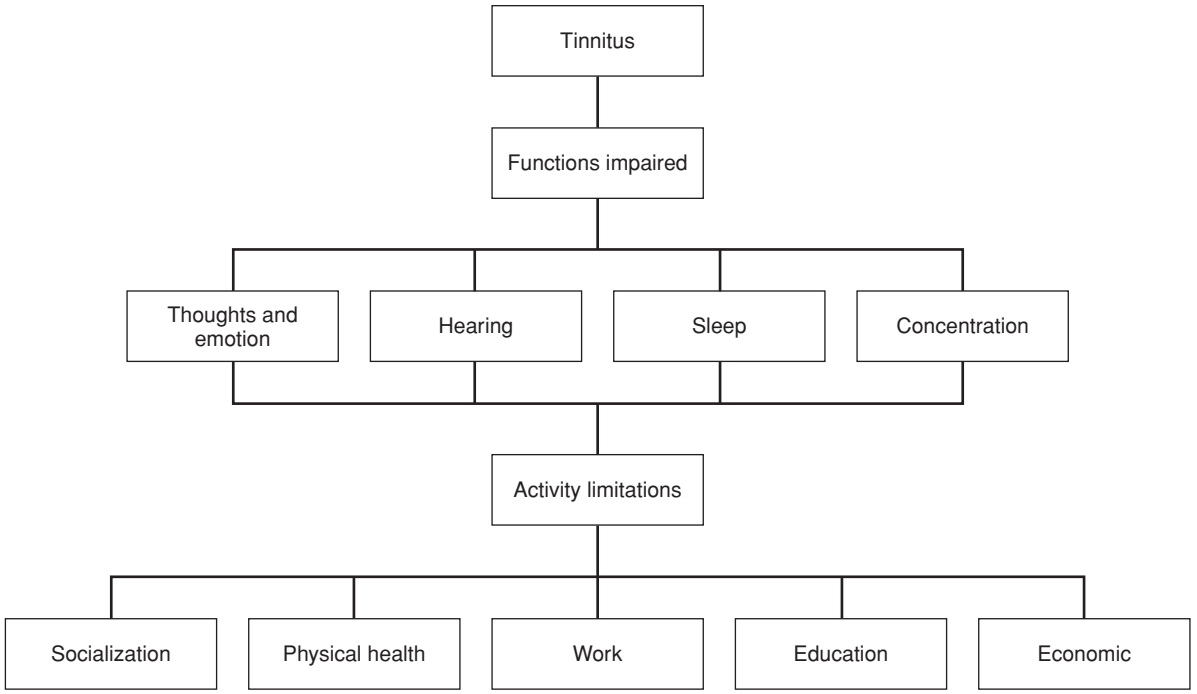


FIGURE 35.1 Domains where tinnitus can have an impact.

Auditory Hallucinations as Tinnitus

When someone reports hearing sounds that are like music or voices, it is important to consider mental illness. Reports of imagined voices or music can occur as part of psychotic illness such as schizophrenia. If there is no record of such illness, but there is evidence of depression, anxiety, or unrealistic thoughts or actions, then these should be addressed with the client and a referral provided to a mental health professional. In the absence of indications of mental illness, one could treat this as with other types of tinnitus. Certainly tinnitus can have a central origin. Patients who present no signs of mental illness and who hear music and voices could benefit from support, reassurance, and the counseling and sound therapy programs described later. It is important not to overreact to the patient's reports.

EVALUATION

Medical

Referral to an otologist is appropriate for pulsatile, sudden onset, worsening tinnitus, asymmetrical signs, and diseases of the auditory system (Perry and Gantz, 2000). A detailed clinical history could offer important clues about etiology and to help select laboratory and radiologic exams that will be required to investigate a particular case. It is often important to know what medications and dietary supplements are in use, diet, alcohol and smoking habits, food allergies, and lactose intolerance. A patient's and family's health history could give important information. Often, a focus of the evaluation will be on the cardiovascular system and on metabolic disturbances, such as diabetes and hypercholesterolemia. Laboratory examinations (e.g., cholesterol levels, glucose, zinc, screen for ototoxic drugs) and imaging tests (e.g., ultrasound, computed tomography scan, magnetic resonance imaging, magnetic resonance angiography) might be utilized. Generally, physicians are interested in identifying a possible treatable cause.

Middle-ear tinnitus is associated with either abnormal middle-ear blood flow or middle-ear muscle contraction. Some call this "objective" tinnitus, because it can be amplified and heard by the examiner. However, some spontaneous otoacoustic emissions, that are produced in the cochlea, can also be heard. Therefore, we prefer the term middle-ear tinnitus. Otologists may determine whether the tinnitus sensation changes with manipulations of blood flow (by asking patients to perform a brief vigorous exercise or by partially constricting a blood vessel of the neck). These manipulations can change the pulsing sensation. A proportion of these can be addressed surgically. Some vascular tumors also touch the eardrum and can be visually observed. Movements of the eardrum can sometimes be observed visually or with the help of measurements of air pressure in the external canal with tympanometry. Oral cavity examination may demonstrate myoclonic activity (palatal myoclonus).

Tinnitus can sometimes be influenced by movements of the head and neck. Some otologists search for signs of temporomandibular dysfunction which can involve jaw or facial pain or tenderness and difficulty or discomfort in chewing.

Another focus is a search for treatable sensory/neural tinnitus. This includes some forms of sudden hearing loss, Ménière's disease, or a tumor of the auditory nerve. It could be that some forms of tinnitus might be caused by metabolic diseases and deficiencies (e.g., anemia, diabetes, hypercholesterolemia, zinc and vitamin deficiency). Evaluations for these conditions would involve studies of the blood and urine.

Measuring the Tinnitus

The pitch, loudness, and amount of noise necessary to mask tinnitus can be measured to quantify tinnitus, provide assistance for fitting maskers, and monitor changes in tinnitus perception. (Often, this can be reimbursed in the United States when using Current Procedural Terminology (CPT) code 92625: Assessment of Tinnitus.) Patients can usually compare the pitch produced by a puretone to the "most prominent pitch" of their tinnitus (Tyler, 2000). Pitch matching can be highly variable, and an indication of the variability should be reported in the patient chart. Patients can also adjust the intensity of a tone so that it has the same loudness as their tinnitus. Sensation level is not a measure of loudness. The results of a tinnitus loudness match can be reported in dB sensation level (SL), but this level can only be interpreted over time for a particular patient if the hearing threshold at that frequency does not change. An alternative approach is to convert the physical intensity of the sound into the subjective loudness scale based on sones. Sones represent an international standard; 1 sone equals the loudness of a 40-dB sound pressure level (SPL) 1,000 Hz tone (about 49 dB HL) in a normal listener. A sound that has a loudness of 4 sones is four times as loud. Another measure of the magnitude of tinnitus is the amount of noise required to mask the tinnitus, sometimes referred to as the minimum masking level. The noise level (specify the frequency characteristics of the noise, e.g., broadband 250 to 8,000 Hz) is increased until it just masks the tinnitus.

Several things can contribute to the variability of tinnitus measurements. First, one should be aware that the test stimuli can change the tinnitus. This is probably more likely to happen for intense stimuli and when stimuli are presented ipsilaterally to the tinnitus. The ear receiving the stimuli should be reported. Second, in many patients the perception of tinnitus is not constant but varies throughout the day or from day to day. A reasonable approach to this is to make multiple measurements and report each value. The variability of the measurements can be documented by replicating the measures and recording the results of each trial in the patient's chart. For example, we often use the average of three loudness matches, three minimum masking levels, and six pitch matches (because pitch tends to be more variable). In patients with highly variable tinnitus, additional measurements can be made, and the measurements can be repeated at subsequent visits (particularly for a patient whose tinnitus changes).

Measuring the Reaction to the Tinnitus

People's reaction to their tinnitus covers a broad range. Some appear not to be particularly bothered by it, whereas for others, the tinnitus can have a dramatic effect on their lifestyle. The primary impairments can result in difficulties with thoughts and emotions, hearing, sleep, and concentration (Figure 35.1) (Erlandsson, 2000; Noble, 2013; Noble and Tyler, 2007). Sleep disturbance is one of the most common of these impairments (McKenna and Daniel, 2006; Tyler and Baker, 1983) causing some to have difficulty falling asleep, whereas others have difficulty falling back asleep if they wake up in the night.

When determining the impact tinnitus is having on an individual's life, an easy first step is to ask the person to "list all the problems you have that you associate with your tinnitus, starting with the problem that bothers you the most" (Tyler and Baker, 1983). This can be done before the first appointment and can lead to an open discussion of the important problems as perceived by the patient.

Several questionnaires designed to quantify the problems caused by tinnitus are available. These differ based on the scale used. Our experience is a 0 to 100 scale is easy for patients (a familiar decimal scale like dollars), in which a patient will respond 0, 5, 10, 15, . . . 100, which enables a 21-point scale. This provides greater resolution than a 0 to 10 scale. Questionnaires also differ on the scope of questions asked.

Tinnitus Handicap Questionnaire (Kuk et al., 1990) has been widely used to assess the influence of drugs (Coelho et al., 2013), cochlear implants (Pan et al., 2009), and sound therapy approaches (Tyler et al., 2012). Others (Meikle et al., 2012) also include general questions on the quality of life. We believe this can make the questionnaire less sensitive to treatment effects, as the quality of life can be influenced by many factors not directly captured by treating tinnitus. Tyler et al. (2006) developed the Tinnitus Primary Function Questionnaire (2014), shown in Appendix 35.1, which focuses on emotional, hearing, sleep, and concentration difficulties and is sensitive for clinical trials and assists in determining treatment needs. All appendices can be found at the end of the book.

TREATMENTS

There are two basic types of tinnitus treatment strategies: Those designed to reduce or eliminate the physical perception and those designed to change the patient's reaction.

Counseling

There are various counseling approaches: They range from providing information to more engaged collaborative counseling (for a review, see Tyler, 2006). Many of these are based on the work of Hallam (1989) known as tinnitus habituation

therapy. Others include strategies for improved coping, management, and behavioral change (Tyler et al., 1989). Among these are tinnitus activities treatment (Tyler et al., 2006), tinnitus retraining therapy (Jastreboff, 2000), and tinnitus cognitive behavior therapy (Anderson et al., 2002; Henry and Wilson, 2001; Robinson et al., 2008). The aim of these procedures is to provide ways for the person suffering with tinnitus to adjust his or her reactions to the experience.

The goals of these psychologically based therapies often overlap (Tyler et al., 2006). For example, patients can be helped to habituate to their tinnitus by de-emphasizing the fear associated with it. Another approach is to decrease the attention given to the tinnitus, often with the help of background sound. The way a patient thinks about the tinnitus can influence his or her reactions to it. Therefore, some clinicians will help patients consider how they think about their tinnitus. These thoughts can be challenged and revised. Another approach is to assist patients to change their focus away from their tinnitus. This can be facilitated by refocusing on other enjoyable and engaging activities. Having planned activities during which time it is known that the tinnitus is less intrusive can be very helpful.

It is our general view that many patients concerned about tinnitus can adapt to it after the explanation of its origin and its nonthreatening nature (where careful assessment has established that it is not a sign of a more serious medical condition); however, for a substantial number of patients such reassurance is less effective, and a more elaborate intervention is needed. The descriptions in the following sections provide guidance on the sorts of appropriate counseling within the context of general audiologic practice. When more elaborate counseling is required, reference to the clinical psychologist is appropriate.

Important attributes of the clinician include

- Ability to listen
- Patience
- Ability to be encouraging to the patient
- Emotional insightfulness
- Self-awareness
- Ability to laugh at the bittersweet aspects of life
- Positive self-esteem
- Ability to talk candidly about depression, anxiety, and other psychologic stressors

At the initial interview, it is helpful to determine if patients are curious, concerned, or distressed about their tinnitus (see Figure 35.2) (Tyler et al., 2008b). Much of the anxiety associated with tinnitus stems from uncertainty regarding its source and consequences. Curious patients typically require only basic information regarding possible causes, mechanisms, prevalence, consequences, and likely outcomes. These patients find that once the mystery of tinnitus is explained to them, their reaction is largely resolved. Concerned patients require more detail and benefit from information regarding things they can do on their own or other treatment options. Depending on the level of concern, these patients can require a more formal evaluation that includes the questionnaires and psychoacoustical measurements discussed earlier. Distressed patients require specific tinnitus treatment. Patients with severe anxiety and depression should obtain help from psychologists or psychiatrists. Patients who report suicidal thoughts or self-harm need to be further questioned regarding their intentions, and a referral to clinical psychology or psychiatric services should be made immediately if any concern exists.

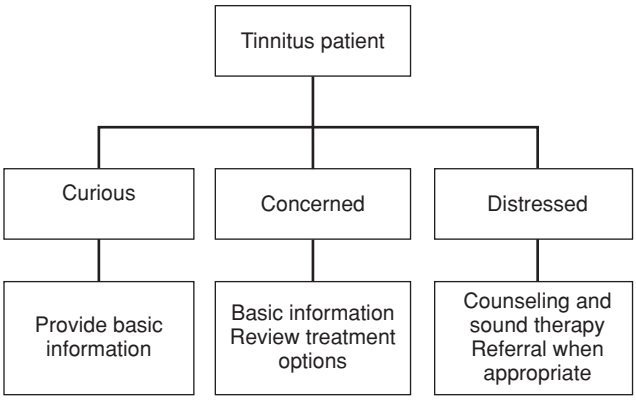


FIGURE 35.2 Broad categories of patients that reflect level of severity and therefore level of treatment needed.

Whereas individual counseling approaches will vary, the common elements of successful counseling strategies include the items listed in the following sections.

Providing Information

Most approaches provide information about hearing, hearing loss, and tinnitus. They usually include the causes, prevalence, and common consequences of tinnitus. For many people, the unknown aspects of tinnitus are the most alarming. They often find this basic information about tinnitus reassuring and may require no further assistance.

Thoughts and Emotions

It is helpful to distinguish the tinnitus itself from the person's reaction to the tinnitus. The way people think and feel about their tinnitus can have a major influence on their reactions. One focus of cognitive behavior therapy, and other counseling strategies, is on challenging a person's thoughts about tinnitus and thereby facilitating changes to the reactions to the tinnitus (Hallam, 1989; Henry and Wilson, 2001).

Meeting the Person's Needs

Some counseling procedures go beyond providing information and attempt to understand and influence the overall emotional well-being of the patient. By necessity, these procedures are collaborative and require more time. Several approaches are available to help individuals understand and

change the emotional consequences of their experience with their tinnitus. Mohr and Hedelund (2006) have developed a person-centered tinnitus therapy (trying to understand how the tinnitus fits into the larger scope of the individual's life).

Coping/Management Strategies

Some counseling approaches include coping/management strategies to help patients understand and change their perceptions about tinnitus and to modify their reactions and behaviors. Activities are planned to determine situations in which tinnitus might be a problem and then to modify their specific situation to reduce these occurrences. For example, patients might report that their tinnitus is worse when they first get home from work. This might be a result of sitting in a quiet room reflecting on the day's activities. An alternative activity might be to go for a walk while listening to music, or physical exercise, such as Tai Chi or yoga, to limber up. Just about any activity that reduces stress can be helpful.

Relaxation and Imagery Procedures

Some patients benefit from learning specific relaxation or imagery procedures. These can be used when people experience stress, and it can be helpful for them to learn relaxation strategies or to focus attention to other thoughts. Exercises to learn how to redirect attention away from the tinnitus are also employed. For example, in a quiet room, patients can imagine the sound of waves on a deserted beach. Then, they can redirect their attention to their tinnitus—then back to the waves. In a pair of excellent books (one for patients and one for clinicians), Henry and Wilson (2001) lay out programmatic exercises that patients can do on their own or that can be done in cooperation with the clinician.

Sound Therapies

Sound therapies include strategies that use background sounds to reduce the prominence of tinnitus or decrease its loudness or level of annoyance (Folmer et al., 2006; Vernon and Meikle, 2000).

The Use of Hearing Aids

Most patients with tinnitus also have hearing loss. Properly fitted hearing aids should help with communication and often also help with tinnitus by reducing the stress involved with intensive listening and by amplifying low-level background sounds. Hearing aids are often the first component of sound therapy for patients with tinnitus (Kochkin et al., 2011; Searchfield, 2006).

The Use of Wearable Sound Generators

Wearable ear-level devices are available that produce low-level noise. Some patients prefer this to listening to their tinnitus, perhaps because it is more pleasant to listen to or because the devices may decrease the loudness or prominence of their tinnitus. Some patients wear devices while their tinnitus is particularly annoying, whereas others use these devices during all waking hours. These devices look like hearing aids and are worn either behind the ear or in the ear. The noise should be adjusted to a level so that it does not interfere with communication.

The level of the background sound that is suggested varies with different sound therapies. There are two types of masking. Total masking covers tinnitus completely, so the person hears a "shhhhhh" instead of their tinnitus. With partial masking, the noise is set to a level so that both the tinnitus and the noise can be heard. This technique usually reduces the loudness, annoyance, or prominence of the tinnitus. Some protocols suggest that the ideal place for the noise should be at a level that is about equal to the tinnitus, where the tinnitus is just heard through the masking noise and mixes or blends with the tinnitus. Hazel recommended that "the masking sound does not completely cover the tinnitus." Other protocols focus on a lower level with the noise just in the background. For example, Tyler and Babin (1986, p. 3213) suggested that patients should use the "lowest masker level that provides adequate relief."

A masking device that is set to a high level might hamper listening to everyday sounds and might also make the tinnitus worse (Folmer et al., 2006). Devices are available that combine a hearing aid and a noise generator in a single unit.

The Use of Nonwearable Sound Generators

Many people also find it helpful to use sound in the background around the home or office or while they are going to sleep. Some use common devices, such as a fan, to produce the noise. There are also devices that are produced specifically for the purpose of producing background sounds, such as raindrops on leaves or waves on the shore. Pillows with tiny loudspeakers that connect into other sound devices are available to facilitate sleep. Radios and sound reproduction systems (e.g., MP3 players) have the advantage that they can be set according to preference. It is often helpful to have control of the level.

The Use of Music

Most people can enjoy some types of background music, and it is not surprising that many use a soft, light music in the background to help decrease the prominence of their tinnitus. There are now a variety of signals to choose from, including background music and nonrepeating "fractal tones."

Duration of Device Use

How long someone uses a device throughout the day and how many months someone should continue to use the device

can vary across patients. Some patients will use a device only when their tinnitus interferes with an important task, such as reading for work. We never insist that patients have to "avoid silence" because their speech perception is often worse in noise and some will constantly monitor their environment and their tinnitus in this effort. Some will choose not to use the device when communicating with others. In some cases it may be advisable to set the device to a low partial masking level and leave it on all day. This can help the patient forget about it and avoid focusing on the device and their tinnitus throughout the day. Some patients choose to use their noise generators for life, whereas others may choose to use them until they feel like they have attained some control and their reactions to the tinnitus are sufficiently reduced.

Tinnitus Activities Treatment

Our counseling approach has evolved over the years (Tyler and Babin, 1986; Tyler et al., 1989). We continue to prefer the partial masking strategy we recommended in the 1980s, although some patients benefit from total masking. We now call this approach tinnitus activities treatment (Tyler et al., 2006) which contains four separate modules.

Thoughts and Emotions

The way patients understand and think about their tinnitus influences their reactions to it. Providing information in a collaborative fashion to ensure understanding is essential. Key aspects of this area include

- Listening to the patient and addressing issues that are important to him or her
- Providing information about hearing, hearing loss, tinnitus, and role of conscious and subconscious attention
- Understanding the patient's reactions to unexpected, uncontrollable events
- Suggesting changes in behavior and lifestyle that can facilitate acceptance and habituation

It is important to help patients recognize the difference between the tinnitus itself and their reaction to it. Cognitive therapy separates the tinnitus from the patients' reactions to it and may provide a sense of control over the impact tinnitus has on their lives.

Hearing and Communication

Tinnitus and hearing loss often occur together, but the patients cannot "hear" their hearing loss, so they project their communication problems on the tinnitus. Reviewing the patient's hearing loss and its impact on communication may redirect some of the anxiety to an area where treatment is more obvious. In addition to hearing aid information, a review of assertive communication versus passive or aggressive communication styles is useful.

Sleep

Understanding normal sleep patterns is the first step in gaining control over the problem (McKenna, 2000; McKenna and Daniel, 2006). Other strategies include

- Exploring factors that can affect sleep (e.g., stress, environmental noise, room temperature)
- Arranging the bedroom to promote sleep (e.g., comfortable bedding, remove distracting items from room)
- Avoiding alcohol, smoking, and eating before bedtime
- Using sound to mask tinnitus (e.g., noise generators or soft radio)
- Learning relaxation exercises (e.g., imagery, progressive relaxation)

Concentration

In our therapy, we discuss the importance of concentration and things that affect our concentration. We review factors in the environment (e.g., lighting, background noise, distractions, and temperature) and personal factors (e.g., being tired, current health status, and other stressors in our lives) that impact our ability to focus our attention for a sustained period of time.

Activities in "attention diversion" (Henry and Wilson, 2001) give patients practice switching attention from one engaging task or stimulus to another. This type of exercise shows people that they can control what sounds, images, or other stimuli they consciously focus their attention on. Repeated practice with this type of activity can help give patients a sense of control over their attention as well as their tinnitus.

Medical Approaches

Pharmacologic Approach

We believe that no medication has been shown to effectively eliminate tinnitus in repeated clinical trials. Furthermore, it is likely that specific subgroups (as yet unidentified) of tinnitus patients benefit from some drugs. Evidence-based pharmacological approaches are limited to the treatment of tinnitus comorbidities such as depression, anxiety, and insomnia with antidepressants, antianxiety drugs (anxiolytics), and drugs to facilitate sleep. Potential medications include substances that have an action on blood circulation and viscosity, muscle relaxants, anticonvulsants, steroids, and diuretics. If indicated, they should be used in addition to counseling.

Surgical Approaches

Some forms of vascular abnormality can be treated by cutting away or restricting blood vessels. With myoclonus, surgical section of the tensor tympani and stapedial tendons can be successful.

In severe cases, cutting the eighth nerve (cochlear neurectomy) has been used, sacrificing hearing in that ear, but unfortunately, this has had only limited success in reducing tinnitus. Some physicians believe that tinnitus is a result of abnormal compression of the eighth nerve by a vessel (called vestibulocochlear compression syndrome) and have performed microvascular decompression operations of the vestibulocochlear nerve in the treatment of unilateral severe tinnitus.

Other Possible Treatments

Several other alternative approaches have been promoted to treat tinnitus. A guiding principle in judging these treatments should be that they have been shown to be effective in well-designed clinical studies that have been replicated. Chasing many different promised, but ineffective cures can be detrimental to the patient's overall emotional state.

Herbal supplements, such as ginkgo, and dietary supplements, such as zinc, magnesium, melatonin, copper, niacin/vitamin B_3, and cobalamin/vitamin B_{12}, have been proposed but there is no systematic evidence of beneficial effect (Coelho et al., 2013), although, some patients might experience some relief. Acupuncture has not been shown to be effective.

Some patients also ask about the potential benefit of changing their eating and drinking habits. Maintaining healthy diets and exercising are good for all of us, but no data indicate that these changes necessarily will improve tinnitus.

Some have suggested that temporomandibular joint dysfunction can cause tinnitus and thus treatment with jaw manipulations can cure tinnitus. It is not obvious to us that this is possible.

Patients will ask about these treatments, and we recommend discussing the principle of documented effectiveness in well-designed replicated studies. We also note that individual differences might be important. Some options can be harmless (such as drinking less coffee), but some alternative treatments do have important risks or side effects, and these should be explained to the patient.

One of the most promising treatments for tinnitus is the use of electricity. Studies have included stimulation of the cochlea (Rubinstein and Tyler, 2004) and brain. Electricity has also been successfully applied to the brain with transcranial magnetic stimulation. Numerous studies have demonstrated the effectiveness of cochlear implants in reducing tinnitus in many patients. Thus, it seems that, in a few years, there will be devices available that reduce tinnitus via electricity. The proportion of patients for whom this will help is not known; the details of the appropriate stimulus parameters are also unknown.

TINNITUS IN CHILDHOOD

Investigating tinnitus in childhood is challenging because of its subjectivity. It is rarely reported spontaneously by children and seldom routinely explored in pediatric otolaryngologic evaluation. Nonetheless, children do experience tinnitus. Most do not appear to be bothered, but remarkably, those who are bothered report similar suffering as adults (e.g., with emotional concerns, hearing, sleep, concentration), sometimes resulting in problems at school. It should be cautioned about intervention with children when parents were more concerned than the child because this may increase the child's anxiety about the tinnitus. Kentish and Crocker (2006) have designed tinnitus counseling specifically for children.

CONCLUSIONS REGARDING TINNITUS

There are likely many causes and mechanisms of tinnitus, and therefore, many treatments could be potential candidates. It is important to distinguish between the tinnitus and reactions to tinnitus. There are many counseling and sound therapies that likely help patients with their reactions. No medications or other physical treatments have been shown to be effective in well-designed and replicated trials.

HYPERACUSIS

The concept of hyperacusis includes loudness hyperacusis, annoyance hyperacusis, fear hyperacusis, and pain hyperacusis. One can readily imagine that sounds perceived as being very loud could easily become annoying. The anticipation of loud and/or annoying sounds could reasonably lead to the fear of these sounds. However, it is possible for sounds to be annoying or feared without being too loud. Patients also report that some sounds are physically painful, usually those perceived as loud. Occasionally, patients with tinnitus report that some sounds make their tinnitus worse. It is important to separate each of these symptoms, both for the patient and the clinician, to understand the problems carefully, and to offer treatment suggestions.

Neurophysiological Causes, Mechanisms, and Models of Hyperacusis

Anything that causes a sensory/neural hearing loss can likely also cause hyperacusis. Hyperacusis can also occur without identifiable hearing loss.

As a stimulus is increased, the activity of individual nerve fibers increases, and the number of nerve fibers activated increases (and usually its perceived loudness also increases). Moderately intense sounds might result in loudness hyperacusis if

1. greater than normal activity was produced on individual nerve fibers,
2. more nerve fibers were activated than normal, and/or
3. there was greater than normal synchrony across fibers.

Salvi et al. (2000) suggested that tinnitus could result from altered neural connections in the brain following hearing loss. Cortical neurons that previously have received input from damaged frequency-specific nerve fibers are colonized by neighboring regions of the brain, over-representing their representation at the cortex. We suggest that hyperacusis might also be a function of such brain plasticity. Following a peripheral hearing loss, say at 4,000 Hz, nerve fibers in the brain that normally respond to 4,000 Hz begin to respond to other, nearby frequencies, for example, 3,000 Hz. This results in more nerve fibers in the brain responding to 3,000 Hz than would be present normally. If hyperacusis is related to the number of fibers activated, this could account for it as a phenomenon.

Hazell (1987) suggested that hyperacusis might be the result of an "abnormal gain control." It is as if the brain receives a lack of information after hearing loss and therefore turns up some hypothetical gain control. Although intriguing, there are several problems with this suggestion. First, such a gain control mechanism must not operate on acoustic signals, because the hearing loss is not corrected. Second, our clinical experience is that some individuals without any apparent hearing loss also have hyperacusis. Third, most people with hearing loss do not report hyperacusis. Whenever emotions are involved, for example, in fear hyperacusis, other regions of the brain must also be involved.

EVALUATION OF HYPERACUSIS

Medical

The medical evaluation for hyperacusis parallels that for tinnitus. Some conditions have been associated with hyperacusis, including facial paralysis, head trauma, and metabolic disorders, infections (Lyme disease), and genetic (Williams' syndrome) abnormalities.

Measuring Hyperacusis

Loudness Hyperacusis

Loudness Discomfort Levels

Loudness discomfort levels (LDLs) can be performed with puretones at 500 and 4,000 Hz in each ear. We use the following instructions: "This is a test in which you will be hearing sounds in your right/left ear. We want you to decide when the sound first becomes uncomfortably loud."

Magnitude Estimation of Loudness

It is possible to present tones and ask for a rating of loudness on a scale from 0 to 100, with 100 being the loudest sound a person can imagine.

Hyperacusis scales have been developed to attempt to differentiate loudness and annoyance and to ascertain a general idea of the impact of hyperacusis on a patient's daily activities (see Appendix 35.1). The questionnaire asks individuals to consider several typical events they might encounter in their daily lives. They then separately rate the loudness and the annoyance for the same situations. For example, a patient may rate "telephone ringing in the same room" as 40 out of 100 on the loudness scale (with 100 being unbearably loud), whereas rating it as 85 out of 100 on the annoyance scale (with 100 being unbearably annoying).

Annoyance Hyperacusis

As mentioned, a questionnaire is shown in Appendix 35.2 where we attempt to quantify annoyance of sounds. Appendix 35.3 shows a handicap scale that asks patients to respond to statements in terms of their hearing loss, tinnitus, and hyperacusis. The statements include items such as "You avoid shopping" or "You feel depressed" and allow clinicians to separate the impact on function that patients perceive from their hearing loss, tinnitus, and hyperacusis. Another approach we have tried is to have patients rate recorded sounds. For example, we have patients rate recorded sounds of dishes hitting together, a lawn mower, and crowd noise.

Dauman and Bouscau-Faure (2005) developed a multiple activity scale for annoyance hyperacusis, providing 15 situations (e.g., concert, shopping center, work, church, children). Subjects rated from 1 to 10 each of the "relevant" activities, which were averaged for a total score. They also had patients rate annoyance hyperacusis on a scale from 1 to 10.

Fear Hyperacusis

Patients can develop a fear of very specific sounds or categories of sounds (e.g., those containing high frequencies) or of any intense sound. The simplest approach may be to ask the patients to make a list of sounds they fear to determine if a specific pattern exists.

Pain Hyperacusis

Some patients report that listening to some sounds create pain. Often, they are perceived as loud, and these patients typically have fear of these sounds.

TREATMENT FOR HYPERACUSIS

Treatments for hyperacusis are less well developed than for tinnitus. First, a clear distinction needs to be made about whether one is treating loudness, annoyance, fear, or a combination of these problems. The same basic tenets of good counseling mentioned earlier for tinnitus can be applied. Patients also have very different levels of distress associated with their hyperacusis. It is necessary to determine initially

if they are just curious, somewhat concerned, or very distressed.

Counseling

We believe hyperacusis can influence a patient's emotional well-being, hearing, communication, sleep, and concentration. One approach would include a cognitive behavior modification model, thus focusing on response, emotions, and thoughts (Henry and Wilson, 2001). In hyperacusis activities treatment, we include four sections.

The first section is emotional well-being. Patients with hyperacusis are often anxious and distressed about being exposed to intense noise. We provide information about possible mechanisms such as the coding of intensity by the number of nerve fibers and the activity on nerve fibers. We also review how our reactions are influenced by our expectations. If we expect that a visit from a father-in-law will be trouble, we are more likely to react negatively even to reasonable events. If we are expecting a situation to be unbearably loud, that raises our anxiety levels and influences how we react to sound. Some patients might have developed unreasonable expectations, so we provide some instruction on how we are able to change our reactions. It is important to help patients recognize the difference between the loudness of the sound and their reaction to it.

The second section is hearing and communication. Some patients avoid communication situations where they expect there to be intense sounds. Sound therapy to reduce loudness hyperacusis should be able to provide some assistance with this. Others will avoid using hearing aids or use gain settings that are insufficient. Patients can set the maximum output of their hearing aids temporarily to a lower level (Searchfield, 2006) and gradually increase this over time.

The third section is in the area of sleep. Occasionally, patients with fear hyperacusis will report that they do not sleep as well because of the anticipation of an intense sound. Partial masking sound therapy (e.g., playing music throughout the night) can be helpful for some.

The fourth section is that some patients report that they have difficulty concentrating in anticipation of an intense sound. Again, partial masking sound therapy can be helpful.

Sound Therapies

One fundamental issue is whether to protect the ears from moderately intense sounds, for example, with earplugs. Some patients with severe hyperacusis do this on their own. Of course, everyone (including hyperacusis patients) should protect their ears from potentially damaging high-intensity sounds. However, protecting a hyperacusis patient's ears from moderately intense sounds will not cure the patient's hyperacusis. In fact, restricting one's exposure to moderately intense sounds might have a further negative impact. One can imagine that if it is uncommon to hear a sound at 85 dB SPL, then whenever a sound of this level is perceived, it might result in an overreaction.

There are currently five general sound therapy strategies that we are aware of for hyperacusis. Good evidence to suggest their effectiveness is lacking.

Continuous Low-Level Broadband Noise

One approach is to expose the patient to continuous low-level broadband noise. The rationale is that the reduced input resulting from hearing loss is responsible for the hyperacusis. Correcting this reduced input by continuous noise exposure might reduce the hyperacusis. An advantage of this approach is that the noise can be provided during waking hours with wearable noise generators, and the patient does not have to focus on the device or treatments at times during the day. Having a device also provides some control for the patient, so they do not feel helpless. A possible disadvantage is that noise might interfere with speech perception. Formby and Gold (2002) have reported great success in some individuals with loudness hyperacusis (changes in the LDLs >50 dB). Dauman and Bouscau-Faure also used this procedure for annoyance hyperacusis with some positive results; however, they concluded that "noise generators do not provide a rapid solution to the problem" (p. 506) and that annoyance hyperacusis "does not improve as rapidly as usually reported" (p. 509).

Successive Approximations to High-Level Broadband Noise

A second approach is to allocate select times during the day for noise exposure and to gradually increase the duration and/or level of exposures over time. Another option is for patients to listen to noise for 30 minutes at a soft loudness each night for 2 weeks. For the next 2 weeks, the noise might be increased by a few decibels. For the next 2 weeks, the duration of exposure might be increased by another 30 minutes. The level of the noise can be gradually increased over several weeks. An advantage is that the patient can participate in the strategy for increased exposure. The level should never be uncomfortable, but higher levels can be used because the patient can listen to these levels at times when speech perception is not required.

Successive Approximations to Troublesome Sounds

A third approach that we have used involves recording of specific sounds. These can be selected with the patient and obtained by direct recordings or by prerecorded sound samples. It can be particularly helpful for patients who experience hyperacusis for specific sounds. The patient can then

replay the sounds at times when they are relaxed and at a much reduced (and not annoying) level. The patient can then successively increase the duration and level of listening periods over several weeks. The levels and categories of sounds can successively approximate the troublesome sounds. In parallel, we integrate the exposure of sounds in carefully controlled environments to situations closer and closer to approximating the actual situations resulting in hyperacusis.

Partial Masking

Partial masking with a continuous background sound can be used to reduce the loudness and prominence of intermittent sounds that might otherwise be annoying. For example, low levels of music can partially mask background annoying traffic noise. Additionally, the low-level music can create a background whereby the patient is less likely to anticipate being disturbed while getting to sleep, sleeping, or concentrating.

Gradual Increase of Maximum Output of Hearing Aid

The maximum output of a hearing aid can be initially lowered to a level where sounds are not perceived as loud (Searchfield, 2006). Then, over several days or weeks, the maximum output can be gradually increased. This successively exposes the patient to sounds perceived as louder. If the patient experiences hyperacusis, the maximum output can be lowered again.

Medication

The use of medication to treat hyperacusis has not been investigated in clinical trials, but interest is high because of the lack of cures.

HYPERACUSIS IN CHILDHOOD

Hyperacusis also occurs in children and is frequently associated with tinnitus and noise exposure. Moderately intense sound from the television, games, and telephone rings can cause some children to cover their ears with their hands. The symptoms can be so severe that activities such as car rides, vacuum cleaning, and lawn mowing are avoided. Generally, the approach we use is similar to our approach for adults. However, it is particularly important to consider the influence of peers, school, and parents.

CONCLUSIONS REGARDING HYPERACUSIS

Hyperacusis can be related to loudness, annoyance, and fear, and it is critical to distinguish the particular problems involved with individual patients. Counseling to provide information and reassurance and to challenge beliefs about hyperacusis can be very useful. We have identified four different approaches to sound therapy, including the use of continuous low-level noise, the use of successive approximation of troublesome sounds, partial masking to reduce the loudness and/or prominence of sounds, and gradually increasing the maximum output of a hearing aid. These approaches all require controlled investigations. No medications have been shown to be effective.

SOME FINAL THOUGHTS ON TINNITUS AND HYPERACUSIS

Patients with tinnitus and hyperacusis often find themselves receiving little or no help from healthcare professionals. Dismissive responses to their concerns only exacerbate their frustration. This is unfortunate because many can be helped with brief, supportive counseling. Audiologists are in an excellent position with their training in hearing, hearing loss, and counseling to provide important assistance to these patients. The challenge is substantial, but so are the rewards.

Clinically, most patients report (and likely do) benefit from counseling and sound therapy, including hearing aids. Healthcare reimbursement often requires evidence.

Many cases of tinnitus and hyperacusis can be prevented by reducing noise exposure. This can be accomplished by reducing noise levels at the source, using hearing protection, reducing the duration of exposure to noise, and taking "rests" away from the noise. Never miss an opportunity to promote the importance of hearing loss, tinnitus, and hyperacusis prevention.

ACKNOWLEDGMENTS

We wish to acknowledge grant support provided by the American Tinnitus Association and the National Institutes of Health (Grant No. 5R01DC005972). We thank George Haskell and Aditya Bardia for help with an earlier version of this document.

FOOD FOR THOUGHT

1. A war veteran comes in and complains he can't get to sleep at night because of the crickets in his ear. What is your plan?
2. You are asked by a local attorney for your help accessing whether a factory worker has, or has not hearing loss and/or tinnitus. She also wants to know what level of impairment the worker has, and what recommendations you would make to accommodate any work impediments because of his tinnitus. How can you help?

3. A father comes into the your clinic worried that his 8 year old daughter cries and covers her ears whenever she has two of her school friends over to play? It occurs when they make a lot of noise, but he states it is really not that noisy. His recollection is that it only occurs with the same two friends. What is your plan of evaluation and what do you tell the father?

KEY REFERENCES

thePoint A full list of references for this chapter can be found at http://thePoint.lww.com. Below are the key references for this chapter.

Anderson G, Stromgren T, Strom L, Lyttkens L. (2002) Randomized controlled trial of internet based cognitive behavior therapy for distress associated with tinnitus. *Psychosom Med.* 64 (5), 810–816.

Cacace AT. (2003) Expanding the biological basis of tinnitus: crossmodal origins and the role of neuroplasticity. *Hear Res.* 175, 112–132.

Coelho C, Witt SA, Ji H, Hansen MR, Gantz B, Tyler R. (2013) Zinc to treat tinnitus in the elderly: a randomized placebo controlled crossover trial. *Otol Neurotol.* 34, 1146–1154.

Dauman R, Bouscau-Faure F. (2005) Assessment and amelioration of hyperacusis in tinnitus patients. *Acta Otolaryngol.* 125 (5), 503–509.

Dauman R, Tyler RS. (1992) Some considerations on the classification of tinnitus. In: Aran J-M, Dauman R, eds. *Proceedings of the Fourth International Tinnitus Seminar (Bordeaux, France).* Amsterdam: Kugler & Ghedini Publications.

Davis A, Refaie A. (2000) Epidemiology of tinnitus. In: Tyler RS, ed. *Tinnitus Handbook.* San Diego, CA: Singular Publishing Group.

Erlandsson S. (2000) Psychologic profiles. In: Tyler RS, ed. *Tinnitus Handbook.* San Diego, CA: Singular Publishing Group.

Folmer RL, Martin WH, Shi Y, Edlefsen LL. (2006) Tinnitus sound therapies. In: Tyler RS, ed. *Tinnitus Treatment: Clinical Protocols.* New York: Thieme Medical Publishers.

Formby C, Gold SL. (2002) Modification of loudness discomfort levels: evidence for adaptive chronic auditory gain and its clinical relevance. *Semin Hear.* 23, 21–34.

Hallam RS. (1989) *Tinnitus: Living with the Ringing in Your Ears.* London: Harper Collins.

Hazell JWP. (1987) Tinnitus masking therapy. In: Hazell JWP, ed. *Tinnitus*. London: Churchill Livingston; pp 96–117.

Henry JL, Wilson PH. (2001) *The Psychological Management of Chronic Tinnitus: A Cognitive Behavioral Approach.* Needham Heights, MA: Allyn & Bacon.

Jastreboff PJ. (2000) Tinnitus habituation therapy (THT) and tinnitus retraining therapy (TRT). In: Tyler RS, ed. *Tinnitus Handbook.* San Diego, CA: Singular Publishing Group.

Kentish RC, Crocker SR. (2006) Scary monsters and waterfalls: tinnitus narrative therapy for children. In: Tyler RS, ed. *Tinnitus Treatment: Clinical Protocols.* New York: Thieme Medical Publishers.

Kochkin S, Tyler R, Born J. (2011) MarkeTrak VIII: prevalence of tinnitus and efficacy of treatments. *Hear Rev.* 18 (12), 10–26.

Kujawa SG, Liberman MC. (2009) Adding insult to injury: cochlear nerve degeneration after "temporary" noise-induced hearing loss. *J Neurosci.* 29 (45), 14077–14085.

Kuk FK, Tyler RS, Russell D, Jordan H. (1990) The psychometric properties of a tinnitus handicap questionnaire. *Ear Hear.* 11, 434–445.

Levine RA. (2001) Diagnostic issues in tinnitus: a neuro-otological perspective. *Seminars in Hearing*. 22, 23–36.

McKenna L. (2000) Tinnitus and insomnia. In: Tyler RS, ed. *Tinnitus Handbook*. San Diego: Singular.

McKenna L, Daniel HC. (2006) Tinnitus related insomnia treatment. In: Tyler RS, ed. *Tinnitus Treatment: Clinical Protocols.* New York: Thieme Medical Publishers.

Meikle MB, Henry JA, Griest SE, Stewart BJ, Abrams HB, McArdle R, et al. (2012) The Tinnitus Functional Index: development of a new clinical measure for chronic, intrusive tinnitus. *Ear Hear.* 33 (2), 153–176.

Mohr AM, Hedelund U. (2006) Tinnitus person-centered therapy. In: Tyler RS, ed. *Tinnitus Treatment: Clinical Protocols.* New York: Thieme Medical Publishers.

Noble W. (2013) *Self-Assessment of Hearing.* 2nd ed. San Diego, CA: Plural.

Noble W, Tyler RS. (2007) Physiology and phenomenology of tinnitus: implications for treatment. *Int J Audiol.* 46 (10), 569–574.

Pan T, Tyler RS, Ji H, Coelho C, Gehringer AK, Gogel SA. (2009) Changes in the Tinnitus Handicap Questionnaire after cochlear implantation. *Am J Audiol.* 18 (2), 144–151. PMCID: PMC2952398.

Perry BP, Gantz BJ. (2000) Medical and surgical evaluation and management of tinnitus. In: Tyler RS, ed. *Tinnitus Handbook.* San Diego, CA: Singular Publishing Group.

Robinson SK, Viirre ES, Bailey KA, Kindermann S, Minassian AL, Goldin PR, et al. (2008) A randomized controlled trial of cognitive-behavior therapy for tinnitus. *Int Tinnitus J.* 14 (2), 119–126.

Rubinstein JT, Tyler RS. (2004) Electrical suppression of tinnitus. In: Snow J, ed. *Tinnitus: Theory and Management.* Hamilton, ON, Canada: BC Decker; pp 326–335.

Salvi RJ, Lockwood AH, Burkard R. (2000) Neural plasticity and tinnitus. In: Tyler RS, ed. *Tinnitus Handbook.* San Diego, CA: Singular Publishing Group.

Searchfield G. (2006) Hearing aids and tinnitus. In: Tyler RS, ed. *Tinnitus Treatment: Clinical Protocols.* New York: Thieme Medical Publishers.

Tyler R, Coelho C, Tao P, Ji H, Noble W, Gehringer A, et al. (2008a) Identifying tinnitus subgroups with cluster analysis. *Am J Audiol.* 17 (2), S176–S184.

Tyler RS. (2000) Psychoacoustical measurement. In: Tyler RS, ed. *Tinnitus Handbook.* San Diego, CA: Singular Publishing Group.

Tyler RS, ed. (2006) *Tinnitus Treatment: Clinical Protocols.* New York: Thieme Medical Publishers.

Tyler RS, Babin RW. (1986) Tinnitus. In: Cummings CW, Fredrickson JM, Harker L, Krause CJ, Schuller DE, eds. *Otolaryngology – Head and Neck Surgery.* St. Louis, MO: Mosby.

Tyler RS, Baker LJ. (1983) Difficulties experienced by tinnitus sufferers. *J Speech Hear Disord.* 48, 150–154.

Tyler RS, Gehringer AK, Noble W, Dunn CC, Witt SA, Bardia A. (2006) Tinnitus activities treatment. In: Tyler RS, ed. *Tinnitus*

Treatment: Clinical Protocols. New York: Thieme Medical Publishers.

Tyler R, Ji H, Perreau A, Witt S, Noble B, Coelho C. (2014) The development and validation of theTinnitus Primary Function Questionnaire. *Am J Audiol*. doi: 10.1044/2014_AJA-13-0014. [Epub ahead of print]

Tyler RS, Haskell G, Gogle S, Gehringer A. (2008b) Establishing a tinnitus clinic in your practice. *Am J Audiol.* 17, 25–37.

Tyler RS, Noble W, Coelho C, Ji H. (2012) Tinnitus retraining therapy: mixing point and total masking are equally effective. *Ear Hear.* 33 (5), 588–594.

Tyler RS, Stouffer JL, Schum R. (1989) Audiological rehabilitation of the tinnitus patient. *J Acad Rehabil Audiol.* 22, 30–42.

Vernon J, Meikle M. (2000) Tinnitus masking: theory and practice. In: Tyler RS, ed. *Tinnitus Handbook.* San Diego, CA: Singular Publications.

CHAPTER 36

Tele-audiology

De Wet Swanepoel

INTRODUCTION

Little James, age 6 weeks, and his parents live in a remote community in northern Ontario, Canada. He has not passed an initial and a follow-up screening, so his parents have brought him to a local clinic for a diagnostic auditory brainstem response (ABR) assessment. Under the supervision of an audiologist, the trained screener connects the electrodes and places the insert earphones. The audiologist is not physically present, but is supervising remotely from a videoconferencing facility in Thunder Bay. Once James is set up, the audiologist remotely controls the computer running the ABR software and evaluates his hearing over the next 40 minutes. Until recently, such an evaluation would involve the expense, time, and inconvenience of parents having to take 2 days off work and travel by air from their remote community to Thunder Bay.

In another part of the world, Mr. Omondi, age 74, is taken by his son to the local mission hospital in his local village in western Kenya. For the past 15 years, he has been isolated from his family and community because of a disabling hearing loss. This is devastating in any society, but arguably worse when one is illiterate and resides in a culture where respect for the elderly and the oral traditions they pass on is central to the social fabric of the society. He has been unable to access audiologic services because none have existed apart from a handful of private clinics in the distant capital city. At the local hospital, a nurse instructs Mr. Omondi and puts on a headset and bone oscillator connected to a diagnostic computer–operated audiometer that runs an automated test sequence. Results are uploaded to a secure centralized server through a 3G cellular network connection. An audiologist in Sweden reviews the findings, interprets the results, and sends a recommendation back through the secure connection. For the first time, Mr. Omondi has his hearing loss diagnosed with the possibility of intervention.

These cases represent the possibilities that information and communication technologies (ICTs) are offering and serve as examples of typical services emerging in many parts of the world. This field of health care is referred to as telehealth or, in audiology, as tele-audiology.

Key challenges facing global health care, including audiologic health care, relate to issues of access, equity, quality, and cost-effectiveness (World Health Organization (WHO), 2011). In the past two decades, the world has seen exponential growth and development in ICTs that have revolutionized the way in which modern society communicates and exchanges information. These technologies are also impacting and changing modern health services and may provide a cost-effective and sustainable means of providing much-needed audiologic services to those populations identified as having restricted or limited access. The possible benefits may be far reaching, with telehealth able to improve healthcare access, quality of service delivery, effectiveness and efficiency of health care and towards amelioration of the inequitable distribution of health professionals globally (Dharmar et al., 2013; Wootton et al., 2009).

DEFINING TELEMEDICINE, TELEHEALTH, AND eHEALTH

The most commonly used term to describe the use of ICTs in health service delivery has traditionally been the term "telemedicine" (Fatehi and Wootton, 2012). Perhaps reflecting the rapid development and incorporation of new advances in technology, terminology was evolved and expanded over time. The term "telehealth" has been introduced to encompass a broader spectrum of health-related functions, including aspects of education and administration (Fatehi and Wootton, 2012). More recently, the term "eHealth" has been used to include aspects related to data management and processing. Evidence, however, suggests that these terms are used interchangeably by health providers and consumers and are ambiguous in their definition and the concepts to which they refer (Fatehi and Wootton, 2012). As a result of the ambiguity, the WHO (2010) and the American Telemedicine Association (ATA, 2013) have adopted "telemedicine" and "telehealth" as interchangeable concepts. The WHO defines these terms as "The delivery of health care services, where distance is a critical factor, by all health care professionals using information and communication technologies for the exchange of valid information for diagnosis, treatment and prevention of disease and injuries, research and evaluation and for the continuing education of health care providers, all in the interest of advancing the health of individuals and their communities" (p 9).

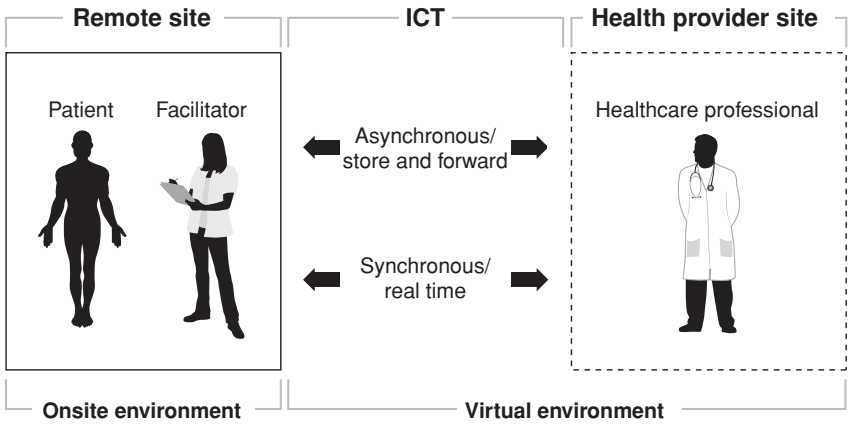


FIGURE 36.1 Illustration of telehealth service-delivery models. ICT, information and communication technology.

Another variation in terminology related to the field has been the use of the prefix "tele" in front of the specific field of healthcare practice, that is, tele-dermatology and tele-psychiatry. Generally, using "tele" has implied the use of ICTs to practice various aspects of these specific healthcare professions. For the purposes of this chapter, the term tele-audiology will be used as a category referring to the practice of audiology using telehealth modalities.

TELEHEALTH SERVICE DELIVERY MODELS

Telehealth is not a field of medicine or health care per se. Rather, it is a means by which existing healthcare needs may be served by using ICT to link healthcare expertise with patients and with other health providers with the ultimate aim of providing better access, efficiency, and cost-effectiveness to healthcare services like audiology. Telehealth may be utilized for educational or clinical purposes.

Providing telehealth services can be classified into two basic models that relate to the timing of the information exchange and the interaction between patient and health professional or between health professional and health professional (WHO, 2010). The first model has been called "store-and-forward" or asynchronous telehealth which involves sharing prerecorded information from one location to another (Figure 36.1). The information may be relayed from a patient site (also called the remote or originating site) to a healthcare provider site (also called the specialist or host site), or between healthcare providers. Importantly, "store-and-forward" telehealth models do not require a healthcare provider to interact with the information in real time. An example may be something as simple as sharing a prerecorded puretone audiogram by e-mail to an expert colleague for an opinion on diagnosis and management. In some cases, a facilitator at the patient site may be present to record the clinical information to be relayed to a healthcare provider or patients might self-monitor or assess and relay this information themselves.

In comparison, "real-time" or synchronous telehealth requires that both individuals (e.g., the healthcare provider and patient) are simultaneously engaging in information exchange (Figure 36.2). A typical example may be a consultation with a patient using videoconferencing, but it may also include diagnostic assessments by a specialist who remotely controls a computer-operated diagnostic device connected to a patient. Applications will vary and may include expert surveillance or expert presence through audio and/or video facilities of procedures at the patient site (Swanepoel et al., 2010a).

CONSIDERATIONS FOR A TELE-AUDIOLOGY SERVICE

Information and Communication Technologies for Telehealth

Sharing information and communication is foundational to the concept of telehealth. This process is facilitated by the use of ICTs that provide the technology and connectivity for sharing information between different sites (Table 36.1). Technology and connectivity are closely related and often contained in the same concept. For example, a cellular phone

TABLE 36.1

Technologies Typically Facilitating Store-and-Forward (Asynchronous) and Real-time (Synchronous) Telehealth Practice

Store-and-Forward (Asynchronous)	Real Time (Synchronous)
E-mail	Videoconferencing (video and voice)
Facsimile	Voice call (Fixed line, mobile, VoIP, or satellite)
Multimedia message (MMS)	Desktop sharing software (to control device)
Text message	Virtual private network (VPN)
Shared online networks	
Patient online portals	
Web-based eHealth patient service sites	

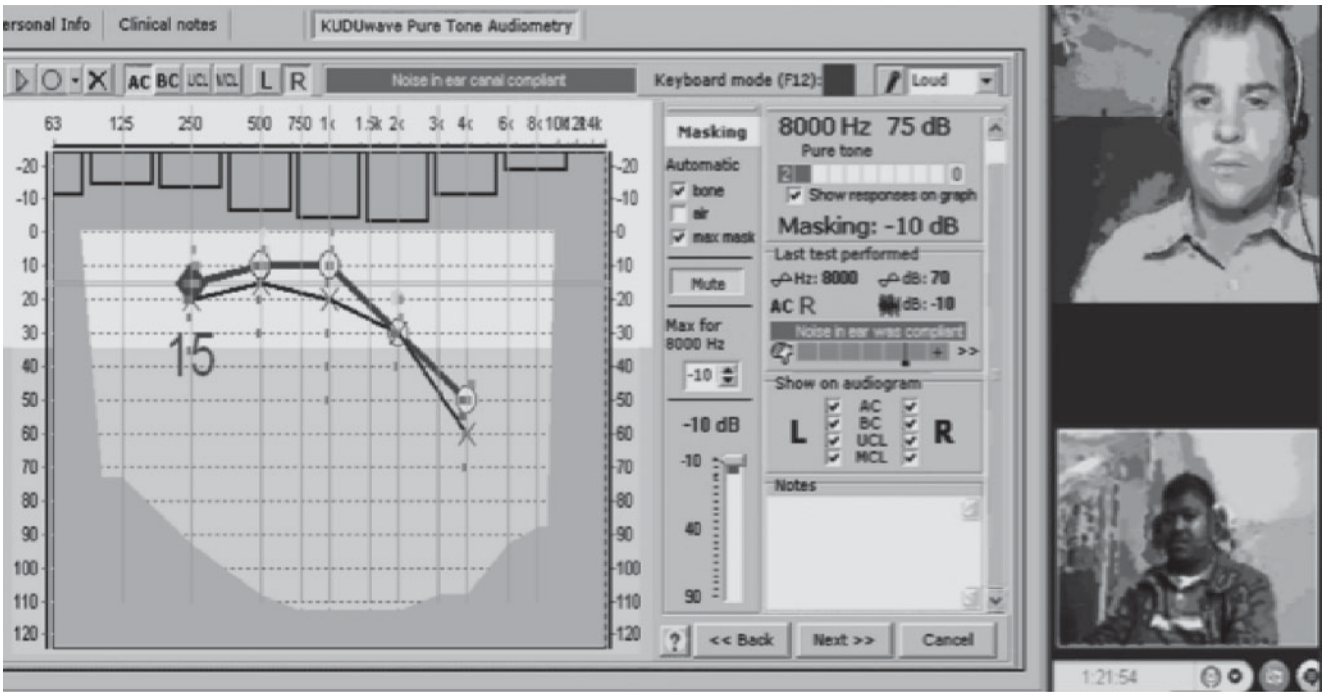


FIGURE 36.2 Remote audiometry test transmitted from Dallas, Texas, to Pretoria, South Africa. The clinician is visualized in the *top right corner* with the patient visualized in the *bottom right corner.* The left and right ear thresholds are visualized on the audiogram on the *left side* of the figure. The test session can be watched at http://www.youtube.com/watch?v=HDfjuvP0Dh0.

is a physical handset (technology) that transmits and receives information using radio signals (connection). A more complex but typical example with overlap of the technology and connectivity concepts is e-mail. The e-mail technology includes the software interface used on a device of some sort (e.g., PC, tablet, or smartphone) and the connection being facilitated through the Internet connection of the particular device (e.g., WiFi; global system for mobile communication or GSM). The technologies that may be used in telehealth may therefore include non-Internet–based technologies such as facsimile, two-way radio or telephone, or Internet-based technologies that may include e-mail, web browsing, file transfer protocol (FTP), shared networks, voice over Internet protocol (VoIP), video traffic for teleconsultation and videoconferencing, and remote desktop sharing software (e.g., TeamViewer). Devices ranging from smartphones and tablets to laptops and personal computers (PCs) usually operate the Internet-based telehealth technologies. Connectivity includes options such as fixed line telephone networks, cellular phone and Internet network, satellite phone and Internet networks, integrated services digital network (ISDN), and digital subscriber line (DSL).

Technologies available for telehealth applications are diverse and continually evolving and developing. Although technologies may be very expensive, there are increasingly more affordable options available. Videoconferencing rooms with advanced equipment, for example, may be cost prohibitive for many healthcare facilities, but free videoconferencing software (e.g., Skype, ooVoo, Google Hangout) could be used on devices such as laptops, tablets, or smartphones.

Equipment for Telehealth Purposes

Apart from the ICTs required for telehealth services, equipment must be considered. For example, if videoconferencing is being done via a computer, an internal or external webcam and microphone must be used at both sites to capture the video and audio images for transmission.

Computer-Operated Equipment

Computer-operated equipment allows easy sharing of information in a store-and-forward mode, that is by e-mailing a printout of results. Computer-operated equipment equipped with remote desktop sharing software allows the healthcare provider to control onsite equipment in real time for the desired assessments, procedures, or interventions.

Dedicated Tele-audiology Technologies

The practice of audiology is especially reliant on computer-operated equipment for screening, diagnosis, and intervention (assistive devices), with the potential to be highly compatible with telehealth service provision. However, currently audiologic equipment is usually not made with the intention of using it within a telehealth service-delivery model and therefore may lack some important features

that could add to the reliability and quality of such assessments (Clark and Swanepoel, 2014). For example, when conducting audiometric testing remotely, the audiologists do not know whether the environment is sufficiently quiet or not. Environmental and patient feedback systems may be very useful to incorporate in future equipment as one way of making devices more specifically telehealth enabled (Swanepoel et al., 2010a). A recently developed diagnostic audiometer, specifically developed for telehealth purposes, includes examples of such novel features as live and continuous environmental noise monitoring, lightweight and mobile design with all hardware encased in the earcups and powered from a USB power source, and the ability to record patient responses and response times (Maclennan-Smith et al., 2013; Swanepoel et al., 2010a, 2013b).

Automation in Tele-audiology

Other features of audiologic equipment that may be beneficial to telehealth are the automation or semiautomation of equipment-based procedures (Swanepoel et al., 2010a, 2010b). Automation allows for telehealth facilitators to take the necessary measurements and relay them to audiologists for interpretation/management. Advances in computer technology are making automation of audiologic assessment procedures easier, which has led to growing interest in automation (Margolis et al., 2010; Swanepoel et al., 2010b). A recent meta-analysis of automated diagnostic puretone audiometry , for example, revealed that automated audiometry is equally accurate and reliable compared to the gold standard of manual audiometry. The authors did however highlight the shortage of evidence for audiometry with bone conduction, in patients with various degrees and types of hearing loss and difficult-to-test populations (Mahomed et al., 2013).

Telehealth Facilitators

Technology and connectivity, despite being experienced by users as temperamental at times, are often the easy part of a successful telehealth program. A key element required for the success of a program often depends on the support personnel, in particular the telehealth facilitator. Telehealth models may typically utilize nonspecialist personnel, referred to as telehealth facilitators, to facilitate the telehealth encounter linking the health provider and the patient (Swanepoel et al., 2010a). These personnel may vary, such as community healthcare workers, assistants, nurses, primary healthcare physicians, or IT specialists. They are not qualified to make diagnoses or interpretations but with proper training are qualified to facilitate the information exchange for the specific telehealth encounter. These individuals must be thoroughly trained in the required equipment, procedures, protocols, and patient interactions related to the service provided. Regular monitoring and retraining are important. Often the quality and success of a clinical telehealth service are primarily dependent on the telehealth facilitator.

Patient and Environmental Considerations

Because of the nature of telehealth services, where health professionals are not with the patient in person, additional considerations related to the patient receiving the service and the environment in which the service is provided are required. In fact, some patients and some environments may not be appropriate for provision of telehealth services. In pediatric audiology, for example, it may not be appropriate to conduct visual reinforcement audiometry. The timed behavioral response to the signal may be difficult to follow because of transmission delays in activating the stimulus and in observing the response. Furthermore, video resolution of the patient may likely be insufficient to observe minor behavioral reactions such as eye movements. Table 36.2 provides a summary of patient considerations that may impact telehealth services, which clinicians should keep in mind when considering service provision.

TABLE 36.2

Patient Considerations in Tele-audiology (Summarized from ASHA, 2013a)

Domains	Characteristics
Physical and sensory	Hearing ability Visual ability Manual dexterity Physical endurance
Cognition, behavior, and motivation	Level of cognitive functioning Attention maintenance Ability to sit in front of camera with minimal extraneous movements (to avoid poor image quality) Willingness of patient/family to receive telehealth services
Communication characteristics	Auditory comprehension Literacy Speech intelligibility Cultural/linguistic variables Availability of interpreter
Support resources	Availability of technology Availability of facilitator Ability to follow directions to operate and troubleshoot telehealth technology and transmission (patient/facilitator/family)

TABLE 36.3

Summary of Core Standards Related to Telehealth in Clinical Practice

Administrative Standards

Organizations

1. Follow standard operating policies and procedures
2. Systematic quality improvement and performance management process
3. Compliance with relevant legislation, regulation, and accreditation requirements
4. Assuring patients are aware of their rights and responsibilities
5. Telehealth integrated into existing operational procedures for obtaining consent
6. Collaborative partnerships aware of applicable legal and regulatory requirements

Health professionals

1. Fully licensed/registered with respective regulatory/licensing bodies where patient is located
2. Aware of credentialing requirements at site where consultant and at site where patient is located
3. Aware of locus of accountability and any/all requirements when practicing in another jurisdiction
4. Cognizant of provider–patient relationship established within the context of telemedicine proceeding with evidence-based, best possible standard of care
5. Necessary education, training/orientation, and ongoing continuing professional development to ensure competencies for safe provision of quality services

Clinical standards

1. Organization and health professionals practicing telehealth aware of own professional discipline standards and those to be upheld via telehealth considering specific context, location, timing, and services
2. Guided by professional discipline and national clinical practice guidelines when practicing via telehealth with consideration of any modifications to specialty-specific clinical practice guidelines for telehealth to meet clinical requirements for discipline

Technical standards

1. Ensure equipment sufficient and functioning to support diagnostic needs
2. Strategies to address environmental elements of care for safe use of equipment
3. Comply with relevant safety laws, regulations, and codes for technology and technical safety
4. Compliant infection control policies and procedures for telehealth practice
5. Policies and procedures to comply with local legislated and regulatory rules for protection of patient health information ensuring physical safety of equipment and electronic security of data
6. Appropriate redundant systems to ensure availability of network for critical connectivity
7. Appropriate redundant clinical video and exam equipment for critical clinical encounters and clinical functions
8. Meet technical standards for safety and efficacy for devices interacting with patient
9. Processes to ensure safety and effectiveness of equipment through ongoing maintenance

Source: Adapted from ATA (2007).

Environmental characteristics are especially important in audiology where most services require ambient noise levels kept to a minimum. Considerations should include quiet rooms with minimal distractions, as well as good lighting and appropriate design to ensure optimal video and audio transmission. Positioning of the patient and placement of equipment in relation to the patient are important variables when videoconferencing is being used (American Speech-Language-Hearing Association (ASHA), 2013a).

Tele-audiology Standards and Guidelines

Professionals and organizations providing health services are required to adhere to administrative, professional, clinical, and technical standards when practicing via telehealth. A summary of core standards specified by the ATA (2007) can be found in Table 36.3. These serve as general but fundamental requirements across healthcare disciplines providing remote health services, interactive patient encounters, and other electronic communications between patients and health professionals. Discipline-specific guidelines are necessary when considering telehealth for particular patient groups, disorders, and contexts. In audiology, discipline-specific guidelines have been proposed by the ASHA (2013a), the Canadian Association for Speech-Language Pathologists and Audiologists (CASLPA, 2006), and the American Academy of Audiology (AAA, 2008).

These position statements and resolutions on telehealth in audiology endorse and support its use to improve access

to audiologic services. There is agreement that telehealth has the potential to overcome accessibility barriers related to distance, travel costs, weather, mobility, patient schedules, and personnel shortages. At present, tele-audiology is proposed primarily as a way to increase access to care when deemed in the best interest of the patient and not to replace existing services that could be provided through face-to-face services without difficulty. As more research evidence accumulates, the role of tele-audiology is likely to evolve beyond only improving access to care to also improving the efficiency in service delivery and cost efficiency in existing audiologic practice (Swanepoel et al., 2010a).

Current guidelines and resolutions also agree that telehealth service must be equivalent to in-person service provision in scope and nature. Audiologists must also be attentive to patient perceptions and cultural diversity that may result in differences in comfort level with telehealth technology. Furthermore, tele-audiology service should always be provided or supervised by a qualified audiologist. Audiologists must be aware of and accountable to the ethical and clinical standards of their professional regulating or licensing body and abide by these as specified for the state or province. Cross-border or cross-state telehealth service provision introduces important questions on accountability (ASHA, 2013b). At present, some states in the United States require that the professional must be licensed both at the home state and in the state where the patient is being served. There is an increasing number of states with telehealth provisions for audiology, but characterized by widespread variability (ASHA, 2013c). It is also the responsibility of the service provider to ensure adherence to the privacy and security requirements of federal and state regulations when storing and transmitting patient information. Protecting patient information is not a simple or straightforward task and it has been recommended to consult an expert specializing in these issues (ASHA, 2013a).

Reimbursement and insurance coverage for telehealth services has been identified as one of the most important reasons for the slow adoption of telehealth (Bashshur et al., 2013). Advocacy at the state and national level to enact laws removing reimbursement barriers for telehealth services would increase widespread adoption of these services. Increasingly, communities are changing reimbursement regulations to incorporate telehealth services (ASHA, 2013a; Australian Government Department of Health and Aging, 2013; Bashshur et al., 2013). Unfortunately, when reimbursement regulations include telehealth, they often cover only certain types of services such as face-to-face consultations, as opposed to store-and-forward services that are likely to hold more promise for improved time and cost efficiency (Australia Government, 2013). Reimbursement and insurance coverage programs for telehealth services continue to change, and therefore must be verified prior to initiation of the services (ASHA, 2013a). The responsibility of fee reimbursement should be established with patients prior to service delivery (CASLPA, 2006).

WHY TELEHEALTH FOR AUDIOLOGY

Telehealth has been proposed to hold great potential toward improved access, quality, efficiency, and cost-effectiveness of healthcare services particularly for populations who have traditionally been underserved (WHO, 2010). Improved efficiency in competitive healthcare environments because of telehealth services has been demonstrated to result in increased patient services and hospital and professional billing revenue (Dharmar et al., 2013). Telehealth can potentially bridge the general barriers often created by distance, poor travel infrastructure, severe weather, and unequal distribution of healthcare providers in urban and rural settings or even across world regions (Swanepoel et al., 2010a). These potential advantages of telehealth are particularly appealing in the field of global hearing health care where there is a dearth of hearing health professionals who are able to provide audiologic services to an increasing number of persons who require care. In addition to the prevalence of hearing loss and the shortage of hearing healthcare professionals, the advances in technology and rapid expansion in connectivity are opening up new avenues for delivering tele-audiology.

Addressing Current Challenges

Increasing Hearing Loss Caseload

Recent estimates indicate a global prevalence of just over 10% for permanent bilateral adult hearing loss of 35 dB or greater, which translates to more than half a billion adults affected (Stevens et al., 2013). Excluding milder losses, the WHO (2013) estimates that 328 million adults (hearing losses >40 dB) and 32 million children (hearing losses >30 dB) have a disabling hearing loss. It is not surprising, therefore, that it is the most prevalent chronic disability with 5.3% of the world population suffering from disabling hearing loss.

An important characteristic of global hearing loss prevalence is the fact that it is increasing significantly because of the increase in life expectancy. Since 1990, the average life expectancy has increased from 64 to 68 years of age in 2009 with an increase from 76 to 80 years of age in high-income countries (WHO, 2011). With aging as the most common cause of hearing loss, longer life expectancies globally mean an unmatched growth in hearing loss prevalence in the foreseeable future (Swanepoel et al., 2013a). In countries like the United States, Baby Boomers (born between 1946 and 1964) are now entering the geriatric age categories, and because of longer life expectancies and the disproportionately large numbers in relation to previous generations, there will be unprecedented demands for hearing health services. The growing number of persons with hearing loss globally raises the question of how they will be able to access audiologic

care. Tele-audiology potentially offers a way to provide the growing number of patients with access to services.

Shortage of Audiologic Services

Unfortunately there is a global shortage of ear and hearing health professionals (Fagan and Jacobs, 2009; Goulios and Patuzzi, 2008). As a result, close to 80% of persons with hearing loss globally cannot access hearing healthcare services because they reside in developing countries where hearing healthcare services are often unavailable (Fagan and Jacobs, 2009; Goulios and Patuzzi, 2008; WHO, 2006). In sub-Saharan Africa, for example, many countries have no audiology or ENT services (Fagan and Jacobs, 2009). Of the 46 countries constituting this world region, which comprises almost 1 billion people, only one (South Africa) provides tertiary-level education for audiologists.

A shortage of hearing healthcare services is not only a developing world problem, however. According to estimates in the United States, there is a major capacity shortage in terms of the need for hearing evaluations and the capacity of the current audiologic workforce to deliver these. Estimations indicated that in the year 2000 there was an annual shortfall of 8 million audiograms projected to increase to 15 million by 2050 (Margolis and Morgan, 2008). A more recent analysis of audiologic demand in the United States in relation to current and projected growth rate of new graduates demonstrate a growing mismatch between the demand for audiologic services and capacity to deliver these (Windmill and Freeman, 2013). Estimates indicate that the number of persons entering the profession will have to increase by 50% immediately and attrition rate lowered to 20% to meet the demand. Alternatives, including increased capacity for service delivery through telehealth, are suggested as ways of improving the audiologic service-delivery capacity.

In addition to professional shortages, underserved regions also persist in developed countries including Canada, Australia, and the United States because of traveling distances and geographical and weather obstacles. Utilizing telehealth approaches in hearing health care has been suggested as a means of addressing the availability and distribution of audiologic expertise and increasing the access to audiologic care (Swanepoel et al., 2010a, 2010d).

Enhancing Audiologic Efficiency

Apart from the potential telehealth has for improving access to the growing need for audiologic services, future applications of telehealth may also enhance existing services by improving efficiency with resultant cost-savings for health systems and individuals. Use of asynchronous screening and even diagnostic methods of assessing and monitoring patients may ensure that only those requiring advanced assessments, counseling, or intervention are referred for full audiologic assessments. An example of this is a national tele-audiology service providing mandated monthly audiologic monitoring for patients with multidrug-resistant tuberculosis receiving ototoxic medications (Bashshur et al., 2013). Many of these patients are in remote rural locations where automated audiometry is facilitated at primary healthcare facilities and results uploaded through cellular networks to a server, allowing remote interpretation and recommendations by audiologists. This saves traveling costs for patients who would otherwise have to attend facilities with an audiologist, which may be hundreds of miles away. Prior to this service, the majority of these patients were not monitored as required because of the travel and cost barriers. In this way, the efficiency of audiologic services is enhanced. Many more such examples could be provided but systematic research documenting enhanced efficiency and cost containment should be prioritized.

Advances in Technology and Expanding Connectivity

The past two decades have seen unprecedented growth in technology. There has been a revolution in the processing capabilities and size of computing equipment such as personal desktop computers. In the past 5 years, a new market of tablet computers, smartphones, and phablets (hybrid phone and tablet) has emerged that is charting new ways of engaging with information. Alongside the hardware developments, the Internet has changed and developed to become the predominant workspace from which devices access, store, and share information. These advances allow new and innovative ways of utilizing technology in hearing health care (Kelly and Minges, 2012). Interfacing with audiologic equipment may be facilitated through tablets and smartphones as opposed to desktop or laptop computers. Novel applications of technologies may also have facilitated automation of audiologic testing in some respects. Recent evidence, for example, has suggested that there is renewed and increasing interest in automated audiometry largely because of the possibilities for efficient, accurate, flexible, user-friendly and reliable functioning offered by newer technologies (Mahomed et al., 2013).

In addition to technologic advances, the rapid improvement and distribution of connectivity is providing an increasing opportunity for implementation of telehealth globally. Connectivity around the world has grown exponentially with one in every three people worldwide having access to the Internet in 2012 (Internet World Stats, 2012). Although Internet penetration in a region like Africa is still reasonably low and bandwidth costs are expensive, the large-scale rollout of cellular networks across Africa and the rest of the world is opening doors through mobile connectivity in the most remote and underserved areas (Kelly and Minges, 2012; Swanepoel et al., 2010a). Growth in the spread of mobile phones globally has been unmatched in the history of technology. In 2012 there were more than

6 billion mobile subscriptions, with 75% of the world's people having access to a mobile phone (Kelly and Minges, 2012). It is estimated that by 2015 there will be more mobile subscriptions than people, with more than 80% from developing countries. This growth in connectivity and in mobile phone technology is already transforming the delivery of healthcare services. It allows access to information and communication sharing with the most underserved areas, which allows for provision of telehealth services.

Continuing advances in technology and connectivity are therefore making tele-audiology a feasible and opportune area of practice and research set to change the way in which audiologists provide services. It is important that the profession of audiology capitalizes on these advances and directs its development to ensure that not only optimal access but also best practice and care is delivered to patients.

TELE-AUDIOLOGY IN PRACTICE

Tele-audiology is becoming increasingly used as part of audiologic service delivery across a variety of applications. Examples are provided within the broad categories of audiologic screening, diagnosis, intervention, and continuing professional development.

Screening

Screening lends itself quite naturally to store-and-forward (asynchronous) telehealth applications with trained nonspecialist personnel being able to conduct screenings. In fact, the widespread screening of newborns for hearing loss, although not often termed as such, may be considered a form of asynchronous telehealth. Trained screeners conduct the measurements in hospitals and subsequently upload the results to secure servers, where the data are managed and the necessary follow-up measures are recommended. Although results are not necessarily reviewed remotely, they are still managed as part of an electronic health information system.

In a systematic review of telehealth applications in audiology (Swanepoel and Hall, 2010), several reports of tele-audiology screening applications were identified, including real-time screening of newborns with automated otoacoustic emissions (OAE) and ABR (Krumm et al., 2008). Unsurprisingly, the screening results were similar between remote and onsite screening. Real-time screening of elementary school children with puretone audiometry revealed similar test sensitivity but slightly poorer test specificity values when compared to onsite screening (Lancaster et al., 2008). These applications, however, would seem best mediated through a store-and-forward (asynchronous) telehealth model but these "proof-of-concept" studies support the viability of remote screening.

Another important development for audiologic screening has been the use of a self-screening over the telephone or Internet as a store-and-forward (asynchronous) application. This type of screening has the inherent advantage of providing widespread access to hearing screening, which is especially important considering the aging world population, especially the current aging Baby Boomer generation (Swanepoel et al., 2013). It also has important limitations, however, that include a lack of absolute calibration of stimuli and control of environmental variables at the remote test site (ambient noise levels, variable transducer types, etc.). One way to overcome some of these limitations is to use a test that does not require absolute calibration but rather uses a relative signal-to-noise ratio. For this purpose, simple automated speech-in-noise tests have been developed for delivery over the telephone or Internet with normative signal-to-noise ratios to indicate a refer result. In the Netherlands, for example, the national hearing screening service is a triple-digit-in-noise test using an adaptive procedure that can be used reliably over the telephone or computer headset (Smits et al., 2006). In the future, these screening tests will also be available as mobile smartphone applications.

Diagnosis

Conducting diagnostic audiologic test procedures within a telehealth framework requires the sharing of information in a store-and-forward or real-time manner between the patient and audiology professional sites. The most common form of telehealth practice in audiology is probably something most audiologists have done at some point in time—to ask for a second opinion on a patient or test result. Although not commonly recognized as such, asking for an expert second opinion using an ICT medium such as the telephone or e-mail constitutes a form of real-time or store-and-forward tele-audiology.

In cases where information sharing is from a site without the specialist knowledge of an audiologist, but where there is access to audiologic equipment and ICT, an audiologist may provide services directly in real time or using store-and-forward methods (Table 36.4). Real-time remote services will require the audiologist to engage with the patient and to control the diagnostic test equipment remotely (see section on telehealth equipment). Diagnostic store-and-forward tele-audiology may require some degree of automation to conduct test procedures facilitated by nonspecialist personnel (telehealth facilitator or nurse). Alternatively, the telehealth facilitator should be trained to conduct the specific test procedure but this may not be possible in many cases (e.g., diagnostic puretone and speech audiometry, ABR).

Case History

Case histories can be taken quite easily in real time using technologies such as video-conferencing. Store-and-forward methods may also be utilized and could include completion of questionnaires and standardized forms online (e.g., tinnitus and hearing loss handicap inventories) prior to consultations.

TABLE 36.4

Summarizing Applications of Common Diagnostic Audiologic Procedures Using Telehealth

Diagnostic Procedure	Telehealth Modes Applicable	Automation	Populations
Case history	Store-and-forward or real time	Can be automated	All
Video-otoscopy	Store-and-forward	–	All
Tympanometry	Store-and-forward or real time	Automated	All
Acoustic reflexes	Store-and-forward or real time	Automated/ semiautomated	
Puretone audiometry	Store-and-forward or real time	Can be automated	Older children and adults
Speech audiometry	Real time	–	Older children and adults
Otoacoustic emissions	Store-and-forward or real time	Automated	All
Auditory brainstem response	Real time	–	All
Intraoperative monitoring	Real time	–	All
Balance testing	Real time	–	Older children and adults

Video-otoscopy

Video-otoscopy has been used for telehealth purposes to assist in establishing outer and middle-ear status. Video-otoscopic images can be e-mailed or uploaded to online servers for remote interpretation. Studies have confirmed the reliability of this technique in children and adults compared to face-to-face interpretations (Biagio et al., 2013; Patricoski et al., 2003). A recent study demonstrated that a nonspecialist telehealth facilitator could be trained to record video-otoscopy images comparable to an otolaryngologist for remote interpretation (Biagio et al., 2013). A follow-up study on children demonstrated similar findings and was the first to report using brief videoclips as opposed to still images (Biagio et al., in press). Videos have the added advantage of capturing more area of the ear canal and tympanic membrane from various angles and allow remote clinicians to pause and rewind to specific frames for detailed analyses.

Immittance

Current immittance equipment is largely automated, requiring only an ear canal seal for the test sequence to commence. Nonspecialist personnel could therefore be trained to acquire a tympanogram or even an automated acoustic reflex threshold sequence. If the results are recorded on a computer-based system, the findings may be uploaded directly to a server or e-mailed. Results from older systems that are not computer-based can be printed and faxed, scanned and e-mailed, or even photographed by a smartphone and sent to the remote site for interpretation. Immittance test findings are usually part of a larger test battery of results required for a thorough audiologic or otologic diagnosis.

Puretone Audiometry

In a systematic review of telehealth applications in audiology the majority of reports validated the use of puretone audiometry in real time to remote locations. The evidence demonstrates that diagnostic puretone audiometry can be conducted remotely on patients with the same accuracy as face-to-face testing (Swanepoel and Hall, 2010). Figure 36.2 provides a screenshot of a remote audiometry study conducted between Dallas, Texas, and Pretoria, South Africa (Swanepoel et al., 2010c). To date, no remote diagnostic puretone audiometry assessments have been reported on children apart from puretone audiometry screening on elementary school-aged children. Obvious challenges emerge when considering remote testing using conditioned play and visual reinforcement audiometry on young children. At present, the complete lack of research evidence in this regard leaves the validity of these measures for tele-audiology questionable. Developing alternative approaches and using well-trained facilitators may allow pediatric audiometry to be conducted on younger children but must be supported by validation research.

Automated puretone audiometry can also be used within a store-and-forward telehealth paradigm (Mahomed et al., 2013; Swanepoel et al., 2010b). A nonspecialist trained in setting up patients and providing the necessary instructions may facilitate automated diagnostic audiometry with the results forwarded to remote audiologists for interpretation and recommendations (Swanepoel et al., 2010a). A single report is available on the validity of automated puretone audiometry in children 4 to 8 years of age (Margolis et al., 2011). Findings indicated that automated puretone audiometry is possible in young children but a measure of test quality should be included to identify unreliable test findings. The system used included a validated measure of automated puretone audiometry reliability.

Speech Audiometry

Conducting speech audiometry through telehealth means is complicated because of the requirement to clearly hear patient responses. The audio quality on videoconferencing links may

not always be sufficient to clearly differentiate between similar sounding words. Alternative real-time method may be to have a trained listener at the patient site who can cross-check the patient responses. The only published tele-audiology study on speech audiometry to date used the Hearing-in-Noise-Test (HINT) (Nilsson et al., 1994), and despite possible confounding factors mentioned above, the results of the study were comparable to the face-to-face testing (Ribera 2005). This testing would require very good connectivity with little or no deterioration in audio transmission and no obvious time delay.

Alternative diagnostic speech audiometry test paradigms may be developed using various options such as closed set word lists or touchscreen response options to speech stimuli. It may even be possible to present a number of speech lists to patients with appropriate instruction and make high-quality audio recordings that are uploaded to secure servers where the lists may be scored asynchronously.

Auditory-Evoked Responses

A few validation studies have been reported applying tele-audiology to diagnostic OAE and ABR (Krumm et al., 2007; Swanepoel and Hall, 2010; Towers et al., 2005). OAE and ABR measurements taken remotely were comparable to those measured onsite. These test setups require a facilitator to correctly place probes (OAE) or transducers and electrodes (ABR). Using interactive videoconferencing allows the remote audiologist to instruct and guide the facilitator in preparing the patient for the test procedure. Auditory-evoked potential measures conducted in real time by expert audiologists on patients in underserved areas are appealing tele-audiology applications. This is especially true in light of the shortage of experienced pediatric audiologists and the increase in babies requiring diagnostic ABR testing following newborn hearing screening. Tele-audiology allows pediatric audiologists to assess patients in different counties, states, and even across continents. In Canada, this type of service has been used for several years to test babies in remote or underserved areas (Polovoy, 2008).

Intraoperative Monitoring

Intraoperative monitoring by audiologists may include several types of monitoring procedures, including electrocochleography, ABR measures during surgical excision of acoustic schwannomas, and other surgical procedures where the cochlea or neural pathway of the auditory system may be involved. During cochlear implant surgery, intraoperative device testing and patient responses to electrical stimulation are widely practiced. Device checks include determining the integrity of the implant and its electrodes, and responses to electrical stimulation may include stapedial reflex threshold, neural response telemetry, and electrically evoked ABR (Shapiro et al., 2008).

Intraoperative monitoring services for audiologists are characteristically time consuming with traveling involved and preparation and waiting for the surgery. Remote intraoperative monitoring may increase time efficiency of audiologic resources. Technicians can set up the monitoring equipment and link the devices to the Internet, where an audiologist may take control of the equipment from his or her office and conduct and monitor the specific intraoperative monitoring procedures. A study compared onsite audiologic monitoring and remote monitoring during cochlear implant surgery for several patients (Shapiro et al., 2008). These authors conclude that remote intraoperative monitoring during cochlear implant surgery is feasible, time saving, practical, and efficient (Shapiro et al., 2008).

Balance Assessment

One study has reported using telehealth for vestibular assessment. It involved a single case study with a remote consultation for a patient with benign positional vertigo using two-way videoconferencing and the use of cameras to view the patient's eye movements remotely (Virre et al., 1997). Real-time consultations are certainly possible and equipment-based measures could be conducted remotely, but would require a competently trained facilitator to set up patients for assessments and to facilitate some of the physical maneuvers. Caution must be taken to ensure patients are not put at an increased risk of injury because the assessment is conducted remotely.

Intervention

Audiologic intervention covers various practices that may include prescription, fitting and verification of hearing aid and assistive listening devices, cochlear implant mapping, counseling, vestibular rehabilitation, tinnitus treatment, and aural rehabilitation. In some cases, audiologists may also provide early intervention services to children with hearing loss and their families. Telehealth provides unique opportunities to provide many of these services in ways that may be more cost effective, less time consuming, and offering a greater reach to underserved areas (Swanepoel and Hall, 2010).

Hearing Aids and Assistive Devices

Since hearing aids are now almost always digital and programmed by computer-operated software to be patient specific the application of telehealth for remote programming is likely a future development. There are different levels, however, at which telehealth may support the continuum of hearing aid selection, fitting, verification, counseling, and troubleshooting. It may be used across all of these aspects or only for one or two. Current reports have only utilized telehealth means for one or two at time (Campos and Ferrari, 2012; Pearce et al., 2009; Wesendahl, 2003).

An important concern with hearing aid fittings conducted through telehealth means is taking the initial earmold impression. Someone who is qualified and sufficiently trained to take an impression without risks to the patient should be used. Telehealth may be used as a quality control measure to supervise remote earmold impressions in real time. Each context must apply the guidelines and prescriptions of the responsible professional bodies in regard to this practice. Using noncustom earmold hearing aids solves a lot of the issues with quality control and risks related to custom earmolds within a telehealth framework. The possibility of completing a fitting shortly after diagnosis is a further advantage of a noncustom earmold. Unfortunately, not all hearing losses can be accommodated with noncustom earmolds but improvements in technology for feedback reduction are expanding the fitting ranges of these devices (McPherson, 2012).

Remote hearing aid fitting, verification, and subsequent outcomes have been demonstrated to be comparable to a control group of adults who received the same services in a face-to-face setup (Campos and Ferrari, 2012). The remote audiologist conducted hearing aid programming, real-ear verification, and patient education using remote desktop sharing software and videoconferencing equipment with the assistance of an onsite facilitator without experience in hearing aid fitting (Campos and Ferrari, 2012). Outcomes were measured a month later using the HINT and the International Outcomes Inventory for Hearing Aids (IOI-HA) (Cox and Alexander, 2002). No significant differences were found between remote and face-to-face consultation time, real-ear measures matching respective targets, and outcomes in relation to hearing aid use in hours, or between HINT results and IOI-HA results. In an earlier study, remote verification of hearing aid fitting was also verified to be comparable to face-to-face real-ear verification procedures (Ferrari and Bernardez-Braga, 2009). A multiple case study report from Australia confirms these applications as practical and beneficial with a series of patients presented that were assisted remotely with hearing aid fittings, including real-ear measures for verifications, hearing aid program changes, informational counseling, and hearing aid troubleshooting (Pearce et al., 2009).

Although no reports are available on using telehealth methods to assist with provision of assistive listening devices such as FM systems, the applications demonstrated for hearing aids presume that these could also be tailored for tele-audiology provision.

Cochlear Implants

Cochlear implants are specialized devices that are implanted and managed by professional teams, usually located in cities where advanced and highly specialized medical and audiologic support is available. This means that after implantation, patients who reside long distances from these cochlear implant centers of excellence have to travel regularly to have their implant mapped. Tele-audiology offers a way to provide some of these follow-up services at remote sites with the use of desktop sharing software and videoconferencing.

Current telehealth evidence indicates that there is no significant difference between remote and onsite electrode-specific measure including impedance, ECAP thresholds, psychophysical thresholds, and map levels (Hughes et al., 2012; McElveen et al., 2010; Wesarg et al., 2010; Eikelboom et al., 2014). As a result, remote cochlear implant mapping can be done as a replacement for face-to-face mapping service provided adequate onsite personnel and technology support is offered (Hughes et al., 2012; Ramos et al., 2009). Furthermore, the remote session duration was only slightly higher than the face-to-face sessions on average. Significantly poorer speech perception scores were recorded for the remote test session but were attributed to the influence of environmental noise since the remote site did not have a sound booth (Hughes et al., 2012). The measurements did not require a facilitator with specialized knowledge of cochlear implants or audiology, and patients were generally comfortable connecting the programming cables to the processors (Hughes et al., 2010).

Some of the challenges include incompatibilities between different generation software, hardware, and speech processors. Furthermore, the communication with patients during the remote session was challenging at times, especially when the processor was connected directly and the cochlear implant microphone deactivated as a result. Videoconferencing can also be difficult to facilitate effective speech reading if there is some compromise in the connectivity (Hughes et al., 2012).

Counseling and Rehabilitation

Since videoconferencing is able to connect individuals with live audio and video feeds, real-time counseling and rehabilitation can be conducted without the need for expensive equipment. The counseling and rehabilitation can also occur in a store-and-forward paradigm using something as simple as e-mail exchanges between a patient and a professional. The use of an Internet-based counseling program for new hearing aid users through daily e-mail interchanges for 1 month indicated that it was a powerful medium for observing changes in behavior and perception of patients and allowed for timely responses to concerns (Laplante-Levesque et al., 2006). Utilizing an online education program for adult hearing aid users, Thoren et al. (2011) demonstrated that the Internet can be used effectively to reduce residual problems and that online discussion forums could be useful for rehabilitation. In work currently underway, researchers are developing Internet-based rehabilitation methods for adults with hearing loss using a patient-journey model (Manchaiah et al., 2013). If this type of program proves effective, it may offer ways of providing services that are easily accessible in addition to ensuring cost and time savings.

Internet-based treatment has also been investigated extensively for tinnitus patients (Andersson and Kaldo, 2004; Kaldo et al., 2008). The treatment program consisted of a web-based self-help manual that applied cognitive behavioral principles. Patients submitted weekly diaries to follow progress and give feedback. In comparison to conventional cognitive behavioral therapy for the control group, the treatment group improved to a significantly greater extent than the control group but also had a much higher dropout rate (Andersson et al., 2002). In a follow-up nonrandomized clinical trial, the Internet-based treatment demonstrated significant reductions in distress associated with tinnitus that persisted at 3 months follow-up (Kaldo-Sandström et al., 2004). In a follow-up randomized control trial with improvements to the Internet-based therapy, the treatment groups (Internet-based vs. group cognitive therapy) yielded significant positive results with no significant differences on main outcome measures with relatively stable results persisting at 1-year follow-up. The attrition rate was lower than for previous Internet treatments for tinnitus and was almost twice as cost effective as conventional group treatment (Kaldo et al., 2008).

Tele-intervention

Early intervention services to infants and young children with hearing loss are essential to improve acquisition of developmental milestones, empower families to meet the needs of their child, and minimize the need for special education services (Cason et al., 2012). There is a general shortage of early intervention personnel, especially for the increasing number of families with children who have hearing loss (McCarthy et al., 2010). Additionally, many families live in remote areas; using videoconferencing equipment allows interventions to connect to families in their homes to provide the necessary services (Houston et al., 2013).

Tele-intervention provides intervention at home, which is a great convenience to the family. For example, consider the disruptions, starting with packing up the patient and siblings for a long drive. Other advantages include the fewer cancellations in the event of a minor family illness and the capacity to record sessions for both the family and interventionist to review. During videoconferencing, the caregivers are the ones interacting primarily with the child, not the clinician. The professional is appropriately functioning as a "guide on the side," supporting the caregivers as the primary facilitator of the child's communication, language, and behavior (Houston et al., 2013).

There has been an emerging increase in tele-intervention programs for infants with hearing loss in several countries (Davis et al., 2012; Houston et al., 2013; Richardson, 2012). Initial results have demonstrated positive acceptance by families and interventionists with recognition of the significant benefits offered by tele-intervention (Constantinescu, 2012).

Continued Professional Education

ICT is a powerful tool to enable professionals to obtain remote education. This may be achieved through several different avenues. Online lectures or courses on a variety of audiologic topics are already available from different providers as either live video streaming or offline downloading of prerecorded presentations, designed to facilitate long-distance continued development. Other educational tools include online forums where professionals can interact and share information about cases in an interactive manner. Experienced audiologists may also mentor less experienced colleagues through ICT by providing second opinions or even by using videoconferencing to observe specific procedures. An example may be an experienced audiologist having access to the desktop of the inexperienced colleague while they conduct an ABR assessment. As the experienced colleague observes the recordings, they could comment via an audio link and discuss certain aspects of the auditory-evoked potential software package. This concept has been coined as telementoring by other medical disciplines. Apart from professional education, audiologists may also use ICT to facilitate ongoing training and monitoring of telehealth facilitators, assistive personnel, or other healthcare providers.

CLINICIAN AND PATIENT PERCEPTIONS OF TELE-AUDIOLOGY

An important and relatively unexplored aspect of tele-audiology is the perceptions of both patients and clinicians regarding the provision of services through telehealth (Swanepoel and Hall, 2010). If patients and clinicians are not willing to participate in telehealth services, the technologies and the services will not be effective no matter how advanced the technology.

A common fear among clinicians and patients is the perceived challenge to establish a meaningful clinical relationship through telehealth means (e.g., videoconferencing) as opposed to a face-to-face consultation (Eikelboom and Atlas, 2005). However, many telehealth services include initial contact with patients through face-to-face consultations and following up via telehealth means, which will allay much of that fear. Early reports indicate that perceptions of patients and clinicians who have experienced services via telehealth are all positive. In a study on remote cochlear implant mapping, positive patient experiences on par with face-to-face assessments were reported (Ramos et al., 2009). Perceptions of patients who underwent an asynchronous online tinnitus treatment program also demonstrated similar perceived benefits to those who had face-to-face treatment (Kaldo et al., 2008; Kaldo-Sandström et al., 2004). A study of clinician and caregiver perceptions of tele-intervention for children with hearing loss indicated that that all parents were comfortable and

all interventionists were satisfied with the tele-intervention program (Constantinescu, 2012).

Future studies should be careful to investigate patient and clinician perceptions of individuals who have experienced tele-audiology across a range of services. As more tele-audiology programs emerge both patient and clinician perceptions should be documented to improve these services (Swanepoel and Hall, 2010).

FUTURE OF TELE-AUDIOLOGY

In keeping with the rapid pace of technologic developments, telehealth is a dynamic and rapidly changing healthcare delivery medium. The future of tele-audiology is likely to follow the trends in general technologic developments. This is reflected by the continuous emergence of new terminology to describe different forms of healthcare provision using ICTs (Fatehi and Wootton, 2012). One such area of current interest and rapid growth and development is mobile health (mHealth), often seen as a subset of eHealth but relating to the use of mobile phone technologies to promote, provide, and monitor healthcare services (Kelly and Minges, 2012). This field is particularly appealing with the widespread penetration of mobile phones and cellular network reception globally but particularly in underserved developing countries (Kelly and Minges, 2012). A 2013 review paper indicated that there are more than 15,000 healthcare applications for smartphones (Fiordelli et al., 2013). At present, the evidence in support of these applications is still largely absent but governments are increasingly employing mHealth for public healthcare initiatives (Kelly and Minges, 2012). In hearing health care, there are already numerous smartphone applications available to conduct hearing assessments (e.g., puretone audiometry, speech audiometry) and measure ambient noise levels. Smart phone apps now interact directly with hearing aids and can even function as a hearing aid. Although there are significant challenges when calibration is not controlled, these technologies have the potential to serve as access points for additional services (Foulad et al., 2013; Handzel et al., 2013; Szudek et al., 2012).

As tele-audiology services are validated and cost-effectiveness benefits are demonstrated, it is expected that these services will become integrated components of current ear and hearing healthcare service-delivery models. Major obstacles to increasing these services remain the challenges related to reimbursement and cross-state or even cross-country licensure. On the legislative and regulatory level, much work remains to be done to find compatible ways in which these aspects can be accommodated while ensuring best practice service delivery.

An area of current development in the broader field of audiologic service delivery that is sometimes wrongly associated with telehealth is patient acquisition of hearing aids over the Internet. Internet hearing aid sales are not a telehealth service, since there is no health professional taking responsibility for the patient as is required through telehealth service delivery. With the proliferation of Internet and mobile phone–based services, audiologists must consistently promote best practice services and the validation of new technologies. Telehealth services may utilize the Internet, but the audiologist is accountable for the service provided. The profession of audiology should not shun new developments incorporating ICT, but should instead lead the way in evaluating these developments.

Audiology has always been a profession that has relied heavily on technology to diagnose and treat patients with hearing loss. As technologies change and advance more rapidly than ever before, audiologic practices must be grounded firmly on research evidence ensuring best practices for the patients we serve.

FOOD FOR THOUGHT

1. The fact that clinical audiology is heavily reliant on technology makes it uniquely suited to telehealth. This reliance on technology may also impact the patient–professional relationship. Consider this possible impact. Is it possible to develop positive patient relationships via tele-audiology? How?
2. Consider whether the automation of audiologic test procedures (typical in asynchronous telehealth services) such as puretone audiometry is a threat or asset to the profession.
3. Consider what population groups may be particularly difficult to serve through tele-audiology and what adaptations may be considered in these cases.

KEY REFERENCES

thePoint A full list of references for this chapter can be found at http://thePoint.lww.com. Below are the key references for this chapter.

American Speech-Language-Hearing Association. (2013c) State provisions update for telepractice. Available online at: http://www.asha.org/Practice-Portal/Professional-Issues/Telepractice/State-Provisions-Update-for-Telepractice (accessed September 17, 2013).

American Telemedicine Association. (2013) What is telemedicine? Available online at: http://www.americantelemed.org/learn (accessed September 17, 2013).

Andersson G, Kaldo V. (2004) Internet-based cognitive behavioral therapy for tinnitus. *J Clin Psychol.* 60, 171–178.

Andersson G, Strömgren T, Ström L, Lyttkens L. (2002) Randomized controlled trial of Internet-based cognitive behavior therapy for distress associated with tinnitus. *Psychosom Med.* 64, 810–816.

Australian Government Department of Health and Aging. (2013) Specialist video consultations under Medicare. Available online at: http://www.mbsonline.gov.au/internet/mbsonline/publishing.nsf/Content/mbsonline-telehealth-landing.htm (accessed September 17, 2013).

Biagio L, Swanepoel D, Laurent C, Lundberg T. (2014) Video-otoscopy recordings for diagnosis of childhood ear disease using telehealth at primary health care level. *Journal of Telemedicine and Telecare*. (in press).

Campos PD, Ferrari DV. (2012) Teleaudiology: evaluation of teleconsultation efficacy for hearing aid fitting. *J Soc Bras Fonoaudiol.* 24 (4), 301–308.

Cason J, Behl D, Ringwalt S. (2012) Overview of states' use of telehealth for the delivery of early intervention (IDEA Part C) services. *Int J Telerehabil.* 4 (2), 39–46.

Clark JL, Swanepoel DW. (2014) Technology for hearing loss: as we know it and as we dream it. *Disabil Rehabil Assist Technol.* (in press).

Cox R, Alexander G. (2002) The International Outcome Inventory for Hearing Aids (IOI-HA): psychometric properties of the English version. *Int J Audiol.* 41 (1), 30–35.

Davis A, Hopkins T, Abrahams Y. (2012) Maximizing the impact of telepractice through a multifaceted service delivery model at the Shepherd Centre, Australia. *Volta Rev.* 112, 383–391.

Eikelboom RH, Jayakody DMP, Swanepoel DW, Chang S, Atlas MD. Validation of remote mapping of cochlear implants. *J Telemed Telecare*. (in press).

Fiordelli M, Diviani N, Schulz PJ. (2013) Mapping mHealth research: a decade of evolution. *J Med Internet Res.* 15 (5), e95.

Goulios H, Patuzzi RB. (2008) Audiology education and practice from an international perspective. *Int J Audiol.* 47, 647–664.

Houston KT, Behl D, Walters KZ. (2013) Using telepractice to improve outcomes for children with hearing loss and their families. Available online at: http://www.infanthearing.org/ehdi-ebook/2013_ebook/18Chapter17UsingTelepractive2013.pdf

Internet World Stats. (2012) Internet usage statistics. Available online at: http://www.internetworldstats.com/stats.htm (accessed September 17, 2013).

Kaldo V, Levin S, Widarsson J, Buhrman M, Larsen HC, Andersson G. (2008) Internet versus group cognitive-behavioral treatment of distress associated with tinnitus: a randomized control trial. *Behav Ther.* 39, 348–359.

Kaldo-Sandström V, Larsen HC, Andersson G. (2004) Internet-based cognitive-behavioral self-help treatment of tinnitus: clinical effectiveness and predictors of outcome. *Am J Audiol.* 13, 185–192.

Kelly T, Minges M. (2012) *Executive Summary. Maximizing Mobile.* Washington, DC: International Bank for Reconstruction and Development/The World Bank. Available online at: http://siteresources.worldbank.org/EXTINFORMATIONANDCOMMUNICATIONANDTECHNOLOGIES/Resources/IC4D-2012-Report.pdf (accessed September 17, 2013).

Manchaiah VK, Stephens D, Andersson G, Rönnberg J, Lunner T. (2013) Use of the 'patient journey' model in the Internet-based pre-fitting counseling of a person with hearing disability: study protocol for a randomized controlled trial. *Trials.* 14, 25.

Margolis RH, Frisina R, Walton JP. (2011) AMTAS(®): automated method for testing auditory sensitivity: II. Air conduction audiograms in children and adults. *Int J Audiol.* 50, 434–439.

Margolis RH, Glasberg BR, Creeke S, Moore BC. (2010) AMTAS: automated method for testing auditory sensitivity: validation studies. *Int J Audiol.* 49 (3), 185–194.

Margolis RH, Morgan DE. (2008) Automated pure-tone audiometry: an analysis of capacity, need, and benefit. *Am J Audiol.* 17, 109–113.

McPherson B. (2012) Innovative technology in hearing health care: matching needs in the developing world. *Trends Amplif.* 15 (4), 209–214.

Nilsson N, Soli S, Sullivan J. (1994) Development of the hearing in noise test for the measurement of speech reception thresholds in quiet and in noise. *J Acoust Soc Am.* 95, 1085–1099.

Patricoski C, Kokesh J, Ferguson AS, Koller K, Zwack G, Provost E, et al. (2003) A comparison of in-person examination and video otoscope imaging for tympanostomy tube follow-up. *Telemed J E Health.* 9, 331–344.

Polovoy C. (2008) Audiology telepractice overcomes inaccessibility. *ASHA Lead.* Available online at: http://www.asha.org/Publications/leader/2008/080617/080617c/ (accessed May 31, 2013).

Ribera J. (2005) Interjudge reliability and validation of telehealth applications of the Hearing in Noise Test. *Semin Hear.* 26, 13–18.

Richardson LL. (2012) Children's hearing and speech centre-telepractice programs. *Volta Rev.* 112, 429–433.

Stevens G, Flaxman S, Brunskill E, Mascarenhas M, Mathers CD, Finucane M. (2013) Global and regional hearing impairment prevalence: an analysis of 42 studies in 29 countries. *Eur J Public Health.* 23 (1), 146–152.

Swanepoel D, Eikelboom R, Hunter ML, Friedland PL, Atlas MD. (2013a) Self-reported hearing loss in Baby Boomers from the Busselton Healthy Aging Study – audiometric correspondence and predictive value. *J Am Acad Audiol.* 24 (6), 514–521.

Swanepoel D, Mngemane S, Molemong S, Mkwanazi H, Tutshini S. (2010b) Hearing assessment – reliability, accuracy and efficiency of automated audiometry. *Telemed J E Health.* 16 (5), 557–563.

Swanepoel D, Myburgh HC, Howe DM, Mahomed F, Eikelboom RH. (2014) Smartphone hearing screening with integrated quality control and data management. *Int J Audiol*. (in press).

Towers AD, Pisa J, Froelich TM, Krumm M. (2005) The reliability of click-evoked and frequency-specific auditory brainstem response testing using telehealth technology. *Semin Hear.* 26, 19–25.

Virre E, Warner D, Balch D, Nelson JR. (1997) Remote medical consultation for vestibular disorders: technological solutions and case report. *Telemed J.* 3, 53–58.

Wesendahl T. (2003) Hearing aid fitting: application of telemedicine in audiology. *Int Tinnitus J.* 9, 56–58.

Wootton R, Ho K, Patil NG, Scott RE. (2009) Introduction. In: Wootton R, Patil NG, Scott RE, Ho K, eds. *Telehealth in the Developing World.* London: Royal Society of Medicine Press Ltd; pp 3–8.

World Health Organization. (2006) *Primary Ear and Hearing Care Training Manuals.* Geneva: Author. Available online at: http://www.who.int/pbd/deafness/activities/hearing_care/en/index.html (accessed June 11, 2009).

World Health Organization. (2011) *Mortality Data.* Geneva: Author. Available online at: www.who.int/healthinfo/statistics/mortality/en/

World Health Organization. (2013) *Millions of people in the world have a hearing loss than can be treated or prevented.* Available online at: http://www.who.int/pbd/deafness/news/MillionslivewithhearingIoss.pdf (accessed September 17, 2013).

SECTION IV

Management of Hearing Disorders

CHAPTER 37

Room Acoustics and Auditory Rehabilitation Technology

Joseph Smaldino, Brian Kreisman, Andrew John, and Lindsay Bondurant

INTRODUCTION

There is ample evidence that sensory/neural hearing loss (SNHL) causes communicative difficulty, particularly in listening environments that are noisy and/or reverberant (Needleman and Crandell, 1996). Because of the deleterious effects of SNHL on communication, individuals with hearing loss may exhibit reduced psychosocial function, including increased feelings of frustration, anger, fear, isolation, loneliness, and depression (Vesterager and Salomon, 1991). In addition, perhaps as the result of reduced psychosocial functioning, persons with SNHL tend to exhibit a higher incidence of health-related difficulties, including hypertension, ischemic heart disease, arrhythmias, osteoarthritis, and reductions in activity level, quality of life, and physical mobility (Mulrow et al., 1990).

There exists a broad range of potential disruptions of communicative and psychosocial function as well as health-related quality-of-life (HRQOL) issues that can be caused by hearing loss. It is therefore important that the audiologist consider intervention options in addition to hearing aids. In many cases, hearing aids alone may not be sufficient to restore effective communication, particularly if the patient is communicating in an environment with excessive background noise. In these cases, other assistive listening technologies, communication strategies, and auditory rehabilitation training must also be considered and used in conjunction with the hearing aids. It is reasonable to speculate that, if communication function is improved, then the negative psychosocial and/or HRQOL effects of reduced communication can be minimized. With these considerations in mind, the purpose of this chapter is to discuss rehabilitative technologies and communication strategies that have been shown to improve communicative efficiency in listeners with SNHL (and individuals with normal hearing who have difficulty processing auditory information) within the following environments: (1) Room settings that are commonly used for communication, such as churches, restaurants, classrooms, meeting/conference rooms, and theaters; (2) face-to-face situations; (3) telecommunications; and (4) broadcast media (radio, television [TV], etc.). In addition, this chapter will address signal/alerting technologies that can assist individuals with hearing loss in the awareness of sounds within their listening environment. The term *hearing assistance technology* (*HAT)* will be used in this chapter, rather than the older term *assistive listening device* (*ALD),* to discuss technologies that improve communicative status through the transmission of an amplified auditory, tactile, or visual signal to the listener since many of these technologies are not limited to improvement of listening.

IMPROVING COMMUNICATION IN ROOM SETTINGS

Perhaps the most persistent complaint heard from listeners with SNHL is difficulty communicating in places used for verbal communication. Such environments include churches, restaurants, classrooms, therapy rooms, shopping establishments, meeting/conference rooms, and theaters. To understand why these difficulties occur, it is important that the audiologist has a basic understanding of acoustic variables that can interfere with the perception of speech. These acoustic variables include (1) background noise; (2) speech signal level compared to background noise level; (3) reverberation time (RT); (4) distance between the talker and the listener; and (5) interactions among these variables.

Background Room Noise

Background noise refers to any auditory disturbance within the room that interferes with what a listener wants to hear (Smaldino and Flexer, 2012). A common way of measuring noise in a room is with a sound level meter (SLM). An SLM can range from a compact, inexpensive, battery-operated unit designed to measure sound amplitude to a computer-based device that can measure and record numerous acoustic properties of a signal. SLMs are classified according to standards set forth in American National Standards Institute (ANSI) S1.14-1998 (R2013). Type I meters meet the most precise standards, type II are general purpose, and type III are for hobby use. Detailed measurement of room noise requires, at minimum, a type II (and preferably a type I) SLM. Many SLMs incorporate weighting filter networks. The A-weighting network is designed to simulate the sensitivity of the average human ear under conditions of low

sound loudness (40 phons), the B-weighting simulates loud sounds (70 phons), and the C-weighting approximates how the ear would respond to very loud sounds. The convention for room measurements is the use of the A-weighting network. Unfortunately, the same single number obtained from a sound pressure measurement performed with the A-weighting scale can be obtained from a variety of very different sound spectra. Thus, a more accurate and complete way to measure room noise is to do a spectral analysis of the noise instead of attempting to use a single descriptor.

Noise criteria curves (NCCs) are one way to measure the frequency content of background noise in a room (as described in ANSI 12.2-2008). NCCs are a family of frequency and intensity curves based on the use of one-third octave-band sound pressure levels (SPLs). The NCC rating of a space is determined by plotting the SPLs within each frequency band relative to established NCC. Whenever possible, it is recommended that ambient noise levels in classrooms be measured using NCC measures since this procedure gives the examiner a more comprehensive assessment of the spectral characteristics of the noise.

Noise within an enclosure can come from several possible sources, including *external* sources (noise generated from outside the building), *internal* sources (noise originating from within the building, but outside the room), and *room* sources (noise that is generated within the room). High background noise levels have been measured in many enclosures including classrooms, professional office spaces, and residences (Bess et al., 1986; Smaldino et al., 2007). Crandell and Smaldino (1995) reported that background noise levels in 32 unoccupied classroom settings were 51 dBA and 67 dBC. More recently, studies have reported noise levels as high as 64 to 72 dBA in classrooms of schools as geographically disparate as the United States, Australia, and Hong Kong (see John and Kreisman, 2012, for a review). As will be discussed in a later section, such high levels of background noise can impair speech perception of not only listeners with SNHL, but also many with normal hearing sensitivity.

Background noise in a room can compromise speech perception by masking the acoustic and linguistic cues available in the message. Generally speaking, background noises in a room mask the weaker transient consonant phonemes more than the longer and more intense vowels (typically 10 to 15 dB more intense than consonants). A reduction of consonant information can have a significant impact on speech perception because approximately 80% to 90% of the acoustic information important for speech perception comes from the consonants (French and Steinberg, 1947). The extent to which speech is masked by background noise is influenced by a number of factors, including (1) the long-term acoustic spectrum of the noise; (2) the average intensity of the noise compared to the intensity of speech; and (3) fluctuations in the intensity of the noise over time. Often the most important factor for accurate speech perception is not the overall level of the background noise, but rather the relationship between the level of the signal as a function of frequency and the level of the background noise as a function of frequency. This relationship is often simplified and referenced as the signal-to-noise ratio (SNR). Because the decibel is logarithmic, SNR can be stated simply as a difference between the overall level of the signal and the level of the noise. For example, if a speech signal is presented at 70 dB SPL and a noise is 60 dB SPL, the SNR is +10 dB. Because of high background noise levels, diminished SNRs have been reported in many communication settings. Pearsons et al. (1977) reported that average SNRs were +9 to +14 dB in urban and suburban residential settings, respectively. In outdoor settings, SNRs decreased to approximately +5 to +8 dB. In department store settings, the average SNR was +7 dB, whereas transportation settings yielded an average SNR of −2 dB. Plomp (1978) reported that the average SNR found at cocktail parties ranged from +1 to −2 dB. In classroom environments, the range of SNRs has been reported to be from +5 to −7 dB (Smaldino and Flexer, 2012).

Speech perception is generally greatest at favorable SNRs and decreases as the SNR of the listening environment is reduced (Finitzo-Hieber and Tillman, 1978; Smaldino and Flexer, 2012). In general, speech perception ability in adults with normal hearing is not severely reduced until the SNR reaches 0 dB. However, this is not the case for listeners with SNHL. To obtain perception scores comparable to those of normal hearers, listeners with SNHL require the SNR to be improved by 4 to 12 dB; an additional 3 to 6 dB is required in rooms with moderate levels of reverberation (Johnson, 2000; Moore, 1997).

Although a number of acoustic, linguistic, and articulatory factors influence the determination of appropriate SNRs in a room, the literature suggests that, for young listeners with SNHL, the SNRs in communication environments should exceed +15 dB (Bradley and Sato, 2008; Finitzo-Hieber and Tillman, 1978). To accomplish this SNR, unoccupied room noise should not exceed 30 to 35 dBA (ANSI S12.6-2002 [R2010]). The recommendation of a +15 dB SNR is based on the finding that the speech perception of listeners with hearing loss tends to remain relatively constant at SNRs in excess of +15 dB but deteriorates at poorer SNRs. Moreover, when the SNR decreases to below +15 dB, those with hearing loss tend to expend so much attentional effort in listening to the message that they often prefer to communicate through other modalities. In addition to listeners with SNHL, some children with "normal" hearing sensitivity have greater than normal perceptual difficulties in noise and/or reverberation (Bess, 1985; Nabelek and Nabelek, 1994). A list of populations that may or may not exhibit hearing loss but often find it difficult to listen and learn is presented in Table 37.1. A prominent feature of these populations is that they all have a developmentally delayed, incomplete, or distorted knowledge of language. Because of their language deficit, these individuals cannot always use the structure of language to fill in or predict

TABLE 37.1

Populations that Find it Difficult to "Listen and Learn"

- Young children (<15 y old)
- History of recurrent otitis media
- Language disorder
- Articulation disorder
- Dyslexia or other reading disorders
- Learning disabilities
- Nonnative English
- Central auditory processing deficit
- Developmental delays
- Attentional deficits

Source: Adapted from Crandell C, Smaldino J, Flexer C. (2005) *Sound Field Amplification: Applications to Speech Perception and Classroom Acoustics*. Clifton Park, NY: Thompson Delmar Learning.

speech information when the information is distorted or inaudible. Because of the important relationship between the quality of the acoustic signal and language development, a favorable SNR is widely recommended for children in the developmental stages of language acquisition as well as children with language knowledge deficits. The importance of a favorable SNR in the classroom was highlighted in the ANSI standard S12.6-2002 (R2010) entitled "Acoustical Performance Criteria, Design Requirements and Guidelines for Classrooms," which stipulates an unoccupied classroom background noise level for permanent classroom structures of no more than 35 dBA. Applying the ANSI classroom acoustics standard, Knecht et al. (2002) found that most of the 32 elementary grade classrooms they studied exceeded the recommended background noise level of 35 dBA.

Reverberation

Another factor impacting on speech perception in enclosed settings is reverberation. Reverberation refers to the prolongation or persistence of sound within an enclosure when sound waves reflect off hard surfaces (e.g., bare walls, ceilings, windows, floors). RT is often stated as the amount of time it takes for a sound, at a specific frequency, to decay 60 dB after termination of the signal. For example, if a 110-dB SPL signal at 1,000 Hz takes 1 second to decrease to 50 dB SPL, the RT of that enclosure at 1,000 Hz is 1 second. Generally, RT increases as room volume increases and decreases as the amount of absorptive material in the room increases. Specifically, reverberation is decreased when surfaces in the room have a large sound absorption coefficient, or alpha (α), which is calculated as the amount of sound energy absorbed by surfaces in the room divided by the total sound energy from the signal source. The α of a room varies with the thickness, porosity, and mounting configuration of materials in a room and the frequency of the signal (Siebein et al., 1997). Materials with an α less than 0.2 are considered to be sound reflective, whereas materials with an α greater than 0.2 are considered to be sound absorbent. For instance, a brick wall has an α ranging from 0.03 at 125 Hz to 0.07 at 4,000 Hz, whereas a carpeted concrete floor ranges from 0.02 at 125 Hz to 0.65 at 4,000 Hz. Use of absorbent materials can decrease noise by 3 to 8 dB (Siebein et al., 1997). Note that rooms with irregular shapes (e.g., oblong) often exhibit longer RTs than rooms with more traditional quadrilateral dimensions.

All rooms exhibit some degree of reverberation. Audiometric test booths usually exhibit RTs of approximately 0.2 second (Smaldino et al., 2007). Living rooms and offices often have RTs between 0.4 and 0.8 second (Nabelek and Nabelek, 1994). RTs for classrooms are usually reported to range from 0.4 to 1.2 seconds (Bradley, 1986; Smaldino and Flexer, 2012), whereas auditoriums, churches, and assembly halls may exhibit RTs in excess of 3.0 or 4.0 seconds (Nabelek and Nabelek, 1994; Siebein et al., 1997).

The presence of people in a room further affects RT. A room full of people will have an RT that is 0.05 to 0.1 second less than when it is empty (Boothroyd, 2005). RT can be (1) measured using commercially available, handheld, special-purpose reverberation meters that directly measure the decrease in intensity of a test signal as a function of time or (2) derived from the impulse response of the room. The impulse response methods require the introduction of controlled noise bursts to energize the acoustics of the room. The responses obtained from the bursts can be used to calculate nearly all standard acoustic performance measures, including reverberation.

Ideally, RT should be calculated at each octave interval from 63 to 8,000 Hz. More commonly, however, one low-, one middle-, and one high-frequency octave RT are calculated. For example, reports of RT are often obtained using the average of 500, 1,000, and 2,000 Hz (Siebein et al., 1997). Generally speaking, RT is longest at low frequencies (i.e., below 500 Hz), about equivalent in the range between 500 and 2,000 Hz, and shortest for higher frequencies (Nabelek, 1982). This is because of the fact that sound-absorptive materials have a greater α for higher frequency energy than for lower frequency energy.

Reverberation degrades speech perception through masking of the directly transmitted sounds (Nabelek, 1982). That is, reverberant speech energy reaches the listener some time after the corresponding direct sounds, overlapping subsequently presented speech sounds. This results in a "smearing" or masking of the directly transmitted speech signal. In other words, reverberation causes a prolongation of the spectral energy of the vowel sounds, which tends to mask succeeding consonant phonemes, particularly consonants in word final positions. The masking effectiveness of reverberation involving vowels is greater than for consonants since vowels exhibit greater overall power and are of longer duration than consonants.

Speech perception tends to decrease as the RT of the environment increases (Finitzo-Hieber and Tillman, 1978; Gelfand and Silman, 1979). Speech perception in adults with normal hearing is not compromised until the RT exceeds approximately 1.0 second (Gelfand and Silman, 1979). Listeners with SNHL, however, need considerably shorter RTs (0.4 to 0.5 second) for maximum speech perception (Finitzo-Hieber and Tillman, 1978). In addition, studies have indicated that the populations of "normal-hearing" children discussed previously have greater speech perception difficulties in reverberation than were traditionally suspected (see John and Kreisman, 2012, for a review of these studies).

Appropriate RTs (0.4 to 0.5 second) for persons with hearing loss are rarely achieved (Crandell and Smaldino, 1995). Crandell and Smaldino (1995) reported that only 9 of 32 classrooms (27%) examined in their study had RTs of 0.4 second or less. ANSI (2002; revised 2010) recommended an RT of 0.6 second for moderately sized permanent learning environments. Knecht et al. (2002), applying the ANSI criteria for reverberation, found that most of the 32 elementary grade classrooms they studied did not meet the 0.6-second maximum RT recommended in the standard.

Effects of Noise and Reverberation

Noise and reverberation do not occur separately in a room. In most enclosures, both noise and reverberation combine in a synergistic manner (Finitzo-Hieber and Tillman, 1978; Smaldino and Flexer, 2012). That is, the sum of the deleterious effects of noise and reverberation is greater than one would expect by simply adding these two variables together. It appears that this synergy occurs because reverberation fills in the temporal gaps in the noise. These gaps and modulations contribute significantly to speech perception for listeners with normal hearing (Hygge et al., 1992). However, reverberation eliminates these gaps, making the noise more steady state in nature and, thus, a more effective masker.

Similar to the findings obtained for noise and reverberation in isolation, research indicates that listeners with hearing loss and children with normal hearing (and even more so for children with processing-related deficits) experience greater speech perception difficulties in noise plus reverberation than do adults with normal hearing. Studies by Klatte et al. (2010) and Neuman et al. (2010) reported that elementary school–aged children show speech perception declines in the presence of typical classroom levels of noise and reverberation, and that those declines are significantly greater than those seen in adults. Notably, the participants in Klatte's study self-reported that the noise and reverberation in the classroom did not prevent them from hearing and understanding speech. This finding suggests that the interference to understanding caused by a poor acoustic environment may be underrated by children in an informal evaluation of the classroom.

TABLE 37.2

Mean Speech Recognition Scores (% Correct) by Children with Normal Hearing (*N* = 12) and Children with Sensory/Neural Hearing Loss (*N* = 12) for Monosyllabic Words Across Various Signal-to-Noise Ratios (SNRs) and Reverberation Times (RTs)

	Groups	
Testing Condition	**Normal Hearing (%)**	**Sensory/Neural Hearing Loss (%)**
RT = 0.0 s		
QUIET	94.5	83.0
+12 dB	89.2	70.0
+6 dB	79.7	59.5
0 dB	60.2	39.0
RT = 0.4 s		
QUIET	92.5	74.0
+12 dB	82.8	60.2
+6 dB	71.3	52.2
0 dB	47.7	27.8
RT = 1.2 s		
QUIET	76.5	45.0
+12 dB	68.8	41.2
+6 dB	54.2	27.0
0 dB	29.7	11.2

Source: Adapted from Finitzo-Hieber T, Tillman T. (1978) Room acoustics effects on monosyllabic word discrimination ability for normal and hearing-impaired children. *J Speech Hear Res*. 21, 440–458.

An example of the synergistic effects of noise and reverberation on the monosyllabic word perception of children with normal hearing and SNHL is shown in Table 37.2. Note that, even at the best SNR and RT conditions (SNR = +12 dB, RT = 0.4 second), 83% of children with normal hearing did not recognize speech perfectly, and children with hearing loss performed even more poorly (60%). As the SNR became poorer or as the RT lengthened, speech perception continued to decrease. In the listening condition of SNR = 0 dB and RT = 1.2 seconds, children with normal hearing achieved a score of 30% correct, whereas children with hearing loss achieved a score of only 11%. Each of the listening situations mentioned here have been reported in numerous classroom environments.

Distance

A final factor affecting speech perception in a room is the distance between the talker and the listener. Sound is distributed essentially in three different ways in a room (Figure 37.1). The "direct" sound is the sound that travels from the speaker to a listener without striking other surfaces in the room. This

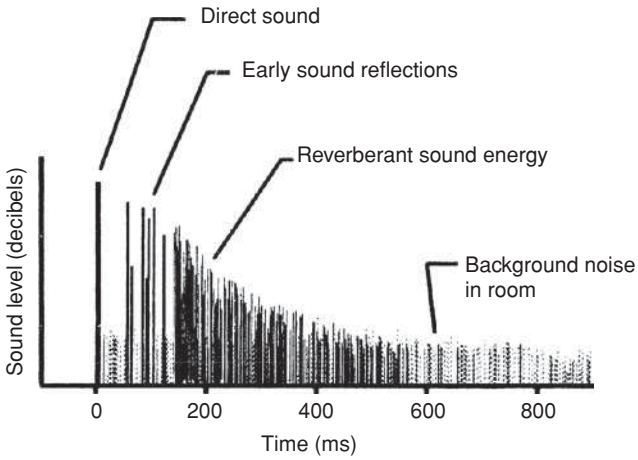


FIGURE 37.1 Components of sound (direct sound, early reflections, and late reflections or reverberation) within a room. (Siebein G, Crandell C, Gold M. (1997) Principles of classroom acoustics: reverberation. *Educ Audiol Monogr.* 5, 32–43, with permission.)

is usually the first sound to arrive at the listener since it travels the shortest path between the speaker and the listener. The power of the direct sound decreases with distance since acoustic energy spreads over a larger area as it travels from the source. Specifically, the direct sound decreases in accordance with the inverse square law, or approximately 6 dB SPL with every doubling of distance from the sound source. For example, if a speaker's voice is 80 dB SPL at 1 m, then it will be 74 dB at 2 m, 68 dB at 4 m, and so on. Because the direct sound energy decreases so quickly, only those listeners who are seated close to the speaker will actually hear the direct sound energy.

The auditory system allows the listener to orient to the sound source using information from the first direct sound energy received. This ability is variously known as the "Haas effect" (after Helmut Haas, the first researcher to describe it in 1951), the "precedence effect," or the "law of the first wavefront" (Haas, 1972). Using the precedence effect, the auditory system uses the physical characteristics of the head (two ears at different points in space with a barrier, the head, in between them) to determine the direction from which an incoming sound originates by analyzing the first sound energy to arrive at each ear. The precedence effect requires that the sound be discontinuous and be followed by reverberated sound energy no sooner than 1 ms and no later than about 40 ms after the first sound received (Moore, 1997). These conditions are usually met in reverberant rooms that are not very large, as sound reflecting off solid surfaces in a room typically reaches the ear first at around 30 to 50 ms after the direct sound.

Slightly greater distances from the speaker result in early sound reflections reaching the listener. Early sound reflections are those sound waves that arrive at a listener within very short time periods (approximately 50 ms) after the arrival of the direct sound. In a typical room, most of the early reflections strike minimal room surfaces on their path from speaker to listener. Early sound reflections are usually combined with the direct sound and may actually increase the perceived loudness and intelligibility of the sound (Bradley, 1986; Nabelek and Nabelek, 1994). This increase in loudness may actually improve speech perception in listeners with normal hearing.

Early reflections are dependent on the intensity level of the original sound, the directionality of its source (tendency of the source to radiate energy in a forward direction rather than equally in all directions), and the volume and RT of the room (Boothroyd, 2005). Early reflections are increased when the room is small and highly reverberant because these conditions allow the sound to reflect off many surfaces before decaying. A source with low directionality also produces more reflected energy; that is, when sound is radiated omnidirectionally, less energy will be directed at the listener, and more energy will need to strike room surfaces to reach that listener.

As a listener moves farther away from the speaker, reverberation begins to dominate the listening environment. As discussed earlier, reverberation consists of sound waves that strike multiple room surfaces as they move from the speaker to the listener. As they strike multiple room surfaces, the sounds generally decrease in loudness because of the increased path length traveled and the partial absorption that occurs with each reflection from the room surfaces. Late reflected energy is degraded more than early reflections because this energy has traveled a greater total distance and undergone more partial absorption (especially high frequencies) subsequent to striking many surfaces (Nabelek, 1982). In addition to the change in spectrum, late reflections interfere with speech perception by masking meaningful parts of the signal.

Distance from the speaker can affect speech perception directly (Leavitt and Flexer, 1991). Specifically, speech perception tends to decrease until the critical distance (i.e., the point at which the direct and reverberant sound energies are equal) of the room is reached. The critical distance in most rooms is approximately 2 to 6 m from the speaker. Beyond the critical distance, perception ability tends to remain essentially constant unless the room is very large (e.g., an auditorium), where speech perception may continue to decrease as a function of increased distance. In general, direct sound is the major component of sound level within the critical distance, whereas reverberation is the dominant component beyond the critical distance (Boothroyd, 2005; Bradley, 1986).

These findings suggest that speech perception can be improved by decreasing the distance between a speaker and listener only if it is within the "critical distance" of the room. This explains why the simple recommendation of preferential seating in the classroom is often inadequate to ensure an appropriate listening environment. That is, teachers often

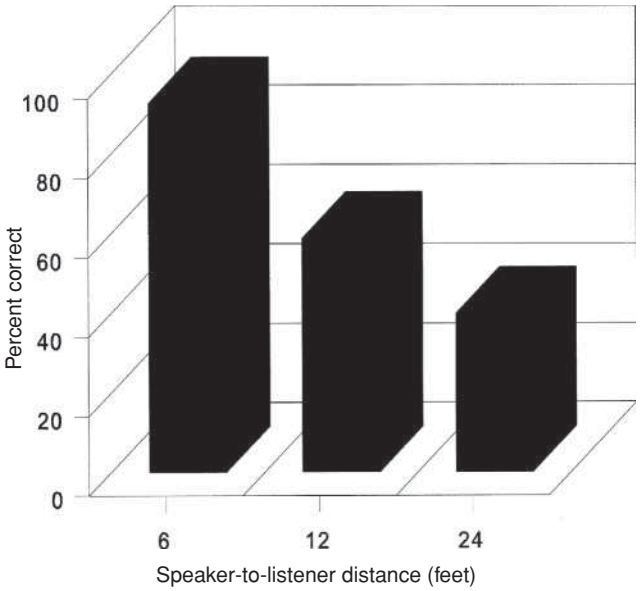


FIGURE 37.2 Mean speech recognition scores [% correct] of children with normal hearing in a typical classroom environment [signal-to-noise ratio = +6 dB; reverberation time = 0.6 second] as a function of speaker-to-listener distance. [Adapted from Crandell C, Smaldino J, Flexer C. [1995] *Sound Field FM Amplification: Theory and Practical Applications*. San Diego, CA: Singular Publishing Group, with permission.]

move around the room or turn their back to write on the blackboard, thus moving them out of the critical distance of the listener.

Crandell and Bess (1986) examined the effects of distance, noise, and reverberation on the speech perception of 20 children (5 to 7 years old) with normal hearing in a classroom environment. The classroom had an SNR of +6 dB and an RT of 0.45 second. Multitalker babble served as the noise competition. Sentences were presented to the children at distances of 6, 12, and 24 feet. Results from this investigation are shown in Figure 37.2. As can be seen, a significant decrease in speech perception occurred as the speaker–listener distance increased (i.e., 89%, 55%, and 36% were obtained at 6, 12, and 24 feet, respectively).

ACOUSTIC MODIFICATIONS OF THE ROOM

The first strategy for improving speech perception within an enclosure is acoustic modification of that environment. The most effective procedure for achieving this goal is through appropriate planning with contractors, school officials, architects, architectural engineers, audiologists, and/or teachers of individuals with hearing loss *before* the design and construction of the building. Recall that acoustic guidelines for populations with hearing loss indicate that (1) SNRs should be above +15 dB; (2) unoccupied noise levels should not exceed 30 to 35 dBA; and (3) RTs should not surpass 0.4 second. Unfortunately, as mentioned previously, such guidelines are rarely achieved in most listening environments. One reason for the discrepancy between acoustic guidelines and actual room settings is that rooms often exhibit minimal degrees of acoustic modification. Bess et al. (1986) reported that, although 100% of the classrooms examined had acoustic ceiling tiles, only 68% had carpeting and only 13% had draperies. None of the classrooms contained any form of acoustic furniture treatment such as glides on the chair legs, projector tables of a height to keep the noise above ear level, or smaller desks to reduce the amount of sound-reflective surface in the room. Findings of Crandell and Smaldino (1995) were even less favorable.

STATUS OF CLASSROOM ACOUSTIC STANDARD

ANSI standards are reviewed on a regular cycle. A working group considered revisions and debated public comments concerning the original 2002 classroom standard. As a result of these debates and discussions, it was decided that a separate part should be devoted to issues unique to portable classrooms.

The first part of the revised standard, American National Standard Acoustical Performance Criteria, Design Requirements, and Guidelines for Schools, Part 1: Permanent Schools (ANSI/ASA S12.60-2010), is a refined version of the 2002 standard. The major performance requirement for furnished but unoccupied classrooms is basically unchanged from the 2002 standard. The 1-hour average A-weighted background noise level cannot exceed 35 dB (55 dB if C-weighting is used) and for average-sized classrooms (with a volume less than or equal to 10,000 cubic feet) the reverberation time (RT60) cannot exceed 0.6 second (35/55 dBA/dBC) and 0.7 second if the volume is greater than 10,000 but less than or equal to 20,000 cubic feet). Among other changes are improvement of the requirements for exterior walls and roofs in noisy areas, consideration of activities close to classrooms, clarification of the definition of a "core learning space," addition of the limit of 45 dBA for sound in hallways, clarification and simplification of measurement procedures, and addition of the requirement that if an audio distribution system is deemed appropriate it should provide even coverage and be adjustable so as not to disturb adjacent classes.

The second part of the revised standard, American National Standard Acoustical Performance Criteria, Design Requirements, and Guidelines for Schools, Part 2: Relocatable Classroom Factors (ANSI/ASA S12.60-2010), phases in performance requirements for portable classrooms. The current standard sets a 41-dBA limit for background noise in unoccupied classrooms, which would be lowered to 38 dBA in 2013 and 35 dBA in 2017. Reverberation time (RT60) in unoccupied relocatable classrooms must not

exceed 0.5 second in classrooms with volumes of 10,000 cubic feet or less and 0.6 second in classrooms with volumes of 10,000 to 20,000 cubic feet. Both parts of the standard are available without charge from the Acoustical Society of America store (http://asastore.aip.org).

A third part is currently under development and will focus on control of noise from informational technology in the classroom.

As of this writing, compliance with the revised standards remains voluntary. Special effort was made during the crafting of the revision to include language so that the standard could be considered for incorporation into the International Building Code, which would make compliance mandatory for new school construction. Efforts to incorporate the standards into the 2012 building code failed; another opportunity will occur as the International Code Council will begin the 2015 building code development in 2013.

PERSONAL AND GROUP AMPLIFICATION SYSTEMS

Because of a lack of appreciation of the impact of acoustics on communication and additional construction costs, compliance with favorable classroom acoustic guidelines is often not a priority during construction. Because of this, even with subsequent room modifications, noise and reverberation levels often remain excessively high. As noted earlier, appropriate planning prior to the design and construction of a building is the most effective procedure for meeting acoustic guidelines. Because of inappropriate room acoustics, other methodologies, such as the use of assistive technologies, should be implemented. One well-recognized strategy for improving speech perception in rooms is through the use of personal or group amplification systems. Investigations of room amplification systems have shown that they can improve significantly speech perception, listening, attention, academic performance, and on-task behaviors (John and Kreisman, 2012). The goals of room amplification systems are to (1) maintain a high SNR with minimal reverberation at the listener's ears; (2) allow the signal to be modified to meet the acoustic needs of the individual(s); (3) provide wide frequency amplification with a minimal degree of distortion; (4) allow mobility for both the speaker and the listener; (5) allow listeners to hear not only the primary speaker, but also other speakers in the room as well as their own voices; and (6) accept inputs such as computers, portable digital devices, and TVs. Possible room amplification systems include personal hearing aids as well as personal frequency modulation (FM), sound field, induction loop, infrared, and hardwired systems.

Personal Hearing Aids

Numerous investigations have demonstrated that traditional hearing aids offer little speech perception benefit in noisy or reverberant environments (e.g., Duquesnoy and Plomp, 1983; Plomp, 1978, 1986). This result should not be surprising because, although it is improving, traditional amplification technology does little to increase the SNR of the listening environment. Duquesnoy and Plomp (1983) indicated that minimal benefit occurred from personal amplification when background noise levels reached 60 dBA. Plomp (1986) reported that hearing aids offered limited speech perception benefit when background noise levels exceeded 50 dBA. A review of everyday background noise levels suggests that most environments exhibit background noise levels in excess of 50 to 60 dBA. These data strongly suggest that children wearing traditional amplification will require other technologies that enhance SNR and can augment the capabilities of the hearing aid. This situation has changed more recently, however, since several potential SNR-enhancing options for hearing aids have been introduced that may help the listener in noisy or reverberant environments. Some of these new technologies are described below (see also Chapter 38).

Directional Microphone Technology

Directional microphones were first used in hearing aids in 1972. The main design characteristic of a directional microphone is a single microphone, or two or more microphones that are differentially sensitive to acoustic spectra coming from different azimuths around the head: Specifically, more sensitive to spectra from in front of the head and less sensitive to spectra coming from other azimuths (the side or back). The differential sensitivity of a directional microphone can provide an improvement in SNR if the desired signal is coming from a sensitive azimuth and the background noise is originating from a less sensitive azimuth. The advantages of a directional microphone, however, may be compromised in a reverberant room (Dillon, 2012). Both the desired sound and reflections from the background noise can arrive simultaneously at the microphone's most sensitive azimuth, thereby possibly negating the beneficial effects of the directional microphone. Many microphone technologies have been recently developed to improve on typical directional microphone performance. See Chapter 38 for a discussion of these technologies.

Adaptive Signal Processing Strategies

Most hearing aids today use some form of adaptive signal processing in an attempt to enhance the listener's SNR. Recently, some hearing aids have been designed to reduce the effects of reverberation on the hearing-impaired listener. Adaptive signal processing strategies are often based on digital signal processing algorithms and can be simple or very complex. As these strategies are refined, more consistent and larger speech perceptual improvements are likely.

HEARING AIDS MAY NOT BE ENOUGH

Although there have been striking developments in hearing aid technology, this technology alone will not maximize speech perception for the most listening challenged in noise and reverberant environments. Fortunately, there are other technologies that can be used in these situations. Some of these are described in the following section.

Personal Frequency Modulation Amplification

FM systems have a long history of use and benefit with the hearing impaired and other special populations. FM systems have also been beneficial in improving communication in large room areas (conference rooms, theaters, churches) as well as in face-to-face settings.

An example of a personal FM system is shown in Figure 37.3. With a personal FM system, the voice is picked up by an FM wireless microphone located near the speaker's mouth where the detrimental effects of reverberation and noise are minimal (i.e., the microphone placement is well within the direct field). The acoustic signal is then converted to an electrical waveform and transmitted via FM signal to a receiver tuned to the same frequency. The electrical signal is separated from the FM signal and amplified, then converted back to an acoustic waveform and conveyed to the listener. The Federal Communications Commission (FCC) initially allocated the frequency region of 72.025 to 75.975 MHz for ALDs used by individuals with hearing loss. This frequency region was subdivided into 40 narrowband or 10 wideband channels. The FCC also allocated the frequency range of 216 to 217 MHz for assistive device use, which has improved quality and reduced interference.

FIGURE 37.3 An example of a personal frequency modulation (FM) system. (Photo courtesy of Listen Technologies Corporation, Bluffdale, UT.)

Figure 37.4 shows various FM coupling strategies for use with children who have hearing loss, whereas Figure 37.5 shows various FM microphone and transmitter options. As can be seen in Figure 37.4, the signal can be presented through headphones (or ear buds) or directly to the hearing aids via induction loop or direct auditory input (DAI) technology. The FM unit can also be coupled directly to the ear via a button or a behind-the-ear transducer. Furthermore, for children with conductive or mixed hearing losses, the FM system can be coupled to a bone-conduction transducer. However, it is recommended that, for children with hearing loss, the child's personal hearing aid or aids be incorporated with the FM system whenever possible. This allows the child's personal hearing aid, which is often more electroacoustically flexible than the FM system, to more accurately meet the child's puretone sensitivity requirements. By coupling the FM system to the child's hearing aid, a high SNR is provided for the child's listening environment. That is, it is best to allow the hearing aid to do what it does best (improve hearing sensitivity) and let the FM system accomplish what it does best (improve the SNR of the listening environment). Of course, one concern with this recommendation is that it assumes that the child has a completely functional hearing aid with the option of switching between the following transmission modes: (1) FM only, for the purpose of focusing primarily on the talker; (2) environmental microphone (EM) only, for the purpose of listening to all individuals in the immediate listening environment as well as monitoring his/her own voice; and (3) FM + EM for listening to the teacher as well as other individuals in that listening environment.

The American Academy of Audiology's Clinical Practice Guidelines for Remote Microphone Hearing Assistance Technologies for Children and Youth Birth-21 Years (2008) provides a rationale and comprehensive protocol for devices that use remote microphones such as personal-worn FMs, classroom audio distribution systems (CADS), and Loop systems. These guidelines apply not only to children with all degrees of hearing loss, but also to children with normal hearing who have special listening requirements; that would include children with CAPD.

The protocol contains a core statement that addresses the complex process of HAT selection, fitting, and management plus supplements that outline procedures for fitting and verification of ear-level FM (Supplement A) and CADS (Supplement B). A third supplement for personal neck loops is under development.

The guidelines discuss regulatory considerations and qualifications of personnel as well as candidacy, fitting, and verification protocols. Monitoring and managing equipment is discussed in detail including procedures for checking systems to be sure they are working. Strategies for implementing guidelines in the schools are offered. For access to the full document, please refer to the American Academy of Audiology Clinical Practice Guidelines (2008, 2011).

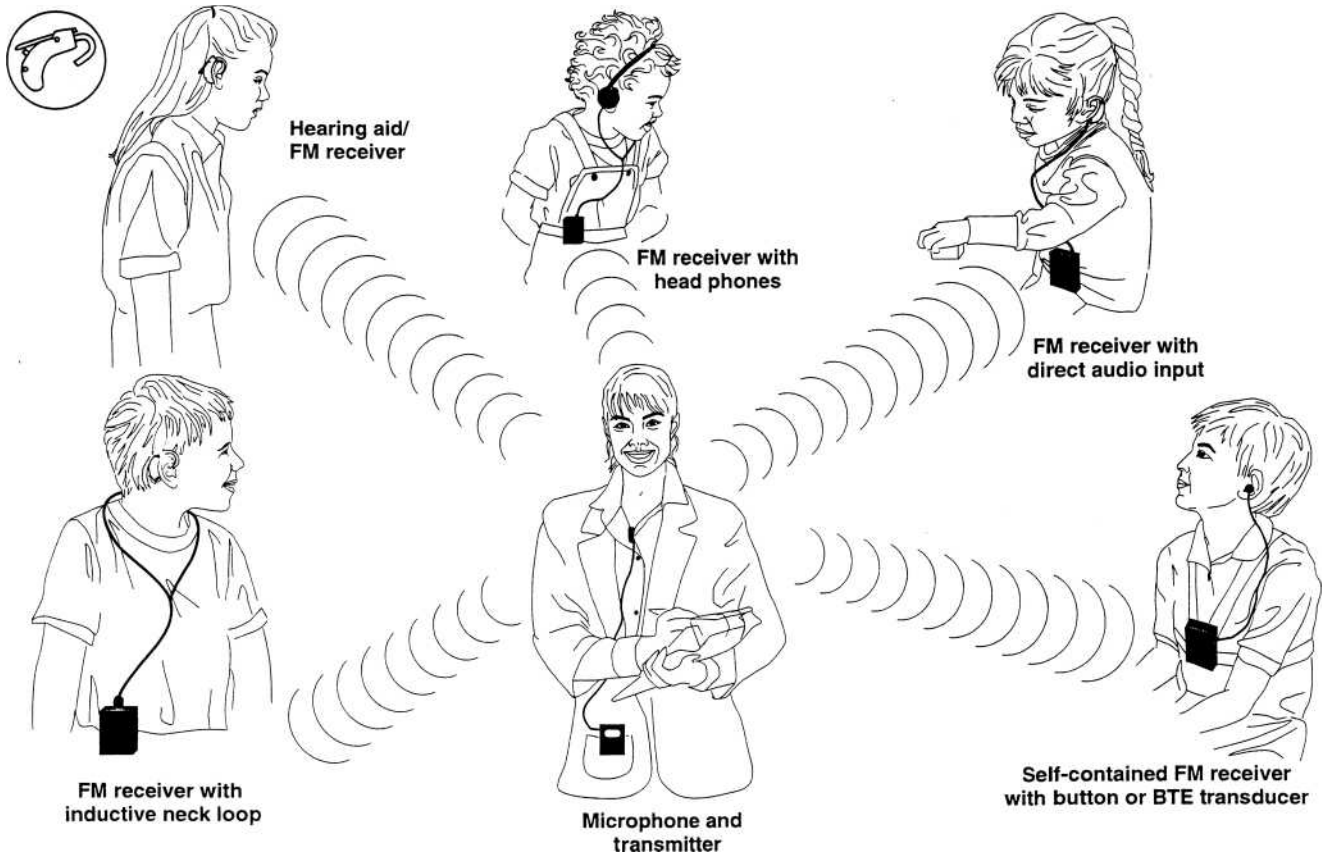


FIGURE 37.4 Various coupling options for use with frequency modulation (FM) systems for children with hearing loss. (Reprinted from Lewis D. (1998) Classroom amplification. In: Bess F, ed. *Children with Hearing Loss: Contemporary Trends*. Nashville, TN: Bill Wilkerson Center Press; pp 277–298, with permission.)

For children with "normal" hearing, the signal can be presented through earphones (see Figure 37.4). It is imperative to realize that there are personal FM systems manufactured for children with hearing loss as well as FM systems developed for children with normal hearing or slight degrees of hearing loss. Systems manufactured for children with hearing loss often offer the user a high degree of electroacoustic flexibility, including the potential for extended frequency response, high gain, and elevated OSPL90s. In addition, these latter systems usually have external controls

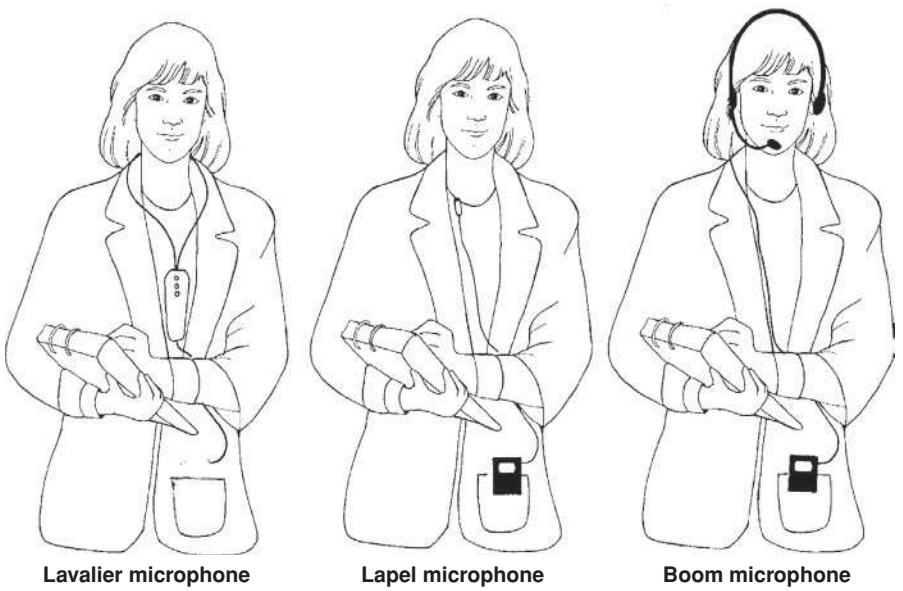


FIGURE 37.5 Various frequency modulation (FM) microphone and transmitter options. (Reprinted from Lewis D. (1998) Classroom amplification. In: Bess F, ed. *Children with Hearing Loss: Contemporary Trends*. Nashville, TN: Bill Wilkerson Center Press; pp 277–298, with permission.)

that allow the child to switch between the various transmission modes (i.e., FM only, EM only, or FM + EM). In contrast, systems developed for children with "normal" hearing offer limited electroacoustic variability with limited gain and reduced OSPL90s. These systems are designed simply to provide the child an improved SNR with little to no amplification. Unfortunately, we, the authors, have often seen children with "normal" hearing wearing FM devices designed for those with hearing loss. This form of FM use is alarming in that hearing loss could ensue if a child was wearing a device set for moderate gain and a high OSPL90. Thus, it is imperative that all children with "normal" hearing be fit with the appropriate FM system. In fact, since even these devices provide some gain, it is recommended that these children use an attenuated headphone when using FM technology in the classroom. An attenuated headphone will reduce the gain/output of the unit by approximately 10 to 20 dB, thus reducing the potential for over amplification. Certainly no FM system (or any other HAT) should be fit prior to the verification of that fitting via real-ear measures. The reader is directed to the American Academy of Audiology Clinical Practice Guidelines (2008, 2011) for further information regarding selection and verification of FM systems.

Personal FM systems are also available in ear-level models. The FM-only models are designed for children with auditory learning difficulties, auditory processing disorders (APDs), attention deficits, or mild conductive hearing loss. For children with hearing loss, integrated hearing aid/FM systems have been developed that provide the user with a combination of both a hearing aid and an FM system in the same ear-level device. For children with hearing within normal limits (WNL), the FM system is simply located in a behind-the-ear configuration, often in conjunction with an open earmold. An example of a system specifically designed for normal-hearing individual with difficulty communicating in noise (APDs, attention deficit disorders, or other learning disabilities) is the Phonak iSense which is offered as an ear-level device similar to a cell phone Bluetooth receiver or as an MP3 player-sized device (Figure 37.6). Unobtrusive devices like these may be more acceptable for classroom use, especially by image-conscious students.

With the continuing evolution of "boot technology," FM receivers can be added to virtually any BTE hearing aid or ear-level cochlear implant. The FM "boot" is a miniature FM receiver that permits a transmitting microphone to be located close to a desired sound source. The FM "boot" usually attaches through a direct audio input connection to the hearing aid (Figure 37.7). High-quality sound from an associated FM transmitter is received at the ear level by the FM boot, providing clear sound from a distance and in noisy environments. Similarly, an "audio shoe" can be attached to the hearing aid, allowing an FM receiver to communicate with the hearing aid via direct audio input, similar to the FM "boot." Audio shoes provide flexibility to attach FM receivers to hearing aids from multiple manufacturers.

FIGURE 37.6 Example of an iSense micro personal frequency modulation (FM) system. (Photo courtesy of Phonak, Warrenville, IL.)

Recently design-integrated FM receivers have become available, which replace the battery door and are internally integrated with the amplification circuitry.

Infrared Light Wave Systems

Infrared systems consist of a wireless microphone, infrared converter, and an infrared receiver. The microphone converts the acoustical signal to an electrical signal that is then transmitted to the converter. The converter transduces the electrical signal to an invisible infrared signal and transmits it to a receiver worn by the listener. The receiver (which also serves as an amplifier) contains photo detector diodes that pick up the infrared signal and transduce the infrared signal back into electrical energy. The electrical signal, in turn, is

FIGURE 37.7 Frequency modulation (FM) system coupled to a hearing aid via a wireless "audio boot." (Photo courtesy of Phonak, Warrenville, IL.)

then changed into acoustic energy and routed to the listener via an induction loop/hearing aid telecoil setup or through headphones/insert earphones. Direct audio input can also be used by those listeners whose hearing aids have the required audio boot. Currently, the majority of infrared systems designed for individuals with SNHL use a narrowband carrier frequency of 95 kHz or a wideband carrier frequency of 250 kHz. Recently, other infrared carrier frequencies have been introduced, making system compatibility an issue. Infrared systems are often used in larger room settings, such as auditoriums, conference halls, theaters, and churches. For large rooms, such as theaters and auditoriums, arrays of transmitters must be used to ensure that all listeners are appropriately placed relative to the transmitted infrared light beams. In the home setting, infrared systems are often used for TV viewing. This application will be discussed in a later section.

For optimal sound quality with infrared systems, the listener must be in a direct line with the transmitter. Infrared light waves cannot pass through or bend around obstacles such as walls. Of course, this can be an advantage or a disadvantage. For example, in the classroom setting, it may not be practical to keep the child in direct line with the transmitter throughout the school day. That is, if other children move in front of the child using the infrared system, the signal may be blocked and not reach that child. Infrared systems also cannot be used outdoors or in highly sunlit rooms since they are susceptible to interference from sunlight. Because infrared light cannot penetrate solid barriers, this form of technology is excellent in large room settings (in which there is limited individual movement when the infrared is used) or adjacent room settings (e.g., multiplex cinemas) to avoid interference from the other rooms.

FIGURE 37.8 Components of a classroom audio distribution system (CADS). (Photo courtesy of Phonak, Warrenville, IL.)

Classroom Audio Distribution Systems

Another form of SNR-enhancing technology is the CADS, formerly referred to as "sound-field amplification." A CADS is similar to a personal FM system; however, with this form of technology, the speaker's voice is conveyed to listeners in the room via one or more strategically placed loudspeakers (Figure 37.8).

The speaker's voice can be transmitted using a transmitting microphone operating in the infrared, FM, or radio frequency (RF) band. The radio or light signal is sent to a receiver connected to an amplifier, and the amplified signal is distributed to loudspeakers in the room. Infrared has become the preferred technology in sound-field amplification systems because of a major limitation of FM technology. There are a finite number of FM frequencies that can be used in proximity with one another without interference. If an entire school is outfitted with CADS technology, then there may not be enough available frequencies for all of the classrooms. Since infrared signals are confined by the walls of the classroom, interference with another classroom system is unlikely, and so the number of infrared systems that can operate in nearby rooms is virtually infinite. Additionally, if a second transmitting microphone (the pass-around microphone) is desired, another FM frequency is required, which further exacerbates the FM limitation. Infrared systems are typically stereo, and so there is another infrared channel available for the pass-around microphone. CADS are generally used to assist children with "normal" hearing in the classroom who require a better SNR. The objectives when placing a CADS in a classroom are twofold: (1) To amplify the speaker's voice by approximately 8 to 10 dB, thus improving the SNR of the listening environment; and (2) to provide amplification uniformly throughout the classroom. Systems vary from compact, portable, battery-powered, single-speaker units to more permanently placed, alternating current (AC)-powered speaker systems that use multiple (usually four) loudspeakers. Typically, loudspeakers are placed on stands and are strategically placed within the classroom. However, several companies now sell loudspeakers that can be placed in ceiling mounts (Figure 37.9). In addition, portable sound-field systems that can be placed on a student's desk and carried easily from classroom to classroom are also available (Figure 37.10). For a more detailed discussion of CADS technology options, the reader is referred to

FIGURE 37.9 Classroom audio distribution system placed in the ceiling. (Photo courtesy of Lightspeed Technologies, Tualatin, OR.)

Smaldino and Flexer (2012) and the American Academy of Audiology Clinical Practice Guidelines (2011).

Numerous investigations have shown that when CADS systems are positioned within the classroom, educational and psychosocial improvements occur for children with normal hearing sensitivity (e.g., Langlan et al., 2009; Massie et al., 2004). The original investigation concerning the effectiveness of CADS was a 3-year longitudinal project called the Mainstream Amplification Resource Room Study (MARRS) (Sarff, 1981). The project demonstrated that students with minimal hearing loss and children with learning disabilities (without any hearing loss) who received instruction using CADS made significantly greater academic gains, at a faster rate, to a higher level, and at one-tenth the cost compared with students in unamplified classrooms receiving instruction with pullout resource room intervention. Younger children tended to demonstrate greater academic improvements than older children. A number of subsequent studies have reported similar findings (see John and Kreisman, 2012, for a review of these studies).

FIGURE 37.10 Portable classroom audio distribution system (FM) on desk. (Photo courtesy of Lightspeed Technologies, Tualatin, OR.)

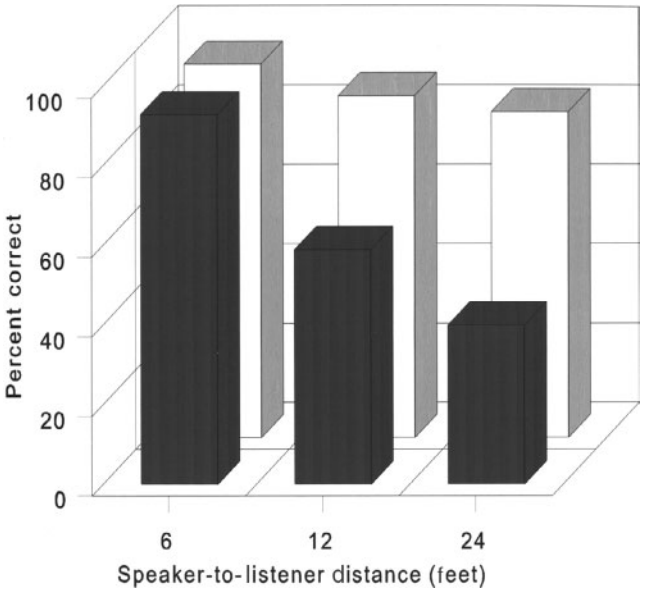


FIGURE 37.11 Mean speech recognition scores (% correct) of children with normal hearing in a "typical" classroom environment (signal-to-noise ratio = +6 dB; reverberation time = 0.6 second) without (*dark bars*) and with (*light bars*) classroom audio distribution (FM) amplification. (Adapted from Crandell C, Smaldino J, Flexer C. (1995) *Sound Field FM Amplification: Theory and Practical Applications*. San Diego, CA: Singular Publishing Group, with permission.)

It is reasonable to assume that these academic improvements were the result of the improved listening environment offered by CADS. For example, Crandell and Bess (1987) examined the effects of an FM CADS on the speech perception of children without any history of learning difficulty or hearing loss in a classroom environment (SNR = +6 dB, RT = 0.45 second). Bamford–Kowal–Bench (BKB) sentences were recorded in both amplified and unamplified listening conditions at speaker–listener distances of 6, 12, and 24 feet. Multitalker babble was used as the noise competition. Subjects consisted of 20 children, aged 5 to 7 years, who listened to the experimental tapes and repeated back the stimuli that they heard. Results from this investigation (Figure 37.11) showed that use of the FM CADS improved speech perception at every speaker–listener distance, particularly at 12 and 24 feet.

Obviously, the same CADS unit and loudspeaker arrangement cannot be suitable for all classrooms. Consequently, Smaldino and Flexer (2012) recommended a pragmatic approach to installing CADS equipment. The pragmatic approach takes into consideration the individual classroom, the individual teacher/teaching style(s), and the pupils in that particular classroom. For instance, if group learning is the primary mode of teaching, the goal is to have each student in the classroom perceive the teacher's voice maximally at all times during the school day. Typically, the

larger the classroom, the more loudspeakers are needed. If angled properly, three or four loudspeakers positioned about 5 feet up on the walls, or in a ceiling array, should provide "surround" sound for all students. If the classroom has specific learning centers/areas, a loudspeaker can be positioned close to each learning center for maximum effective amplification at each of the critical locations. If only one learning center is used at a time, then the other loudspeakers can be turned off. If a small resource classroom is used, two loudspeakers can provide an even and consistent SNR throughout the area. In fact, if the room is quite small, with only a few students seated close to the teacher, even a single loudspeaker might be effective. If the classroom and class size are small, with only one teacher-instructed learning center in use at any given time, then a single battery-powered loudspeaker can be carried by the teacher to each teaching location to amplify that specific environment. In each of these cases, it is imperative that SPLs of the teacher's voice be measured via an SLM to ensure that a uniform, 8- to 10-dB improvement in SNR has been obtained in each of the specific learning areas.

There is the potential for CADS to provide several benefits. First, CADS can provide benefit to virtually *all* children in the classroom. As previously noted, with a CADS, the teacher's voice is transmitted to *each* of the children in the classroom via one or more strategically placed loudspeakers. Consequently, whereas CADS is usually recommended for "normal-hearing, at-risk" children, *all* of the children in that classroom receive, and subsequently can benefit from, an improved SNR. Second, CADS can provide benefit to children with mild degrees of SNHL while malfunctioning hearing aids or auditory trainers are being repaired. Logically, an increase in classroom SNR should augment, at least minimally, the perceptual cues available to these children until their own amplification systems are returned.

Third, CADS are often the most inexpensive procedure for improving speech perception in the classroom. Recall that guidelines for acoustic conditions in classrooms indicate that, for adequate communicative efficiency to occur in the classroom, RTs should not surpass 0.4 second, classroom SNRs should not be less than +15 dB, and background noise levels should not exceed 30 to 35 dBA. Extensive acoustic modification of the classroom (acoustic ceiling tiles, acoustic wall panels, or acoustically modified furniture) can be cost prohibitive for schools. Use of CADS has been shown to be extremely cost effective in overcoming poor room acoustics. An illustration of the cost effectiveness of CADS is shown in Table 37.3. If we estimate the average cost of a CADS at approximately $1,500 and this cost is divided by all of the children in the classroom (since it will benefit the vast majority of children in the room), this equates to approximately $60.00 per child (considering a class size of 25 students). If this cost per student is prorated over a 10-year period (the estimated average life span of a CADS), the annual unit cost per child is only $6.00. There are additional savings of CADS that should be noted. Specifically, CADS has been shown to reduce the number of children requiring resource room assistance, which is often the most expensive assistance offered by schools (Smaldino and Flexer, 2012).

TABLE 37.3

Example of the Cost Effectiveness of a Sound-Field Frequency Modulation Amplification System

Approximate initial cost of sound-field unit	$1,500.00
Cost per child in classroom (25 children)	$60.00
Cost per child over 10-year time span	$6.00

Source: Adapted from Crandell C, Smaldino J, Flexer C. (1995) *Sound Field FM Amplification: Theory and Practical Applications*. San Diego, CA: Singular Publishing Group.

Fourth, use of a CADS does not stigmatize children, which can be the situation with auditory trainers or hearing aids (i.e., because the latter require the children to wear hardware). Children with "normal" hearing who demonstrate perceptual difficulties, particularly those in junior or senior high school, often experience negative reactions from their classmates when using personal listening systems that necessitate the use of headphones or ear buds. Because of this negative stigma, these students frequently choose to use HAT sparingly. Thus, whereas personal listening systems offer an improved SNR compared to CADS, this technology is only useful if the student is motivated to wear it. Rosenberg and Blake-Rahter (1995) reported that 93% of students who used sound-field technology in the classroom responded positively to the use of such systems. Moreover, by passing around the microphone (for oral reports, oral reading, and asking/answering questions) students reported improved classroom interaction and participation.

Fifth, teachers report positive health effects of CADS use during teaching activities. Several studies have found CADS use to significantly reduce vocal load in teachers (reducing the possibility of strain leading to vocal attrition and abuse) as well as lower stress and clearer speech (e.g., Morrow and Connor, 2011).

Sixth, CADS can be used to enhance other instructional equipment. Obviously, in the educational setting, all information presented to children should be audible. Sound-field systems can be connected to equipment such as TVs, media players, computers, and digital devices to make the output more audible in the classroom (Smaldino and Flexer, 2012).

Finally, parents willingly accept CADS. Crandell et al. (1997) reported that more than 97% of parents willingly accept the concept of sound-field technology, even if they have not seen the instrumentation used within the classroom. Presumably, parents overwhelmingly accept this technology not only because of the positive comments they hear

from children and teachers, but also because of the significant improvements in academic performance noted by their children when a CADS is implemented in the classroom.

It must be noted, however, that there are several potential disadvantages of CADS. First, CADS may not provide adequate benefits in excessively noisy or reverberant learning environments. Whereas the exact levels of noise or reverberation that may negate the benefit of CADS is a topic of ongoing research, it is reasonable to assume that, if classroom noise levels are loud enough to mask the speech signal, a 10-dB improvement in the teacher's voice may not be enough to make all elements of speech audible. Moreover CADS are not appropriate in a highly reverberant classroom because these systems do not significantly reduce reverberation and, in fact, may increase the overall RT in some rooms. Therefore, it is imperative to know the acoustic characteristics of a room (noise levels and RT) and minimize these effects with acoustic modifications *before* the installation of sound-field equipment is attempted.

Second, if the loudspeaker arrangement or number of loudspeaker(s) is not appropriate for the classroom, the level from the speakers may not be uniform throughout the classroom. In other words, the teacher's voice may be too loud for some children, whereas not loud enough for other children.

Third, CADS may not be feasible in smaller classrooms. In smaller classrooms or learning environments, it may not be possible to amplify the teacher's voice by 10 dB because of feedback problems associated with the interactive effects of reflective surfaces and speaker closeness. Although it is clear that a system that has frequent feedback will not be of benefit in a classroom setting, it is not clear whether an improvement in SNR of less than 10 dB would still warrant the installation of a system.

Fourth, the teacher and students need appropriate in-service information and follow-up support if a CADS is to provide maximum benefit in the classroom. As with any HAT, it is imperative that the teacher thoroughly understand why a CADS is being recommended and placed in the classroom prior to installation. Specifically, it is critical to make the teacher comfortable with the instrumentation by explaining its theoretical and practical applications in nontechnologic, easy-to-understand terminology. In instances when a CADS is not used effectively in the classroom, inadequate training of the teacher on its use is frequently at fault.

Fifth, CADS may not benefit children with severe recruitment or hypersensitive hearing. By increasing the level of the sound in the classroom by even 10 dB, it is conceivable that a problem could be created for these children.

Finally, classroom-based CADS generally are not portable, which could prove to be a problem if the child uses several different classrooms during the day. However, as noted earlier, individual students may now use personal portable CADS that can be carried from class to class or benefit from the newer personal systems. Also with the growing acceptance of this technology, school districts often equip nearly every classroom in a school with this technology, so moving from classroom to classroom is less of a problem.

Recommending Personal or Sound-Field Frequency Modulation Systems

There is limited information comparing the effectiveness of CADS to personal FM systems (e.g., Anderson and Goldstein, 2004; Flexer, 1992). Clearly, the personal FM system should offer a more favorable SNR than the CADS. Anderson and Goldstein (2004), for example, found that desktop and personal FM systems (combined with personal hearing aids) provided significant benefit for speech perception in noise and reverberation whereas an infrared CADS did not.

Because CADS can be expected to provide only about 8 to 10 dB of amplification, such systems may not provide a sufficient communicative environment for students with moderate to severe degrees of SNHL. Thus, for children with greater than mild degrees of SNHL and/or severe perceptual difficulties in noise, a personal FM system may be the more appropriate amplification. In addition, these children are more likely to require an improved classroom SNR throughout their academic career (Flexer, 1992). It must be remembered, however, that many children, particularly those in junior or senior high school, may not use personal FM systems because of the potential stigma associated with such devices, necessitating the use of a CADS. If a child with more than a mild degree of hearing loss uses a CADS, it is important that some measure of efficacy be obtained to evaluate the effectiveness of the system (see "Outcomes Measures" section in this chapter).

Electromagnetic Induction Loop Systems

An example of an induction loop amplification system (one of the oldest forms of room amplification) is shown in Figure 37.12. As can be noted from this figure, an induction loop system consists of a microphone connected via hard wire (or an FM transmitter) to an amplifier. A length of wire extends from the amplifier. This wire is placed either around the head of an individual (neck loop) or around a designated area, such as in a classroom or theater. When an electrical current flows through the wire loop, it creates an electromagnetic field that can be picked up by any device using telecoil technology. A telecoil is a special device often found in hearing aids that picks up and amplifies electromagnetic signals, and, in turn, converts these signals into acoustic energy that can be heard by the hearing aid user.

Some telecoil-equipped hearing aids and listening systems incorporate digital processing in the conversion from electromagnetic energy to an analog acoustic signal. Interestingly, for years, induction loop systems have commonly

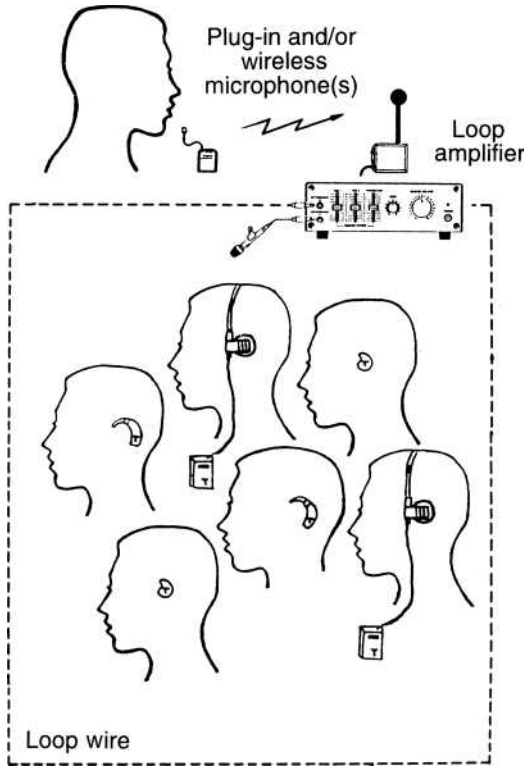


FIGURE 37.12 Components of a large-room electromagnetic induction loop system. Note that, in this figure, the electromagnetic signal is being received by listeners via telecoils located within an in-the-ear (ITE) hearing aid, behind-the-ear (BTE) hearing aid, and a portable induction receiver (for use without a hearing aid). (Photo courtesy of Oval Window, Nederland, CO.)

been used in international conferences to provide simultaneous translation service to conference attendees. In this case, the attendee uses an induction loop ear bud or a handheld wand that contains a receiver/amplifier and speaker. The device can be turned on or simply held to the ear when language translation is required.

There are several advantages of induction loop systems over other forms of room amplification. Primarily, induction loop systems tend to be the least costly of the room amplification systems. One reason for this cost reduction is that induction loop systems do not require additional receivers as do FM or infrared systems. Induction loop systems have relatively few components and are fairly easy to install. Troubleshooting and maintenance of such systems also tend to be relatively easy.

Unfortunately, there are several limitations of such systems in classroom settings. First, recall that induction loop systems require the student's hearing aid to have a functional telecoil that is sensitive enough to pick up the electromagnetic field throughout the classroom. It is incumbent on the dispensing audiologist to be sure to include a strong telecoil in the student's hearing aid(s) if use of an induction system is expected. Failure to do so would make this technology unavailable or less effective to the student and perhaps force the use of more expensive technology to improve the SNR. Furthermore, even when the hearing aid has telecoil technology, past investigations have indicated that many children's hearing aids often malfunction (see Flexer, 1992, for a review). Unfortunately, younger children may not be able to explain the resultant decreased acoustic signal when their telecoil is malfunctioning.

Moreover, many telecoils do not provide enough gain to significantly improve perception with an induction loop system. Variables affecting telecoil gain include the absence/presence of a telecoil preamplifier and the position and/or orientation of the telecoil within the hearing aid. In 2006, a revised inductance loop standard was published by the International Electrotechnical Commission (IEC 60118-4:2006) that specifies reference magnetic field strength levels and coverage as well as acceptable background noise levels. Induction loop systems that are installed in compliance with the standard should demonstrate more consistent volume and frequency response of the system across listeners. However, inductance systems installed without regard to the standard may provide unpredictable and possibly substandard performance.

A second disadvantage of an induction loop system is that some hearing aids do not contain the microphone (M) + telecoil (T) option. Thus, the user without such switching capability would not be able to hear individuals other than the ones closest to the microphone. For example, a student may not hear other students' questions (or even be able to monitor his/her own voice) when receiving the signal from the induction loop system.

Third, the quality of the signal and, therefore, speech perception may decrease as the listener moves away from the induction loop. That is, depending on the orientation of the telecoil within their hearing aid and/or the placement of the induction loop itself, a child seated in the middle of a room may not be obtaining as high a signal quality as the child seated next to the induction loop. Moreover, in larger rooms, the induction loop may not be powerful enough to assist all of the listeners within that enclosure.

Fourth, although the induction loop system is relatively portable, it is often impractical to move such systems to accommodate outdoor activities.

Fifth, since a hearing aid with an appropriate telecoil is required, the system cannot often be used for children with normal hearing. However, it should be noted that there are several devices on the market that use telecoil technology for individuals with normal hearing, such as handheld induction loop receivers with ear buds or a headset.

Sixth, the number of rooms in a building that can be equipped with induction loop technology is often limited because "spillover" (up to 50 to 100 feet) can occur across such systems. Spillover occurs when the electromagnetic signal generated in one room is picked up by a telecoil in an

adjacent room. Notably, modern telecoil installations have successfully decreased this problem, though it remains for older loop installations.

Finally, the quality of the signal produced by induction loop systems and picked up via telecoils may be reduced by other electrical devices in the room that produce magnetic fields (and as a result generate a 60-Hz hum or, in many other parts of the world, a 50-Hz hum). Examples of such devices include fluorescent lighting and electric power lines.

Hardwired Systems

Hardwired systems are those assistive technologies that provide a direct physical connection between the sound source and the individual. Specifically, hardwired systems are so named because a wire connects the microphone to the amplifier, and the amplifier is connected directly to the receiver (headphones, ear buds). An example of such a system is shown in Figure 37.13.

One advantage of such systems is that they provide an inexpensive approach for the amplification of sound. In addition, it is often easy for the consumer to purchase such equipment in electronics stores, through the mail, or over the Internet. Such systems have been shown to be useful for some patients with hearing loss who may not be able to use conventional amplification (e.g., those with cognitive declines, physical disabilities, and/or severe manual dexterity difficulties).

Although inexpensive, there are a number of concerns with hardwired systems. First, such systems are not specified as medical devices by the Food and Drug Administration (FDA). Therefore, there are no standards for the electroacoustic characteristics (gain, frequency response, OSPL90, harmonic distortion) of such devices. The audiologist should perform comprehensive electroacoustic and real-ear measures before placing a hardwired system on an individual with hearing loss. A second concern with hardwired systems is the limitation of movement for the user. That is, since each part of the device is connected via a wire, the user can only be as far from the sound source as the wire length will allow. Hardwired systems are generally not used for larger rooms such as classrooms because they require the child to sit in predetermined locations and the microphone wire can restrict teacher movement.

FIGURE 37.13 Example of a portable assistive listening device. [Photo courtesy of Williams Sound, Eden Prairie, MN.]

Advances in Digital Transmission Technology

The design options available to designers of CADS and other listening systems are improving rapidly as digital technology becomes faster, smaller, and more versatile. New systems have incorporated proprietary communication protocols and processing algorithms to replace the standard FM and infrared channels used in personal listening systems and CADS. Digitization of the signal allows for advanced signal processing, such as error correction in the transmission and dynamic maintenance of desirable SNRs.

Bluetooth is a proprietary open wireless technology standard for exchanging data over short distances using short wavelength radio transmissions in the ISM (Industry, Scientific, Medical) band from 2,400 to 2,480 MHz. Electronic devices, including mobile phones, tablets, and laptop computers, may employ Bluetooth to create personal area networks (PANs) with high levels of security. Bluetooth streaming has enjoyed some application in connecting cell phones and hearing aids. This technology has obvious potential in the CADS arena. Indeed, some CADSs that transmit using other methods do employ Bluetooth communications to interface with other devices such as media players.

Digital inductance transmission is different from the analog inductance systems discussed previously. In a digital induction loop system, the analog audio signal from the microphone is transformed into a digital stream by an analog-to-digital converter. This digital data is then coded and modulated onto an inductive carrier frequency. This coded and modulated signal is fed into a loop of wire which generates a magnetic field. The field is received by a special type of telecoil and is demodulated and decoded to retrieve the transmitted digitized signal. The digital signal is then converted back into an analog signal by a digital-to-analog converter in the receiver or hearing aid and processed as an analog signal. Digital inductance loop technology promises less interference, a wider frequency band, and more consistent signal. The downside is that the signal cannot be received by the typical analog telecoil found in modern hearing aids, and so does not enjoy universal compatibility. Whereas some manufacturers may include coils for both analog and digital inductance in their hearing aids, the simplicity, low cost, and universality of analog inductance systems argue against widespread use of digital inductance technology, at least in the near future.

COMMUNICATION STRATEGIES

Although technology can significantly improve the acoustic environment in the classroom, it should not be entirely depended on to enhance listening and learning. Active participation of the teacher and students is necessary to optimize the effectiveness of the technology. Effort should be made to compliment the technology with physical positioning of teacher–student, the use of clear speech principles, optimization of visual speech cues, and learning to listen.

Reducing Speaker–Listener Distance

In the absence of any HAT, for optimal speech perception to occur, the listener needs to be in a face-to-face situation and in the direct sound field of the talker. Recall that speech perception can only be improved within the critical distance of the room. Beyond the critical distance, speech perception ability tends to remain constant. Therefore, in any listening environment, the speaker–listener distance should not exceed the critical distance of the room. Unfortunately, the critical distance in many rooms occurs only at close speaker–listener distances. To remain within the critical distance of a room, restructuring of the room dynamics may need to be considered. For example, small group instruction (where the speaker addresses one small group at a time) should be recommended over more "traditional" room settings (where the speaker is situated in front of numerous rows of students). Crandell et al. (1997) reported that speech perception scores of children were very good or excellent when such small group instruction was used in a classroom.

Clear Speech

Clear speech procedures may also facilitate speech perception in many enclosures. Clear speech refers to a process in which the speaker focuses attention on clearer pronunciation, while using a slightly slower rate of speech and a slightly higher intensity—a speech style used by most newscasters. Several investigations have demonstrated that clear speech can significantly improve speech perception in noisy and reverberant environments (e.g., Schum, 1996). For example, Payton et al. (1994) demonstrated that, in poor listening environments, the average improvement in speech perception when clear speech was used was 20% for listeners with normal hearing and 26% for listeners with SNHL. It is reasonable to expect speakers, such as teachers, to learn clear speech procedures easily because talkers can be trained to produce clear speech continuously after a minimum amount of instruction and practice (Schum, 1996).

Optimizing Visual Communication

Face-to-face communication at relatively short speaker–listener distances also aids the listener with hearing loss by maximizing speechreading opportunities. Optimal speaker–listener distance is approximately 5 to 10 feet. Speechreading ability tends to decrease significantly at 20 feet. Several investigators have also reported that speechreading benefit increases as a function of decreasing SNR (Erber, 1979; Middleweerd and Plomp, 1987). That is, listeners rely more heavily on visually transmitted information as the acoustic environment becomes more adverse.

Improving Listening Strategies

Listening (as opposed to hearing) refers to the ability to detect, discriminate, identify, and comprehend various auditory signals. Listening is a major component of the communication process. Listening comprises 45% of daily communication for adults, whereas school children spend as much as 60% of the school day in the process of listening (Rosenberg and Blake-Rahter, 1995). Research has demonstrated that listeners who experience difficulty at any level of the listening process will find it more difficult to use auditory information in an efficient manner. Despite the importance of listening in communication, the process of listening is rarely taught to individuals with hearing loss (Rosenberg and Blake-Rahter, 1995). Erber (1979) emphasized the importance of a thorough audiologic and developmental speech assessment to determine the placement of a student on an auditory skill development continuum. This continuum, which includes sound awareness, sound discrimination, identification, and comprehension, forms the basis for the listening training activities that must be taught specifically to children with hearing loss but that develop normally without training in normal-hearing children. It is important to recognize, however, that listening training may also be necessary when new amplification devices are fit (because of the new signal characteristics) and if room acoustics reduce the intensity or distort the quality of the acoustic signal. It is also noteworthy that some normal-hearing children, such as those with APDs or nonnative speakers, may also benefit from listening training.

TECHNOLOGIES WITH APPLICATION TO INDIVIDUALS WITH HEARING LOSS/AUDITORY PROCESSING DEFICITS

Many new technologies have recently become available that can assist individuals with hearing loss; many of these same technologies are also useful for people with normal hearing who have processing-related difficulties and/or find themselves in adverse listening environments. It is beyond the scope of this chapter to overview all of these technologies. A web search will provide the interested reader with access to this ever-expanding list of assistive technologies. We have selected for review a cross section of prominent and

promising technologies. We realize that as technology has advanced, the line between broadcast media devices, telephony devices, and computers has blurred; however, we still think it helpful to distinguish between the technologies as much as possible for the purpose of discussing the available HATs.

Reception of Broadcast Media

Individuals with SNHL often have a difficult time hearing and/or understanding the auditory broadcast over the TV or radio. As was outlined in the first section of this chapter, a number of factors, such as distance from the sound source, background noise, and poor room acoustics, can interfere with the signal to those with hearing losses. Hearing aids alone often do little to reduce background noise or overcome poor room acoustics and thus may not be a viable solution. Modern TVs often have multiple audiovisual inputs and outputs, which make utilizing assistive technologies much easier than in the past. Assistive technologies, such as those discussed for large rooms (e.g., infrared systems), can also be used to link broadcast media to the individual with hearing loss and therefore effectively improve the quality of the audio signal. The use of assistive technologies to improve reception of the TV or radio can be accomplished with or without the use of a hearing aid. For example, one of the more popular TV listening devices (Figure 37.14) sends the audio signal to a receiver worn by the user via infrared technology. The audio signal of the TV or radio may also be enhanced by coupling the individual's hearing aids to the receiving device through DAI or by an induction neck loop. Personal sound-field FM systems can also be useful. In this case, the transmitting microphone is placed close to the TV or radio speaker and the receiving speaker amplifier is placed close to the listener. The increased volume and reduction of room acoustics improve sound reception. Additionally, TVs with audio output jacks can be connected to a stereo receiver. The listener can then plug earphones into the phone jack of the receiver and use the volume control and frequency adjustments to provide an amplified TV signal. If the listener's hearing loss is too great for the use of stereo headphones without feedback, then an inductance neck loop can be connected to the stereo receiver and the signal picked up by the telecoil in the listener's personal hearing aid. Most hearing aid companies now offer proprietary TV devices that plug into an audio out jack of the TV and wirelessly transmit the signal to the hearing aids, usually via a small streaming device that is worn by the listener.

FIGURE 37.14 Personal infrared assistive device used to improve television listening. (Photo courtesy of Sennheiser, Old Lyme, CT.)

Captioned Media

For the individual with hearing loss (or an individual for whom English is a second language), TV viewing can be enhanced with various forms of captioned media (closed, real time, and open). With closed captioning, the text of the broadcast is encoded in the TV signal and must be recoded to appear on the TV screen. Closed-captioned words appear in white uppercase format and are encased in a black box that usually appears in the lower portion of the TV screen, although recent innovations allow the captions to vary in location so as not to block any other text or the talker's face. A TV with a decoder chip or a separate decoder box is necessary to view the closed captioning. As a result of the Television Decoder Circuitry Act of 1990 (PL 101-431), all TV sets more than 13 inches must come equipped with a decoder chip so that individuals can have access to closed captioning when available.

Real-time captioning is available for live broadcasts such as the news and sporting events. In real-time captioning, a trained stenographer-captioner follows the audio signal and, with a delay of 2 to 3 seconds, converts it to text that appears on the TV screen. Recently, voice-to-text technology has become refined enough to use as a real-time captioning device. Using sophisticated voice recognition technology, these devices hold promise of directly converting the speech signal into text that can be read by the individual with hearing loss.

Another form of captioned media is open captioning. With open captioning, the captions are permanently placed on to the video and always available to the viewer with no decoder needed. Open-captioned letters are usually white with a black shadow or border. Some movie theaters now offer open caption viewings of certain movies at given times.

A new captioning technology promises to significantly improve access for people who are deaf or hard of hearing. Discrete personal captioning uses state-of-the-art electronic, optical, and voice recognition technologies to provide text in a "heads-up" display (similar to that found in fighter aircraft) built into a pair of glasses. A text readout seems to float in the air about 18 inches in front of the wearer.

Telephony

Hearing Aid Telecoils, Rehabilitative Technologies, and Telephone Use

A telephone induction pickup coil, or "telecoil," picks up the electromagnetic leakage from the telephone receiver. The signal is then amplified, transduced into acoustic information, and delivered to the individual's ear. Improved telephone communication is obtained because the hearing aid microphone is turned off, thus reducing the level of the background noise. Some hearing aids will also simultaneously turn down the gain of the hearing aid on the opposite ear, further reducing background noise. In addition, the frequency response of most telecoils tends to be smoother than when the hearing aid is coupled acoustically to the telephone. Unfortunately, because of the miniaturization of hearing aids, many amplification systems are not equipped with telecoils at all or strong telecoils because of size restrictions. It is our view that hearing aids, whenever possible, should be equipped with telecoil technology because their use far exceeds telephone communication. It must be noted that there is great variability in telecoil power and frequency responses across hearing aids and even within hearing aid companies. Thus, we recommend that real-ear measures be conducted with telecoils to ensure their proper function. Certainly, whenever telecoil technology is implemented into a hearing aid, it is imperative that the patient be instructed concerning its proper use.

In addition to telecoil technology, a number of rehabilitative technologies can be used in conjunction with telephone use to improve communication in individuals with hearing loss. For example, one device consists of a disc-shaped microphone that attaches to the telephone handset. The signal is then routed into the listener's personal hearing aid via DAI (thus avoiding the use of the telecoil). Often, the use of individual personal rehabilitative technologies, particularly with DAI, can be beneficial in environments where electromagnetic interference (e.g., computers, fluorescent lights) is high.

Telephone Amplifiers and Accessories

Telephone amplifiers and accessories designed specifically for individuals with hearing loss are readily available. Amplified phones offer an array of built-in features, such as hearing aid–compatible headsets, amplifiers with up to 50 dB of amplification, a volume control, and signal processing to maximize comfort and clarity; high-output adjustable ringers; a tone control to adjust the frequency response of the headset to improve intelligibility of words; background noise suppression circuitry to remove unwanted background noise; and visual ring indicators such as a strobe light. Many also have speakerphone capability. Stand-alone in-line amplifiers provide some of the features of the dedicated amplified phone but can be used with many modular unamplified phones.

Cell phone amplifiers are available that can be used with external headsets or earphones. The amplifier plugs into the 3.5-mm audio adapter of the cell phone, and the external headset then plugs into the amplifier. There are also Bluetooth devices that can interface with the cellular phone and be connected via DAI to the hearing aid (see section on Bluetooth). Also, portable telephone amplifiers can be strapped onto a nonamplified telephone to provide up to 30 dB of amplification.

Telecommunication Devices

Individuals with severe to profound hearing losses may not be able to use the telephone effectively, even with amplification devices. In addition, there are individuals with severe speech impairments or extremely poor speech perception who may not be able to use a conventional telephone. For these individuals, telecommunication devices for the deaf (TDDs), also called teletypewriters (TTYs) or text telephones (TTs), may be required (Figure 37.15). Note that the preferred term at this time by the deaf community is TTY. Using typewriter technology, the TTY transmits a typed, visual message (in Baudot code) over standard telephone lines. The typed communication either appears on an LED display or can be printed on paper. Braille TTYs are also available to those with visual as well as hearing difficulties.

FIGURE 37.15 A telecommunications device for the deaf (TDD) that consists of a keyboard and a telephone cradle on top. (Photo courtesy of Silent Call, Clarkston, MI.)

The maximum rate of transmission is approximately 60 words per minute, depending on the sender's typing skills. For a TTY conversation to take place, both the sender and the receiver must have TTY instrumentation that is compatible. Recent evidence indicates that users of TTYs that are connected directly to the telephone network experience more successful conversations with less message interference than with acoustic coupling (Spicer et al., 2005). The Americans with Disabilities Act (ADA; PL 101-336) mandates that all emergency access services have TTY accessibility. Unfortunately, it appears that many agencies and businesses do not use TTYs effectively. A TTY system can be modified to communicate with a computer (i.e., one individual uses a TTY, whereas the other individual receives/sends transmissions on his/her computer).

Mobile Phone

The mobile phone has become a pervasive multimedia platform for communication. Because these phones can connect to the Internet, much of the assistive communication technology such as text messaging, e-mail, video conferencing, and Bluetooth interconnectivity is available in a highly compact and portable form. Downloads of text and video information to mobile phones from the Internet are already possible, and the capabilities of these phones are sure to expand in the future.

Federal Communications Commission Rules Governing Telephony for the Deaf and Hard of Hearing

A common complaint of listeners with SNHL is difficulty understanding speech over the telephone. The Hearing Aid Compatibility Act of 1988 (HAC Act) requires that the FCC ensure that all "essential" telephones and all telephones manufactured or imported for use in the United States after August 1989 are hearing aid compatible. FCC rules require that phones subject to the HAC Act (1) produce a magnetic field of sufficient strength and quality to permit coupling with hearing aids that contain telecoils; and (2) provide amplification with a volume control so the phone can be effectively used by individuals with hearing loss (with or without hearing aids). In 2003, the FCC adopted rules to make digital wireless telephones compatible with hearing aids and cochlear implants. Beginning September 8, 2012, the FCC required nearly all wireless service providers and handset manufacturers that offer three digital wireless handsets in the United States to offer at least one model that is compatible with hearing aids.

Communication through Video Conferencing

Video conferencing technology has become common for business situations, distance teaching and learning, and interpersonal communication. With such technology, a camera-based system is placed on the computer or TV. Both the caller and the person receiving the call must have the camera-based system and supporting software to use this technology and thus receive both the audio and video signals. Systems are also available that provide for full motion video support for sign language. Keyboard-produced text and TDD capabilities are also available options.

RECENT CROSSOVER TECHNOLOGIES WITH APPLICATION TO THE DEAF AND HARD OF HEARING

Multiuse Handheld Devices

Over the past several years, handheld multiuse devices (i.e., smart phones) have become commonplace. For example, a single device may serve as a cell phone with video screen, text messenger, and digital camera. In addition, the device can access the Internet, send and receive e-mail, receive Internet broadcasts or webcasts, and contain technology that enables it to communicate with several other electronic devices at any given time. Although these features may be integrated within a single device, they are described individually in greater detail in the following sections.

Bluetooth

Bluetooth is a short-range wireless technology that can connect a wide variety of electronic devices. Bluetooth operates in the 2.4- to 2.5-GHz frequency range and has a range of approximately 10 m. Because of the high frequency range and the method of transmission, Bluetooth requires only very small antennas and allows several devices to be connected simultaneously. Thousands of products use this technology. One HAT product that has been made commercially available recently is an ear-level device that couples to BTE hearing aids through the DAI port. The device can then communicate with other Bluetooth-enabled devices, such as cell phones, TVs, and computers. Most hearing aid companies now offer a gateway device that will receive a signal to any Bluetooth device to which it is paired, and then wirelessly transmit that signal to the hearing aids. For example, users can hear their cell phones ring through a hearing aid, push a button to activate the Bluetooth gateway device, and receive the phone signal through the hearing aid via the Bluetooth gateway device. Furthermore, the user's voice is picked up via a microphone built into the gateway device, which is relayed to the cell phone. The device automatically ends the connection when the call is finished. Essentially, the cell phone itself is used only for dialing phone numbers. Individuals whose hearing aids do not have DAI can still couple their hearing aids to cell phones by plugging a

compatible neck loop into the audio output jack of the cell phone.

Text Messaging/Text Pager

These technologies enable individuals to send and receive text messages between handheld devices, such as cell phones, or from computers to cell phones. Text messaging is a popular form of communication among individuals with and without hearing impairment. As text-based communication continues to gain popularity, it may assist in breaking down barriers in communication between individuals with hearing loss and those with normal hearing.

Relay Services

Telephone relay services (TRSs) are available to the TTY user who needs to communicate with a non-TTY user. With the TRS, the individual using the TTY types a message to a state-designated central telephone number that is picked up by a normal-hearing operator and, in turn, transmitted verbally to the non-TTY user. The non-TTY user can then respond to the TTY user by speaking to the relay operator, who, in turn, relays the message via written text to the TTY user. Thus, with TRSs, individuals with normal hearing ability can telephone individuals who use TTYs.

Video relay allows a person who is deaf or hard of hearing to make a call to a traditional voice phone number but allows them to use sign language transmitted via a camera or video phone instead of text. When using a computer, the person goes to the appropriate website, enters the number to be called, and can then begin the conversation. An interpreter watches the person signing and verbally translates to the person being called. Responses from the person being called are then translated into sign language by the interpreter.

Relay calls can also be placed using certain instant messenger services or multiuse handheld devices. Federal Relay Conference Captioning (RCC) is also available. This government-mandated service allows individuals with hearing loss to participate in conference calls with users on voice lines by providing services including video relay and live captioning. The caption can then be read on any computer with an Internet connection. This service is free to all federal employees.

Communication Technologies and the Home Computer

Home computers (including desktops, laptops, and tablets) have enabled people with hearing loss (and people with normal hearing) to have better access to communications. Technologies available via a home computer with network/Internet capabilities include electronic mail (e-mail), instant messaging (IM), video conferencing, audio broadcasts (webcasts), and videos. E-mail allows a user to send and receive text messages that, in turn, can also have attachments of photos or electronic files. These messages can be sent and received almost instantly, and most can be of unlimited length. E-mail allows users to sort and store messages sent to them. Limitations of e-mail include spam (receiving unsolicited advertising via e-mail) and the spreading of computer viruses through e-mail.

Unlike e-mail, IM allows two computer users to communicate in real time via text messages sent through the IM software. Therefore, IM is more conversational in nature than e-mail. Many people with hearing loss are using IM more often because IM is faster than TTY. IM conversations can also be saved to an electronic file and can be printed via the computer printer.

Web telephone/videophone website use has increased in popularity recently. Generally, a broadband Internet connection and a computer microphone and speakers are required for the use of such websites. Potential advantages of this technology include high-fidelity reproduction of sound compared to standard phone lines and the ability of each user to raise or lower the volume of the received message using the computer's speakers or internal volume control. One advantage of using computer speakers is that the volume can be adjusted more readily, and unlike standard telephones, the signal should not interfere with the use of hearing aids. The disadvantage of external speakers is that the other person may hear an echo of his/her own voice if the speaker volume is too high. This problem can be alleviated for the most part by using headphones or induction neck loops connected to the headphone slot.

Webcasts, or Internet broadcasts, enable the relatively low-cost distribution of prerecorded audio or video files for download to the computer, portable digital audio devices, and many mobile telephones. Certain websites also offer the option of automated closed captioning for the videos, although the technology for doing so is still not perfected. In an educational setting, netcasts webcasts may be used to reinforce classroom teaching and may enable individuals with hearing loss to access the material at their own speed. Debevc and Peljhan (2004) reported that adults and students who had access to web-based lectures performed better than adults using traditional lectures.

Online Communities

Online community websites are now ubiquitous. These websites allow users to post messages, pictures, and videos, as well as IM and e-mail. Although the IM is synchronous, most of the other features allow for asynchronous communication, meaning that messages and responses are posted by people at different times. Online communities have largely replaced electronic chat rooms and discussion boards, although these forms of online communications

still exist and have also been incorporated into online educational platforms.

Electronic Chat Rooms and Discussion Boards

Internet chat rooms and discussion boards have become a very popular means of communication. Chat rooms can be formed based on a discussion topic and can be specially created for communication among individuals who are deaf or have hearing loss. The chat can be synchronous, meaning that everyone is online and communicating simultaneously. The discussion boards are asynchronous, meaning that messages and responses are posted by people online at different times. Both chat rooms and discussion boards can be used to reinforce classroom teaching or for distance education.

ALERTING SYSTEMS

For a person with hearing loss, common appliances that rely on sound to convey a signal to the user may not be useful. The term "alerting systems" is used to describe devices that can focus the user's attention and/or indicate the presence of sounds in the environment through one of three modalities: Auditory (e.g., amplified or lowered pitch signal), visual (e.g., turning on/off lamp, strobe light, bright incandescent light), or vibrotactile (e.g., devices that vibrate, such as pocket pagers and bed shakers, or increases in airstream, such as a fan). These devices are widely available and are often thought to be appropriate for individuals with profound degrees of hearing loss; however, many such devices may also be beneficial for persons with milder degrees of hearing loss. For example, many persons with high-frequency hearing loss can exhibit difficulties hearing the microwave timer, doorbell, or telephone ringer, particularly if they are not in the same room as that device.

Direct Electrical Connect Systems

Direct electrical connect systems are interfaced permanently with, and activated directly by, the electrical system of the sound-activating device. For example, the alerting device may be connected directly to the telephone, alarm clock, microwave timer, or doorbell. When the device is activated, a visual, auditory, or tactile signal is transmitted to the individual. Generally speaking, such devices, although highly reliable for alerting purposes, are not portable.

Sound-Activated Systems

Sound-activated systems use a microphone to detect the presence of a particular environmental sound and relay the signal to an alerting system. For example, a microphone placed near the microwave or oven timer can inform the individual via an alerting system, such as a body-worn vibrotactile device or on/off activation of a lamp, when that timer has been activated. Another common example of this technology is the placement of a sound-activated microphone near a baby crib so that parents can be informed when the child cries or makes noises. Sound-activated systems generally have sensitivity settings to reduce the possibility of other environmental sounds activating the system. Sound-activated systems are portable and therefore may be advantageous to an individual with hearing loss who is traveling. Recent technologies have been developed that allow the user with hearing loss to monitor important traffic noises (such as emergency vehicles and car horns) when driving.

Induction-Based Systems

Induction-based systems use the electromagnetic field emitted from an activated electrical device to trigger a separate alerting device. An electromagnetic detector is typically placed via suction cup onto an electrical device such as a telephone. When the phone rings, the electromagnetic field that is generated triggers an alerting device such as a flashing table light. Although such systems are easy to use and portable, incorrect placement of the detector can cause the system to malfunction.

Service Animals

In addition to traditional alerting systems, an individual with hearing loss may also choose to use a professionally trained service animal, such as a "hearing dog," to indicate important environmental sounds. Service animals are trained to attract the attention of the person with hearing loss when particular sounds occur (such as a phone ringing) and to lead them to the source of the sound. Restrictions on the use of service animals vary by state, but many establishments welcome service animals such as hearing dogs and guide dogs for individuals with loss of vision.

ASSESSING COMMUNICATION DEFICITS AND NEEDS

The aural rehabilitation process is designed to minimize the communication deficits caused by a hearing loss. The first step in this process is a thorough evaluation of the audiologic dimensions of the hearing loss. In addition to the usual comprehensive audiologic tests, which include pure-tone and speech audiometry and immittance measures, the individual's speech perception ability (using stimuli such as nonsense syllables, monosyllabic words, and sentences in quiet and noise) and central auditory processing capabilities should be evaluated. These procedures are discussed

elsewhere in this handbook. Furthermore, a communication disability and needs assessment is a crucial component of the rehabilitative process. On the basis of the audiologic and communication assessments, audiologists can provide counseling regarding technologies and/or therapies that will minimize the impact of the individual's hearing loss in everyday activities. However, some audiologists may find the prospect of evaluating communication disability daunting. Tye-Murray (2009) discussed four possible issues underlying the difficulty associated with the communication needs assessment. First, communication handicap varies as a function of the communication setting and communication partner. For example, an individual may have a significant handicap in some situations when talking with unfamiliar people, yet have little handicap in quiet situations with a well-known family member. Second, handicap can vary as a function of the topic of conversation. A person may have no handicap when discussing the weather but may experience great obstacles during a discussion involving an unfamiliar topic. Third, handicap does not always manifest itself during conversations between the clinician and the individual with hearing loss. The office assessment is merely a snapshot in time that may not be representative of real communication ability or disability. Fourth, communication handicap is a construct made up of many dimensions; no single assessment measure is likely to capture all of these dimensions. As a result, several assessment measures should be taken to obtain a more comprehensive overview of an individual's communication handicap in various everyday life situations.

A number of procedures have been developed to quantify the extent of communication handicap imposed on the individual as a result of hearing loss. The same procedures can be used to monitor and document the effectiveness of interventions in reducing the communication handicap. Tye-Murray (2009) presents five general procedures. The first of these is the interview process, wherein specific information about a person's hearing problems is elicited through the use of informal or formalized questions. One of the problems with the interview process is that it is hard to quantify the responses of the individual with hearing loss. However, an interview approach called the Client Oriented Scale of Improvement (see Dillon, 2012) requires the interviewees to rank order the five listening priorities they wish to address through the intervention process. The quantification of these problems can then be used as a measure for assessing whether the intervention has been effective. If the initial problem areas are no longer considered to be problematic or if the rank ordering changes to a lower ranking, the intervention might be considered to be successful.

A second procedure is the use of a questionnaire. Many hearing handicap questionnaires have been developed and can be quite useful if the questions match the everyday listening situations of individuals with hearing loss. A hearing handicap scale that is well matched to the individual can provide important information concerning the effectiveness of intervention. If after the intervention the individual reports a reduced hearing handicap, then the intervention can be considered effective.

A third procedure to evaluate communicative handicap is a daily log or diary, wherein the individual provides quantitative information about his/her communication difficulties. These logs provide an ongoing self-report of changes that occur as a result of the intervention and can be used to assess the intervention's effectiveness. In addition, in reporting use of communication strategies, the client may actually become more skilled at using the recommended strategies. For this reason, daily logs can be used as part of a training procedure.

A fourth procedure, group discussion, can also serve as an effective measure of communication handicap. The interactions that occur during a group discussion between persons with hearing loss often force individuals to reflect on their communication problems and possible solutions. Over time, group interactions can provide information and psychosocial support and thereby empower individuals to accept their hearing loss and encourage them to explore technologies that may maximize their potential.

The fifth procedure is called structured communication interactions, in which conversations between the individual with hearing loss and the evaluator are simulated to reflect communication situations typical for that person. The effectiveness of intervention can be assessed directly by simulating difficult communication situations with and without HAT.

Extended audiologic and communication handicap evaluations allow the audiologist to have a comprehensive picture of the auditory capabilities and communication needs of the client. This information is integrated into the counseling phase, and a rehabilitative plan is determined. Typically, such a plan would involve consideration of hearing aids, assistive devices, and a communication strategies training program (Tye-Murray, 2009). All three facets should be considered and integrated for communication handicap intervention to be most effective. Unfortunately, too often only one of the three options, the hearing aid evaluation, is suggested and implemented.

The technology requirements for effective communication for a particular individual will vary depending on the setting and the type of communication (e.g., face to face, over the telephone). The outcomes can be assessed through the assessment procedure as discussed earlier.

Communication Strategies Training

As a result of the audiologic and communication assessments, recommendations can be made regarding appropriate hearing aids and assistive devices. However, to have maximum impact on the communication handicap, the person must

also be provided with the means to take ownership of his/her own communication environment and to have the psychosocial and behavioral tools to minimize miscommunication in everyday listening situations. The process by which the client is provided these tools is known as communication strategies training and is an important component of an overall audiologic rehabilitation plan. Tye-Murray (2009) conceptualizes communication strategies training as being composed of three stages. In the formal instruction stage, the client is provided with information about various types of communication strategies and appropriate listening and speaking behaviors. Included are presentations describing facilitative strategies (tactics a person can use to improve the reception of a message by varying the message, the speaker, the environment, and/or the listener), receptive and expressive repair strategies (tactics a person can use when a message is not understood), and instruction in using clear speech (a speaking technique for making speech highly intelligible).

The second stage is called guided learning. In this stage, the professional creates simulated real-life communication situations in which the strategies acquired in the formal instruction stage can be practiced. The audiologist provides feedback and tips to clients as they progress through the simulations.

The last stage requires the client to engage in prescribed real-world listening situations and to answer prepared questions regarding the effectiveness of the information learned in stage one and practiced in stage two. The reader is referred to Tye-Murray (2009) for a complete description of the communication strategies training component of the rehabilitative process.

OUTCOMES MEASURES

Whenever HAT is recommended, it is important for the audiologist to identify and quantify the effects of the recommendation. Not only is this documentation often required by third-party payers, but also, as a profession, feedback for our recommendations is a means to establish best practices. In the realm of hearing aids, there are a number of approaches to measuring rehabilitative outcomes (see Weinstein, 2000, for a review). Tools for measuring the efficacy of technology in the classroom are also available (Smaldino and Flexer, 2012). These and other measures can be used to document the changes that occur as the result of recommending or fitting an assistive device. For example, the Client Oriented Scale of Improvement could be used with assistive technologies as easily as hearing aids for which the assessment measure was designed. In either case, the client is required to list situations in which hearing help is needed and rate the difficulty experienced in each situation. After intervention (the fitting of a hearing aid or other HAT), the list is reviewed and re-rated to document changes as a result of the intervention.

REHABILITATION TECHNOLOGY IN THE AUDIOLOGY SETTING

Although this chapter has addressed the many advantages that assistive technologies can provide to listeners with hearing loss, unfortunately few audiologists are actively dispensing such technologies. Consumers also demonstrate a much lower knowledge level for HAT than for hearing aids or cochlear implants. To some extent this may be due to the fact that HAT is a less profitable venture for audiologists in private practice than are conventional hearing aids. It seems apparent that audiologists underrate the importance and benefits of assistive technologies. This is unfortunate because there is ample evidence that integrating assistive technologies into rehabilitative plans can be very effective when hearing aids are not enough (e.g., Wayner, 2004).

Consumer Acceptance of Assistive Technologies

Audiologists themselves must first be convinced of the value of assistive technologies in the overall rehabilitative plan for a client. Until now, great emphasis has been placed on the proper fitting of hearing aids, whereas little attention has been given to the benefits of HAT for addressing the rehabilitative needs of the individual with hearing loss. Therefore, the first step in creating consumer acceptance of these technologies is the development of a philosophy of rehabilitation that includes a multidimensional assessment of the individual's auditory and communication capabilities and needs. An outline of how this can be accomplished has been presented in this chapter. Within the context of the comprehensive rehabilitative plan, the value of assistive technologies is self-evident because these technologies are an integral part of the services provided in the rehabilitative plan for an individual, along with hearing aids and communication strategies training.

Outside of a carefully constructed rehabilitation program, there is a need to engage in activities for enhancing consumer acceptance of assistive technology. The same negative stigmas that are attached to hearing loss and hearing aids are likely to be attached to assistive devices and likely will occur to an even greater extent because assistive technologies are typically more noticeable and intimidating than a hearing aid. Sutherland (1995) details the following strategies for increasing consumer acceptance of assistive technologies: (1) Educating consumers about technical devices, including their strengths and limitations; (2) training consumers to use technical devices; (3) helping consumers to make informed choices; (4) providing consumers with support; (5) encouraging experienced consumers to help others who are just learning about technical devices; (6) empowering consumers by working closely with them as part of a team or partnership; and (7) aligning with consumers to advocate for better laws and services and for universal accessibility for

people with hearing loss. Wayner (2004) describes how assistive devices can be integrated into a hearing aid practice to increase awareness and acceptance. Wayner (2004) describes how an assistive device demonstration center was established in the classrooms where hearing aid orientations were performed. In this way, devices could be demonstrated in conjunction with hearing aids in difficult listening situations when hearing aids alone might not provide enough assistance. In a study of the effectiveness of the integrated center, Wayner (2004) reported that 86% of those surveyed found the center helpful and reported satisfaction with learning about assistive technologies and having the opportunity to try the technologies. In the same study, 75% of those purchasing HAT used the technology regularly and reported benefit in conjunction with their hearing aids or cochlear implants in daily activities. It is clear that integration of assistive technologies into our rehabilitative services is a best practice goal for our rehabilitative efforts. To accomplish this best practice goal, higher priority must be given to the evaluation, selection, and dispensing of assistive technologies.

LEGISLATION FOR HEARING ASSISTANCE TECHNOLOGY

During the 1980s and 1990s, the civil and education rights of individuals with disabilities were strengthened, and the important role that assistive technologies have in improving the quality of life of disabled individuals was recognized. Since 1988, federal laws specifically addressing the HAT needs of persons with disabilities have been passed.

Beginning in 1975 with the Education of All Handicapped Children Act and with the implementation of the Americans with Disabilities Act (1990) and, most recently, the 2004 reauthorization of the Individuals with Disabilities Education Act of 1997, there has been a sustained interest in removing barriers for persons with hearing loss and other disabilities. One of the ways that acoustic barriers to communication can be diminished is through the use of assistive listening technology. These technologies have been included as a reasonable accommodation under many federal laws (Education of Handicapped Children, P.L., 94-142, 1977; Education of Handicapped Children, P.L., 99-457, 1986; Education of Handicapped Children, P.L., 101-476, 1990; Individuals with Disabilities Education Act, 1997, 2004). For example, in rooms that require permanently installed assistive technologies, the availability of such technologies must be posted using the international symbol of access for persons with hearing loss (Figure 37.16).

HEARING ASSISTANCE TECHNOLOGY RESEARCH NEEDS

There is an ever-expanding need for research concerning assistive devices. This need is driven by the federally enforce-

FIGURE 37.16 International symbol of access for individuals with hearing loss.

able standards for accessibility and accommodations for hearing loss in the Americans with Disabilities Act, as well as an awareness on the part of many audiologists that, by including assistive technology in their rehabilitative plans, they can significantly improve the quality of care provided. Assistive technologies are constantly changing. As a result, one of the biggest research needs in the field of audiology is the development of protocols that can better evaluate the needs of individuals with hearing loss and a method for efficiently matching needs to technologies.

In addition to developing tools for the selection process, we must also have tools that allow us to measure the efficacy of the technology more accurately. Since any one technology might be used in a variety of situations, it is important to measure whether that technology is equally effective in all circumstances or if there are situations/populations for which that technology has a distinct advantage. Research is needed to explore ways that legislatures and rural school districts can make Internet resources and HAT more widely available in rural educational settings (e.g., the use of electronic technology could be expanded to provide instructional services to individuals in rural areas who are deaf or have hearing loss).

A final area of future research involves exploring ways to make assistive technologies more accessible and acceptable for those in need. A promising possibility is telehealth, defined as the use of telecommunications and information technologies to share information and to provide clinical care, education, public health, and administrative services at a distance. The term "teleaudiology," in turn, refers to the use of technology to provide audiology services when the

client and practitioner are in different locations. Diagnostic applications of teleaudiology have been well documented and validated (see systematic review by Swanepoel and Hall, 2010) and are in use in many areas of Africa, Australia, Europe, and North America. However, there has been less research into the use of teleaudiology applications for intervention, and even fewer studies of teleaudiology as it relates to assistive technology. Given that one of the deterrents to wider use of assistive technology might be the need for extensive or intensive instruction in device setup and use (Boothroyd, 2004), teleaudiology could provide an avenue for reducing some of this burden by allowing audiologists to use videoconferencing to schedule virtual troubleshooting appointments and refresher sessions with a minimum of travel for either party. For devices such as FM systems that require programming, remote programming (in which the audiologist's computer is connected via Internet to a local technician's computer at the patient's location) might further reduce the number and/or distance of office visits for device users. However, for all of the promise offered by these emerging technologies, research is needed to address various challenges. Unfamiliarity with teleaudiology is common and can lead to resistance when a teleaudiology option is offered (Eikelboom and Atlas, 2005). With regard to the use of videoconferencing and other telehealth interactions, some have expressed concern about confidentiality and security (Demeris et al., 2009; Stanberry, 2000). Despite the challenges associated with the growing field of teleaudiology, it offers an exciting new avenue for interacting with and assisting patients in many different domains.

SUMMARY AND FUTURE RESEARCH ISSUES

There is a growing awareness of the negative influence of room acoustics on the adequate perception of speech and on communication. The influences of distance, background noise, and reverberation are well documented. Various assistive technologies (such as induction loop, FM, and infrared assistive listening systems) and communication strategies can be used alone or together to reduce the influence of poor room acoustics on communication. This chapter also covered other assistive devices that have been designed to improve receptive communication in situations such as those involving face-to-face communication, broadcast media, telecommunications, and alerting situations as well as methods to augment the capabilities of assistive devices. For HAT to be accepted and used by an individual with hearing loss, however, the communication needs and proper selection of assistive devices must be conducted within the context of an overall rehabilitative plan. Outside of the comprehensive rehabilitative plan, there is still a need to engage in activities to improve consumer acceptance of assistive devices and to help people understand the federal mandates that are already in place to remove acoustic barriers to communication. Research is needed to improve the type and quality of assistive devices available to individuals with hearing loss and to develop better methods of identifying the individuals who will benefit most from a particular technology. HAT offers the hearing healthcare professional a significant challenge but, at the same time, a wonderful opportunity to maximize the client's communication *and* human potential.

ACKNOWLEDGMENT

The late Carl Crandell provided much of the foundation on which this chapter is built. His enthusiasm for helping people who cannot hear will not pass this way again, but his spirit lives on in us, the authors of this revision. We hope our rendering honors Carl and provides the kind of information and inspiration he would wish for the reader.

FOOD FOR THOUGHT

1. Given the evidence for the value of assistive technologies, why has such little attention been given the role and benefits of HAT within audiologist's overall rehabilitation plans?
2. A child in your classroom could benefit from additional amplification beyond hearing aids. What would be the advantages and disadvantages of advocating for a personal FM system versus a classroom audio distribution system?
3. The influence of distance, background noise and reverberation worsen the situation for those populations "at risk" for listening and learning difficulties. How might we evaluate patients who might need HAT during a rehabilitative evaluation?

REFERENCES

American Academy of Audiology Clinical Practice Guidelines. (2008) Remote microphone hearing assistive technologies for children and youth from birth to twenty-one years. Available online at: http://www.audiology.org/resources/documentlibrary/Documents/HATGuideline.pdf

American Academy of Audiology Clinical Practice Guidelines. (2011) Remote microphone hearing assistive technologies for children and youth from birth to twenty-one years: supplement B: classroom audio distribution systems–selection and verification. Available online at: http://www.audiology.org/resources/documentlibrary/Documents/20110926_HAT_GuidelinesSupp_B.pdf

American National Standards Institute (2010). ANSI/ASA S12.60-2010/Part 1, American National Standard Acoustical Performance Criteria, Design Requirements, and Guidelines for Schools, Part 1: Permanent Schools.

Anderson KL, Goldstein H. (2004) Speech perception benefits of FM and infrared devices to children with hearing aids in a typical classroom. *Lang Speech Hear Serv Sch.* 35 (2), 169–184.

Bess F. (1985) The minimally hearing-impaired child. *Ear Hear.* 6, 43–47.

Bess F, Sinclair J, Riggs D. (1986) Group amplification in schools for the hearing-impaired. *Ear Hear.* 5, 138–144.

Boothroyd A. (2004) Hearing aid accessories for adults: the remote FM microphone. *Ear Hear.* 25, 22–33.

Boothroyd A. (2005) Modeling the effects of room acoustics on speech reception and perception. In: Crandell C, Smaldino J, Flexer C, eds. *Sound Field Amplification: Applications to Speech Perception and Classroom Acoustics.* 2nd ed. Clifton Park, NY: Thomson Delmar Learning.

Bradley J. (1986) Speech intelligibility studies in classrooms. *J Acoust Soc Am.* 80, 846–854.

Bradley JS, Sato H. (2008) The intelligibility of speech in elementary school classrooms. *J Acoust Soc Am.* 123 (4), 2078–2086.

Crandell C, Bess F. (1986) Speech recognition of children in a "typical" classroom setting. *ASHA.* 28, 82.

Crandell C, Bess F. (1987) Sound-field amplification in the classroom setting. *ASHA.* 29, 87.

Crandell C, Smaldino J. (1995) An update of classroom acoustics for children with hearing loss. *Volta Rev.* 1, 4–12.

Crandell C, Smaldino J, Flexer C, Edwards C. (1997) *An update on sound field FM amplification.* Paper presented at the Annual Meeting of the American Academy of Audiology, Los Angeles, CA.

Debevc M, Peljhan Z. (2004) The role of video technology in online lectures for the deaf. *Disabil Rehabil.* 26, 1048–1059.

Demiris G, Doorenbos A, Towle C. (2009) Ethical considerations regarding the use of technology for older adults: the case of telehealth. *Res Gerontol Nurs.* 2 (2), 128–136.

Dillon H. (2012) *Hearing Aids.* New York: Thieme.

Duquesnoy A, Plomp R. (1983) The effect of a hearing aid on the speech-reception threshold of hearing-impaired listeners in quiet and in noise. *J Acoust Soc Am.* 73, 2166–2173.

Eikelboom R, Atlas M. (2005) Attitude to telemedicine, and willingness to use it, in audiology patients. *J Telemed Telecare.* 11 (suppl 2), S22–S25.

Erber N. (1979) Auditory-visual perception of speech with reduced optical clarity. *J Speech Hear Res.* 22, 213–223.

Finitzo-Hieber T, Tillman T. (1978) Room acoustics effects on monosyllabic word discrimination ability for normal and hearing-impaired children. *J Speech Hear Res.* 21, 440–458.

Flexer C. (1992) Classroom public address systems. In: Ross M, ed. *FM Auditory Training Systems: Characteristics, Selection and Use.* Timonium, MD: York Press; pp 189–209.

French N, Steinberg J. (1947) Factors governing the intelligibility of speech sounds. *J Acoust Soc Am.* 19, 90–119.

Gelfand S, Silman S. (1979) Effects of small room reverberation upon the recognition of some consonant features. *J Acoust Soc Am.* 66, 22–29.

Haas H. (1972) The influence of a single echo on the audibility of speech. *J Audio Eng Soc.* 20, 146–159.

Hygge S, Rönnberg J, Larsby B, Arlinger S. (1992) Normal and hearing-impaired subjects' ability to just follow conversation in competing speech, reversed speech, and noise backgrounds. *JSHR.* 35, 208--215.

John AB, Kreisman B. (2012) Review of classroom audio distribution system literature. In: Smaldino J, Flexer C, eds. *Handbook of Acoustic Accessibility: Best Practices for Listening, Learning, and Literacy in the Classroom.* New York: Thieme.

Johnson C. (2000) Children's phoneme identification in reverberation and noise. *J Speech Lang Hear Res.* 43, 144–157.

Klatte M, Lachmann T, Meis M. (2010) Effects of noise and reverberation on speech perception and listening comprehension of children and adults in a classroom-like setting. *Noise Health.* 12 (49), 270–282.

Knecht HA, Nelson PB, Whitelaw GM, Feth LL. (2002) Background noise levels and reverberation times in unoccupied classrooms: predictions and measurements. *Am J Audiol.* 11, 65–71.

Langlan L, Sockalingam R, Caissie R, Kreisman B. (2009) The benefit of sound-field amplification in First Nations elementary school children in Nova Scotia, Canada. *Aust NZJ Audiol.* 31, 55–71.

Leavitt R, Flexer C. (1991) Speech degradation as measured by the Rapid Speech Transmission Index (RASTI). *Ear Hear.* 12, 115–118.

Massie R, Theodoros D, McPherson B, Smaldino J. (2004) Sound-field amplification: enhancing the classroom listening environment for Aboriginal and Torres Strait Islander children. *Aust J Indigenous Educ.* 33, 47–53.

Middleweerd M, Plomp R. (1987) The effect of speechreading on the speech reception threshold of sentences in noise. *J Acoust Soc Am.* 82, 2145–2146.

Moore B. (1997) *An Introduction to the Psychology of Hearing.* San Diego, CA: Academic Press.

Morrow SL, Connor NP. (2011) Voice amplification as a means of reducing vocal load for elementary school teachers. *J Voice.* 25 (4), 441–446.

Mulrow C, Christine A, Endicott J, Tuley M, Velez R, Charlip M, et al. (1990) Quality-of-life changes and hearing loss. *Ann Intern Med.* 113, 188–194.

Nabelek A. (1982) Temporal distortions and noise considerations. In: Studebaker G, Bess F, eds. *The Vanderbilt Hearing-Aid Report: State of the Art Research Needs.* Upper Darby, PA: Monographs in Contemporary Audiology.

Nabelek A, Nabelek I. (1994) Room acoustics and speech perception. In: Katz J, ed. *Handbook of Clinical Audiology.* 4th ed. Baltimore, MD: Williams & Wilkins.

Needleman A, Crandell C. (1996) Speech perception in noise by hearing impaired and masked normal hearing listeners. *J Am Acad Audiol.* 2, 65–72.

Neuman A, Hajicek J, Rubinstein A. (2010) Combined effects of noise and reverberation on speech recognition performance of normal-hearing children and adults. *Ear Hear.* 31 (3), 336–344.

Payton K, Uchanski R, Braida L. (1994) Intelligibility of conversational and clear speech in noise and reverberation for listeners with normal and impaired hearing. *J Acoust Soc Am.* 95, 1581–1592.

Pearsons K, Bennett R, Fidell S. (1977) *Speech Levels in Various Noise Environments.* EPA 600/1-77-025. Washington, DC: Office of Health & Ecological Effects.

Plomp R. (1978) Auditory handicap of hearing loss and the limited benefit of hearing aids. *J Acoust Soc Am.* 75, 1253–1258.

Plomp R. (1986) A signal-to-noise ratio model for the speech reception threshold for the hearing impaired. *J Speech Hear Res.* 29, 146–154.

Rosenberg G, Blake-Rahter P. (1995) In-service training for the classroom teacher. In: Crandell C, Smaldino J, Flexer C, eds. *Sound-Field FM Amplification: Theory and Practical Applications.* San Diego, CA: Singular Publishing Group; pp 107–124.

Sarff L. (1981) An innovative use of free field amplification in regular classrooms. In: Roeser R, Downs M, eds. *Auditory Disorders in School Children.* New York: Thieme-Stratton; pp 263–272.

Schum D. (1996) Intelligibility of clear and conversational speech of young and elderly listeners. *J Am Acad Audiol.* 7, 212–218.

Siebein G, Crandell C, Gold M. (1997) Principles of classroom acoustics: reverberation. *Educ Audiol Monogr.* 5, 32–43.

Smaldino J, Crandell C, Kreisman B, John AB, Kreisman N. (2007) Room acoustics for listeners with normal hearing and hearing impairment. In: Valente M, Hosford-Dunn H, Roeser R, eds. *Audiology Treatment.* New York: Thieme.

Smaldino J, Flexer C. (2012) *Handbook of Acoustic Accessibility: Best Practices for Listening, Learning, and Literacy in the Classroom.* New York: Thieme.

Spicer J, Schmidt R, Ward CD, Pinnington LL. (2005) Evaluation of text telephones designed for people with impaired hearing or speech. *J Med Eng Technol.* 29, 137–144.

Stanberry B. (2000) Telemedicine: barriers and opportunities in the 21st century. *J Int Med.* 247, 615–628.

Sutherland G. (1995) Increasing consumer acceptance of assistive devices. In: Tyler RS, Schum DJ, eds. *Assistive Devices for Persons with Hearing Loss.* Needham Heights, MD: Allyn and Bacon; pp 251–256.

Swanepoel D, Hall JW. (2010) A systematic review of telehealth applications in audiology. *Telemed J E Health.* 16 (2), 1–20.

Tye-Murray N. (2009) *Foundations of Aural Rehabilitation.* 3rd ed. Clifton Park, NY: Delmar Cengage Learning.

Vesterager V, Salomon G. (1991) Psychosocial aspects of hearing loss in the elderly. *Acta Otolaryngol Suppl.* 476, 215–220.

Wayner D. (2004) Integrating assistive technology into a hearing aid practice. *Hear J.* 57, 43–45.

Weinstein B. (2000) Outcome measures in rehabilitative audiology. In: Alpiner J, McCarthy P, eds. *Rehabilitative Audiology.* Philadelphia, PA: Lippincott Williams & Wilkins; pp 575–594.

CHAPTER 38

Hearing Aid Technology

Jennifer Groth and Laurel A. Christensen

INTRODUCTION

This chapter serves as an introduction to hearing aid technology with a discussion of the current state of hearing aids, including how hearing aids work to process sound and deliver this sound to the hearing aid user's ear. The chapter will also include an overview of the styles of hearing aids, hearing aid measurement, and wireless connectivity in hearing aids.

HEARING AID STYLES COMMONLY USED TODAY

Hearing aids used today can be broadly classified into two styles: Those that fit behind-the-ear (BTE) or more specifically are worn over the pinna (commonly called BTE hearing aids) and those that fit in-the-ear (ITE) or in the concha and ear canal (commonly called ITE hearing aids). A BTE hearing aid is coupled to the ear with tubing and either an earmold made custom for the hearing aid user's ear or with an ear dome, also commonly called an ear tip. Figure 38.1 shows an example of an ITE and a BTE hearing aid as worn by a hearing aid user.

Within these two broad categories are many different specific styles and sizes of hearing aids. When considering what style of hearing aid is appropriate for a patient, many factors are taken into account including the ability of the patient to manipulate the controls on the hearing aids and change the battery and the features contained within the hearing aid. Generally, the smaller the hearing aid, the harder it is to manipulate the controls and change the battery. Choosing a style of hearing aid is also related to an individual's cosmetic preference. For the most part, the signal processing can be implemented in any style of hearing aid with the exception of a few features such as telecoils (a feature for phone communication and looping and directionality). If a telecoil is a desired feature, a hearing aid style with a telecoil built inside must be selected. Directionality, a feature that helps hearing aid users to hear better in noise, cannot be implemented in the smallest ITE devices. The largest percentage (about 71%) of hearing aids sold in the United States is BTE hearing aids (Hearing Industries Association (HIA), 2012).

Behind-the-Ear

BTE hearing aids can be further broken down into those with the receiver located within the case of the hearing aid (traditional BTEs) and those with the receiver removed from the case and instead located at the end of the tubing and placed inside the ear canal (receiver-in-the-ear (RIE)). RIE aids have also been termed receiver-in-the-canal (RIC) but for the purposes of this chapter the term RIE will be used. Figure 38.2 shows a traditional BTE hearing aid and an RIE hearing aid. The traditional BTE aid is shown with tubing and an earmold attached to the aid. The RIE is also shown with tubing which includes the receiver and an ear dome for placement into the ear. A better view of the actual receiver at the end of the tube is shown in the circled image

FIGURE 38.1 An example of an in-the-ear (ITE) and a behind-the-ear (BTE) hearing aid as worn by a hearing aid user. (Courtesy of ReSound (**left**) and Starkey Laboratories (**right**).)

FIGURE 38.2 An example of a traditional BTE hearing aid and a receiver-in-ear (RIE) hearing aid. The traditional BTE aid is shown attached to tubing and a custom earmold. The RIE hearing aid is shown with the thin receiver tube attached to the aid and a standard ear dome. The receiver which is at the end of the receiver tube is shown close up in the *circle.* The dome is placed on the receiver and this part is placed in the ear canal of the hearing aid user. (Courtesy of Phonak (**left**) and Beltone (**right**).)

of Figure 38.2. Both traditional BTEs and RIE hearing aids can be worn in an "open" configuration where the coupling includes a tube to the ear and the ear domes do not completely occlude or plug up the ear. These "open" fittings have made BTE hearing aids the most popular styles available today. Open hearing aid styles are particularly good for hearing aid users with normal hearing in the low frequencies because the low-frequency sound can exit the ear canal as it does for listeners with normal hearing. Open styles using thin tubes are generally more cosmetic, comfortable, and better sounding because of this reduction of occlusion.

Traditional BTEs

Traditional BTE hearing aids come in different sizes and are primarily defined by the hearing aid battery that they use and how they are coupled to the ear. Figure 38.3 shows several BTE hearing aids. The one on the left would be fit to the patient with a length of plastic tube attached to a custom-made earmold, whereas the other two are configured to use thin tubing and would be fit to the ear with either a noncustom plastic tip or custom-made earmold. These BTE aids use battery sizes 312, 13, and 675, respectively. As a general rule, the larger the battery used, the more gain the hearing aid is capable of producing. However, the battery size mainly determines how long the patient can use the hearing aid before having to change the battery, with the larger batteries lasting longer. Today, the only hearing aids that use the largest batteries (size 675) are for fitting patients with severe-to-profound hearing losses. As stated earlier, BTE hearing aids are coupled to the ear with a tube running from the aid to the ear with either a custom earmold or an ear dome for placement in the ear.

FIGURE 38.3 Examples of different sizes of traditional BTE hearing aids. (Courtesy of Beltone.)

Receiver-in-the-Ear

RIE hearing aid style makes up over 45% of the hearing aids sold in the United States (HIA, 2012). This may be different in other countries. RIE hearing aids use a thin tube and couple to the ear with an ear tip or an earmold called a micro mold. An RIE is like a BTE device in that part of the instrument fits behind the ear, but the receiver is located at the end of the tube within the ear canal. A small wire cable running through a thin tube connects the device behind the ear to the receiver (see Figure 38.3). RIE devices have become popular in the last few years because of their cosmetics and thin-tube options; however, there are thin-tube options in traditional BTE devices as well. To determine if there are benefits to the RIE style over the traditional BTE, Hallenbeck and Groth (2008) studied the gain (amount of amplification provided to the hearing aid user) before feedback (squealing in hearing aids) in the two styles as this was thought to be one of the reasons that RIE aids had become so popular. Their study concluded that similar gain could be achieved in the two styles and should not be used as a reason to select an RIE product over a traditional BTE. The study did point out that RIE hearing aids potentially offer a smoother, wider frequency response and that there are no moisture problems associated with the tube because of the design. The drawback of RIE hearing aids is that the receiver being located in the ear might cause it to malfunction more often and the receiver is significantly more expensive to replace than a thin tube.

In-the-Ear

ITE hearing aids come in many specific styles and make up approximately 29% of the hearing aids sold in the United States (HIA, 2012). Like BTE hearing aids, this may be

FIGURE 38.4 Examples of ITE hearing aids. [Courtesy of ReSound.]

slightly different in other countries. ITE hearing aids are for the most part custom devices made specifically to fit an individual's ear. There are, however, a few styles that fit into the ear that come in standard, noncustom sizes.

Figure 38.4 shows a variety of ITE hearing aids in different sizes. The largest ITE hearing aid fits completely into the concha and is termed a full-shell ITE. Smaller ITE styles include half-shell styles that partially fill the concha and in-the-canal (ITC) styles where most of the hearing aid is within the ear canal. The completely-in-the-canal (CIC) style fits within the ear canal and terminates at the opening of the ear canal. Finally, a recent style termed an invisible-in-the-canal (IIC) device has been introduced. Figure 38.5 shows this style of hearing aid in an ear. An IIC fits completely in the canal and terminates just before the ear canal opening. It is not visible from outside the ear. In general, more power and output are obtained in the full-shell style. However, because the receiver can be placed closer to the tympanic membrane in a smaller volume in the ear for the IIC and CIC styles, these aids can produce enough gain to fit hearing losses up to the severe range. ITC hearing aids are the smallest ITE styles where directionality using two microphones to improve hearing in noise can be achieved.

FIGURE 38.5 An invisible-in-the-canal (IIC) hearing aid. [Courtesy of ReSound.]

FIGURE 38.6 An example of an MIC hearing aid. [Courtesy of ReSound.]

One additional style of hearing aid is the microphone-in-concha (MIC) design. In this style the microphone has been removed from the hearing aid case and is attached to a wire and tubing and placed in the helix. This type of design allows for several advantages over other ITE styles because the removal of the microphone from the case frees up space for several options including a larger receiver in a smaller style to achieve greater amplification in a more cosmetic package; a very large vent that will make the device less occlusive in the ear; or to build a smaller hearing aid that fits deeper in the ear canal. Figure 38.6 shows an example of MIC hearing aid.

Noncustom ITE Styles

There are some ITE styles that are not custom-made products but do fit in the ear. Figure 38.7 shows examples of three of these types of products. These products are placed in the ear but are not made individually for a patient. Generally these hearing aids do not cost as much as custom-made or BTE devices and they do not have as many features as the other styles overall. Some of these devices are being sold over the counter. If the manufacturer of such devices has not attained Food and Drug Administration (FDA) approval for them as medical devices, they are termed personal sound amplification products (PSAPs) rather than "hearing aids." PSAP is a category of devices defined by the FDA that is "not intended to compensate for impaired hearing, but rather is intended for non-hearing-impaired consumers to amplify sounds in the environment for a number of reasons, such as recreational activities" (FDA, 2009). They are often compared to reading glasses for the ear and typically marketed for part-time use.

FIGURE 38.7 Examples of noncustom ITE hearing aids. (Courtesy of ReSound (**left**), Etymotic Research (**center**), and Siemens (**right**).)

HOW A HEARING AID WORKS

This involves a complex interaction between the device and the individual who uses it. Hearing aids of excellent quality meeting all technical specifications may not always be judged to "work" by the user. The many factors which contribute to the ultimate effect of wearing amplification are discussed elsewhere in Section IV of this book. In this chapter, we limit ourselves to the purely technical aspects of the hearing aid itself.

Components

Hearing aids are wearable electronic amplifiers of sound in the environment that are used to assist with communication for a hard-of-hearing person. At a minimum, hearing aids require a microphone to pick up the sound and convert it to an electrical signal, electronic circuitry to amplify and treat the signal, a speaker—called a "receiver"—to convert the signal back to sound waves, and a battery to power the device. An example of how these components may be assembled in a BTE device is shown in Figure 38.8.

Microphone

The microphone is one of the hearing aid's *transducers.* A transducer converts one form of energy to another. The microphone contains a diaphragm that is set into vibration by the pressure variation of sound waves that enter the opening of the microphone, often called a *port.* The motion of the diaphragm transduces the acoustical energy (sound) to electrical energy. Although various microphone technologies have historically been used in hearing aids, virtually all devices now have either *electret* or, increasingly, *microelectrical-mechanical system (MEMS)* microphones. These microphone technologies offer high-quality technical performance, can be very small, and are well suited to mass production. Hearing aid microphones can faithfully transduce sounds over a broad range of frequencies as well as a large span dynamic range and contribute very little noise to the processed sound that exits the hearing aid. Compared to other hearing aid components, microphones impose the fewest limitations on overall hearing aid electroacoustic design and performance.

Digital Signal Processor

The circuitry which manipulates the signal in a hearing aid has traditionally been referred to as the *amplifier.* The amplifier has traditionally been thought of in terms of how it increased the level of the signal at different frequencies. Although the goal of hearing aids is still to amplify sound for compensation of hearing loss, the circuitry in modern hearing aids treats the signal in many other ways to accomplish

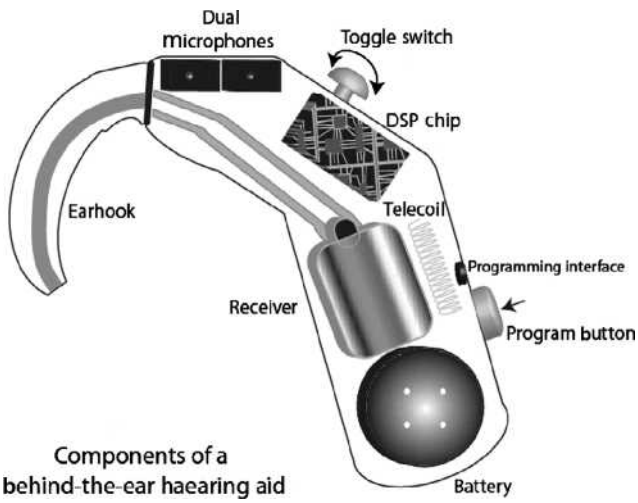


FIGURE 38.8 Schematic of a behind-the-ear (BTE) style hearing aid showing the major components of a modern hearing aid. (Courtesy of Derek Stiles © 2006.)

additional goals, such as noise reduction, prevention of hearing aid squealing, and analysis of the acoustic environment.

Prior to 1996, hearing aids primarily used analog processing. However, it is safe to say that all modern hearing aids are based on digital sound processing. Digital hearing aids became widely commercially available in 1996, with sales completely overtaking that of analog hearing aids within the first few years of the 21st century. Because of the development and production costs relative to sales, it is no longer feasible for hearing aid manufacturers to manufacture analog hearing aids.

How do analog and digital hearing aids differ? Sound is an analog signal, meaning that it is continuous. In analog hearing aids, the time-varying pressure variations of sound are represented by uninterrupted, time-varying changes in electrical voltage. The continuous electrical voltage changes are *analogous* to the continuous changes in the original acoustic signal. The manipulation of the signal that is carried out by the analog circuitry is virtually instantaneous.

In contrast, digital hearing aids represent sound by analyzing it at discrete intervals and converting it to a series of numbers. Processing of the resultant signal occurs by applying *algorithms,* which are sets of arithmetic operations. For example, to amplify a digital signal requires only multiplying the numbers by an amount that would yield the desired output.

The digital circuit in a modern hearing aid consists of a minimum of subcomponents, including the *A/D (analog-to-digital) converter, digital sound processor (DSP), memory, clock,* and *D/A (digital-to-analog) converter.* Of these, the DSP corresponds to what is traditionally called the amplifier, carrying out all the signal processing algorithms. The capabilities of the DSP in modern hearing aids far exceed what was possible in analog hearing aids. However, depending on the technical characteristics of the digital system, there can be limitations that were not found with analog hearing aids. One example of this is that the dynamic range of input levels is smaller for digital hearing aids than for analog hearing aids.

The precision with which the sound can be represented digitally, the signal processing capabilities of the hearing aid, and the time it takes for the signal to be processed depend on a number of digital circuit characteristics. An overview of some of these characteristics is presented in Table 38.1.

Receiver

Like the microphone, the receiver is also a transducer. It converts the processed electrical signal back to acoustic sound waves. The principle is similar to that of the microphone insofar as a diaphragm is set into vibration. The movement of the diaphragm in this case creates the sound waves that are produced by the hearing aid, and the sound waves travel through tubing that connects the receiver to the outside of the hearing aid. Physical properties of the receiver components and the tubing, as well as magnetic forces that drive the vibration of the diaphragm are deciding factors for the output and frequency response of the receiver. The hearing aid receiver most often also incorporates the function of digital-to-analog conversion.

Battery

The battery provides the electrical energy to hearing aids. The most common type of hearing aid battery is the zinc–air. It has small holes that allow oxygen to enter the cell when

TABLE 38.1

Digital Circuit Characteristics Overview

	Unit	What It Means	What Effect It Has
Sampling rate	Typically kHz	How often the incoming signal is sampled per unit of time	Determines the frequency range of sound that can be represented
Bit depth	bits (binary digits)	Refers to the length of the digital "words" that describe the signal; also called the resolution	Increasing bit depth reduces noise in the signal
Clock frequency	Typically MHz	Processor speed	Coordinates arithmetic operations and data transfer; a higher clock speed can "schedule" activities faster and more efficiently
Millions of instructions per second	MIPS	Computational power	Increasing MIPS capacity allows the hearing aid to run more algorithms simultaneously, or more complex algorithms

the user peels off a sticker, causing a chemical reaction that activates the battery. The amount of energy that is stored in the battery is called the *capacity* and is given in milli-Ampere hours (mAh). Larger batteries can store more energy and thus provide more hours of use than smaller batteries. The combination of how much electrical current in milli-Amperes (mA) is drawn by the hearing aid and the capacity of the battery allows an estimation of battery lifetime. For example, a hearing aid battery may have a capacity of 90 mAh. If the hearing aid draws 1 mA of current, then the estimated hours of use per battery is 90 mAh/1 mA = 90 hours. In real-life use, current consumption by the hearing aid is variable, which means that actual battery lifetime is virtually always shorter than estimated in this way. Operating conditions such as humidity and temperature also affect battery performance, and thus, hours of hearing aid use per battery.

Until recently, zinc–air batteries have contained small amounts of mercury amalgamated with zinc. The purpose of the mercury content was to prevent gas from forming inside the battery. This could cause the cell to swell and its components to separate and leak. Although mercury has been banned from other types of batteries since the mid-1990s, zinc–air batteries for hearing aids were exempted because of lack of an alternative technology. Numerous states in the United States, as well as other countries, began to enact bans of the sale of zinc–air batteries containing mercury in 2011. After many years of research, mercury-free zinc–air batteries are now available that provide a reliable energy source for hearing aids and which are largely on par with mercury-containing zinc–air technology.

Rechargeable battery technology is available in virtually every consumer small electronic product, so one might expect hearing aids also to make use of rechargeability. However, rechargeable hearing aids are still a rarity. Hearing aids need a power supply that is small enough to fit inside the device and that has great enough energy density (energy per volume) to power the device for a defined unit of time (1 day of use would be a minimum). Currently, zinc–air batteries are the only technology that can meet these requirements, as rechargeable battery technologies have comparatively low energy capacity and low energy density. This means that a rechargeable cell with the same capacity as a given size of zinc–air battery would need to be much larger. For now, rechargeable solutions are available only for hearing aids with low requirements for power consumption.

Other

There are other components that are common with certain styles of hearing aids or used for specific patient needs.

User Controls

User controls include on/off switches, volume controls, and program buttons. A hearing aid may have any combination of these or none of these. Some hearing aids also have user controls that have more than one function. For example, the on/off switch may be part of the volume control, or the volume control and control to change programs may be combined in one toggle switch. User controls can also be located on a remote control device.

Multiple Memories

Most of today's hearing aids have the capability of multiple memories. Within a memory, certain hearing aid adjustments are stored, and various hearing aid features can be turned on or off. The patient can access a given memory through the use of a button on the hearing aid or using a remote control device. The presumed benefit of multiple memories is that very specific hearing aid settings may be desirable for one listening situation but not another. For example, many audiologists believe that the hearing aid settings for listening to music should be different from those for listening to speech in background noise. The patient, therefore, could simply switch the hearing aid to the "music program" whenever needed. As will be discussed later, hearing aids also have signal classification systems, that is, the hearing aid can detect the content of the input signal (e.g., music vs. speech vs. noise). Automatic adjustments to the signal processing can be made based on the content. As these algorithms have become more advanced, there has been less need for multiple memories. However, new functionality in hearing aids, such as digital wireless connectivity, continues to make multiple memory capability relevant.

Programming Interface

A programming interface refers to a socket or connector, usually on the body of the hearing aid or inside the battery compartment that allows connection to a cable or boot. This can be used for programming the hearing aid and/or for attaching an external audio input device. Hearing aids are becoming available that can only be programmed wirelessly and do not have a mechanical programming interface.

Telecoil

Historically, hearing aids and telephones have not worked well together without some sort of interface. This was because of the feedback that results from placing the telephone receiver in close proximity to the hearing aid microphone (and receiver). Feedback management features have helped in making phone communication easier, but there are still individuals who struggle to use the telephone. There are several ways to effectively couple hearing aids and telephones. One of these is through a telecoil. A telecoil is a tiny coil of wire around a core that induces an electrical current when it is in the presence of a changing magnetic field. Originally, it was intended to "pick up" the magnetic signal generated by the older telephones, whose speakers were driven by powerful magnets. Newer phones often do not carry the strong magnetic signal but contain extra

electronics to generate such a signal and are referred to as "hearing aid compatible" (HAC), allowing the hearing aids to pick up the magnetic signal from the phone. The induction coil is formed by wrapping copper wire many times around a metal rod; the strength of the inductive pick up is determined by the number of turns of the copper wire around the metal axis rod. Larger rods permit more turns and more powerful telephone coils. By using an integrated amplifier to amplify the strength of the signal, the size can be reduced for hearing aid use. The strength of the electric current induced in the telecoil is directly proportional both to the energy in the magnetic field and to the relative positions of the induction coil in the hearing aid to the magnetic field generated from the telephone. This means that, in some positions, little or no electric current will be created in the induction coil. This is why hearing aid users must often experiment with the positioning of unfamiliar telephones to find the "hot spot" where the strongest signal is heard.

When a hearing aid is in the telecoil mode, nearby electromagnetic signals are detected and amplified, including those that are not the desired signal. Some common sources of electromagnetic interference include powerful fluorescent lights, microwaves, televisions, computer monitors, power lines, and electrical transformers. Any of these electrical devices can produce strong electromagnetic "static" or noise. This electromagnetic static can interfere with the telecoil and with telephone reception in general. Because the strength of the electromagnetic field often varies considerably with small changes of position, it is sometimes possible to minimize the amount of the noise just by moving the telephone or hearing aid position slightly.

The telecoil can also be used to pick up electromagnetic fields generated by electric currents traveling through wires, such as induction loop systems (e.g., used in public facilities such as places of worship) or neck loops (e.g., connected to FM receivers). See Chapter 37 for more in-depth discussion of this topic. A coil similar to a telecoil may also be used in some hearing aids to receive and emit digital data and audio signals.

Radio

Some hearing aids contain a radio and an antenna for sending and receiving digital data and audio signals. Such hearing aids use wireless technology based on radio frequency (RF). These will be discussed in the section on digital wireless technology.

Direct Audio Input

Direct audio input (DAI) is a connection found on BTE and some RIE-style hearing aids that allows an analog electrical audio signal to be delivered directly to the hearing aid. It is sometimes the same physical connection as used for the programming interface. An adaptor called an audio boot is attached to this connection, and the user can plug in audio devices via a cable. FM receivers can also be plugged into the audio boot. Some audio boots have integrated FM receivers. The use of FM is discussed in detail in Chapter 37.

Acoustic Coupling to the Ear

Earmolds and Custom Shells

Earmolds and custom shells are fabricated from an impression of the ear. The purpose of the custom shell is to house the electronics of an ITE hearing aid so that it fits within the individual ear, directing the amplified sound into the user's ear canal. For BTE and RIE hearing aids, an earmold may be used to direct the acoustic signal into the ear canal and to assist with retention of the device on the ear.

As with custom ITE hearing aids, custom earmolds come in various styles, ranging from large models that fill the entire concha of the outer ear to skeleton molds in which only a small piece of tubing extends into the ear canal. Custom earmolds can also be made with an opening to insert the receiver of RIE styles or with the receiver encased. Custom earmolds are usually made of a hard acrylic or a soft silicone material. In general, the greater the hearing loss is, the larger the earmold needed. Figure 38.9 shows the samples of many of the earmold styles that are available.

Once the earmold is coupled to the hearing aid, the properties of the sound reaching the user's ear are changed. The acoustic properties of the earmold itself and the length and diameter of the connecting tube play an important part in the final acoustic characteristics of the hearing aid system. To some extent, the signal can be modified by making changes to the earmold. The most common modification is called a vent, or a small hole drilled into the canal portion of the earmold. Earmolds (and custom hearing aids) are vented for four primary reasons: (1) To allow unwanted

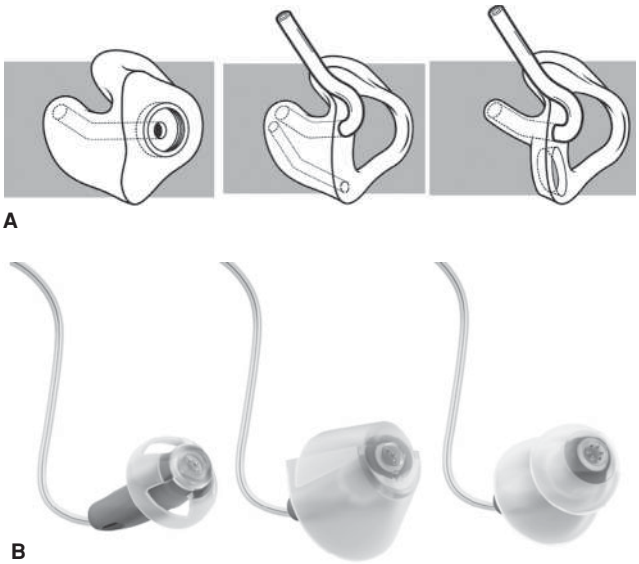


FIGURE 38.9 Earmold styles for BTE hearing aids. [Courtesy of Westone Laboratories [**A**] and Oticon A/S [**B**].]

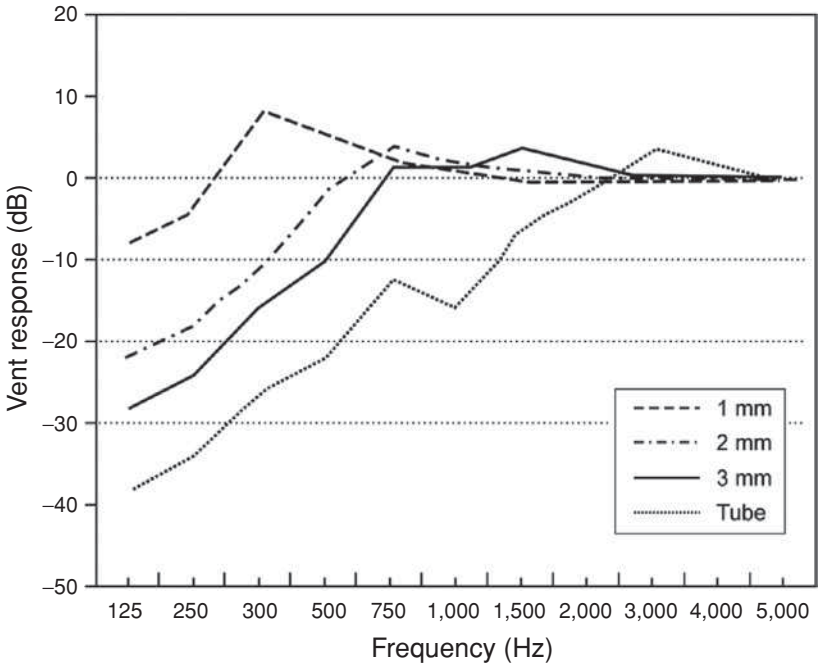


FIGURE 38.10 Example of venting effects. [Adapted from Lybarger S. [1985] Earmolds. In Katz J, ed. *Handbook of Clinical Audiology*. 3rd ed. Baltimore, MD: Williams and Wilkins; pp 885–910].

amplified low frequencies to escape from the ear canal; (2) to release pressure to avoid a "plugged ear" sensation; (3) to reduce the occlusion effect (one's own voice sounds "hollow"); and (4) to allow the normal input of unamplified sound (Mueller et al., 2006). Variable vents are also available that use small plastic plugs or different sizes of tubing that can totally occlude an existing vent or provide smaller openings of various diameters.

Because a vent allows primarily low-frequency energy to enter and leave the ear canal, the effect of venting can be observed as a reduction in low frequency gain. As the size of the vent increases, the degree of reduction increases. The effects of different vent sizes can be seen in Figure 38.10. In general, it is believed that for frequencies where there is normal hearing, it is better to have the hearing aid user hear "natural" sound than amplified sound. Hence, the vent size is selected to correspond with the range of normal hearing in the low frequencies.

Whereas venting can be applied to both ITE hearing aids and earmolds, other physical changes can be made to earmolds that affect the acoustic response (Figure 38.11).

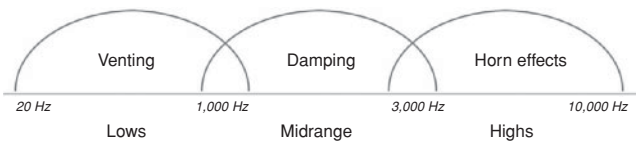


FIGURE 38.11 Venting, dampers and filters, and the sound bore affect different portions of the frequency response. [Redrawn from Libby ER. [1980] Smooth wideband hearing aid responses – the new frontier. *Hear Inst*. 31 [10], 12–18.]

One of these is the sound bore, which is the tube through which sound passes through the earmold to the ear canal of the user. Whereas venting affects the low frequency response of the sound in the ear canal, the sound bore mainly affects the high frequencies. For example, a sound bore with a flared shape (called a *horn* or *Libby horn*) can enhance the high frequencies. In contrast, narrowing the diameter of the sound bore damps the high frequencies. With modern hearing aids, where the digital filtering allows for quite flexible frequency shaping, it has become less common to change earmold acoustics to manipulate the hearing aid response in the ear.

Finally, as part of the overall plumbing of the BTE hearing aid, it is common for manufacturers to place a damper in the tone hook (that fits over the ear) to smooth the response in the 1,200- to 1,600-Hz range. Some manufacturers provide different hooks with different size dampers.

Noncustom Coupling

In recent years, there has been an increase in "open" fittings. An open fitting is one where the coupling to the ear canal allows low-frequency energy to enter and escape the ear canal unhindered. Although this can be attained with a custom earmold and an effective vent, it is most common to use a soft silicone dome-shaped tip. An example of this can be seen in Figure 38.9. It is convenient to fit hearing aids with such tips, as it is not necessary to take an impression of the ear canal and wait for a custom earmold to be manufactured. Because of this convenience, noncustom tips that occlude the ear canal have also come into regular use for fitting of more severe hearing losses, particularly with RIE styles.

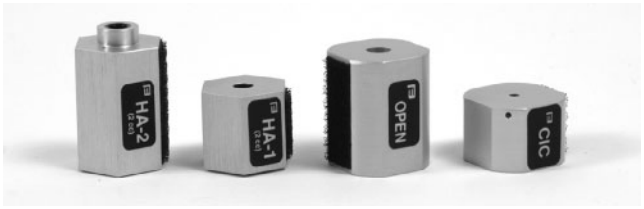


FIGURE 38.12 Examples of hearing aid couplers used to test different hearing aid styles. [Courtesy of Frye Electronics.]

ELECTROACOUSTIC PERFORMANCE

Measurement and Standards

Hearing aid performance must be assessed to make sure that a hearing aid is working as defined by the manufacturer according to a test standard. There are two different types of hearing aid measurements: Coupler tests and real-ear measures (REMs). REM uses a small probe microphone in the ear canal to verify a hearing aid's performance in the user's ear. These measures are the subject of another chapter in this book. Coupler measures are performed inside an acoustically controlled hearing aid test box using a coupler. A coupler is an open metal cavity that substitutes for the ear canal simulating its residual volume. Figure 38.12 shows coupler examples used for different styles of hearing aids. Coupler tests should be performed on all hearing aids to ensure they are meeting specifications according to a standard termed ANSI S3.22 1996 or 2003 or the applicable international standard such as IEC 118 (see also Chapter 39). The measures outlined in these standards provide a level of quality control for hearing aids. Manufacturers are required to include the results of these standards with the hearing aid so that the performance can be tested in individual clinics before fitting the hearing aid to a patient's ear. A series of tests according to the standard are run on the hearing aid to verify its performance.

Figure 38.13 shows two common test box systems used to measure hearing aids. To perform coupler measurements, a hearing aid is attached to the appropriate coupler. The coupler and the hearing aid are then placed into an acoustically controlled environment (test box) which contains a speaker or speakers used to generate sounds. At one end of the coupler a microphone (termed a coupler microphone) is inserted. Figure 38.14 shows the configuration for testing in a hearing aid test box for an RIE hearing aid. The coupler microphone measures the output of the hearing aid to calibrated signals presented through the speakers in the test box.

Some of the tests performed include tests of the amount of amplification provided by the aid throughout the hearing

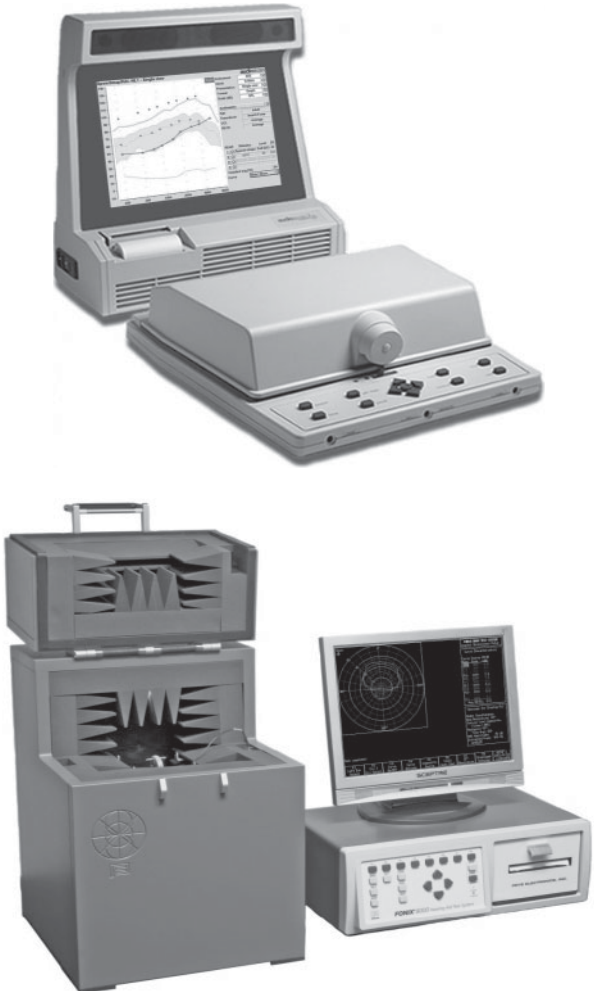


FIGURE 38.13 Two examples of hearing aid test boxes. [Courtesy of Audioscan [**top**] and Frye Electronics [**bottom**].]

FIGURE 38.14 Configuration of the hearing aid, coupler, and microphone for hearing aid testing. [Courtesy of Audioscan.]

aid's frequency response to various input signal levels, the bandwidth of the hearing aid, the OSPL90 or maximum output of the hearing aid, the amount of distortion and internal noise, and measures regarding the function of the amplification system.

SIGNAL PROCESSING

When sound enters the hearing aid it is processed by the digital signal processor mathematically. These signal processing algorithms will be discussed in this section.

Audibility

The most important signal processing that takes place in the DSP of a hearing aid is the amplification of the sound so that it becomes audible to the listener. Simply put, sound entering the hearing aid is provided gain or amplified so that it can be heard by the hearing aid user. Three terms must be defined to understand how sound is amplified in a hearing aid:

- Input: The signal entering the microphone of the hearing aid. It is characterized in terms of frequency (Hz) and intensity (dB SPL).
- Output: The signal that enters the hearing aid user's ears, which is also described in terms of frequency (Hz) and intensity (dB SPL). The output is higher than the input because the sound is amplified.
- Gain: The difference in decibels between the input level and the output level. This is how much the input was amplified. The function showing how the gain varies according to the frequency of the sound is called the "frequency–gain response," or often just "frequency response."

Linear Amplification

There are two common ways to provide amplification in a hearing aid, linear amplification and amplitude compression. Hearing aids using linear amplification often utilize a form of compression to limit the output when the OSPL90 (maximum output) of the device is reached. Linear amplification applies the same gain to the input until the hearing aid reaches the OSPL90. For example, a hearing aid with 20 dB of linear gain would amplify a 45-dB SPL input signal to 65 dB SPL and a 55-dB SPL input signal to 75 dB SPL. The amount of gain, in this case 20 dB, applied to the input signal is independent of the level of the sound entering the hearing aids (input level). When the OSPL90 is reached, the output is limited either by peak clipping or by amplitude compression limiting (discussed below). Peak clipping cuts off the peaks of the input signal that exceed the OSPL90. Peak clipping is not the preferred method of limiting output because this technique adds distortion to the hearing aids and compromises the sound quality. Compression limiting reduces the gain when the input reaches a certain level, called the kneepoint, and thus limits the output. This technique provides a better sound quality than peak clipping and will be discussed in more detail below.

Amplitude Compression

Most hearing aids today use amplitude compression to apply a different amount of gain depending on the level of the input of the incoming signal rather than using linear amplification. Amplitude compression is used in two ways in modern hearing aids; the first discussed earlier is to limit the output of a linear hearing aid. Limiting the output is necessary to protect patients from exposure to amplified sound that exceeds their levels of loudness comfort. This approach is commonly called compression limiting. The second is called wide dynamic range compression (WDRC). The term "wide" refers to the fact that this type of amplitude compression is applied over a wide range of input levels and distinguishes it from other compression schemes that work over a smaller range of input levels, and which are less commonly used today. WDRC applies greater gain to soft sounds and less gain to loud sounds. In this way, it can be applied to restore normal loudness to a hearing aid user within his/her reduced dynamic range. Remember that individuals with sensory/neural hearing loss (SNHL) have a reduced dynamic range. The normal-hearing person has a dynamic range of approximately 100 dB. For this person, 0-dB HL sounds are "very soft" and 100-dB HL sounds are "too loud." A listener with SNHL has a reduced dynamic range which is defined by his/her threshold at a particular frequency and his/her loudness discomfort level (LDL) at that same frequency. Thus, a patient with a 50-dB HL threshold at a frequency and a 95-dB HL LDL has a dynamic range of 45 dB at that frequency. In this case, 50 dB HL is "very soft" and 95 dB HL is "too loud." The loudness growth from "very soft" to "too loud" is steep. The amplitude compression strategy attempts to place the amplified signals within the hearing aid user's dynamic range restoring loudness growth so that soft sounds are still perceived as soft and loud sounds are perceived as loud. This philosophy is in line with making sure all the signals are audible to the listener (within his/her dynamic range) but still comfortable. Loud inputs in the WDRC strategy are often given no gain and are thus transparently processed through the hearing aid.

AGCi and AGCo

Amplitude compression can be referenced to either the input to the hearing aid or the output of the hearing aid. Although the technicalities of the level detection may differ among hearing aids, the conceptual distinction is whether the level detection that controls the compression is done before or after gain is applied. This gain includes the setting of the user-operated volume control if present on the

device. Input-referenced compression detects the level of the sound prior to amplification and applies a certain amount of gain depending on that level. Input-referenced compression is called "AGCi," which stands for "automatic gain control." The "i" refers to "input." The WDRC scheme described above is an example of AGCi amplitude compression. If a hearing aid with AGCi processing has a volume control, turning the volume up or down will have no effect on the gain applied by the compressor, because the gain depends only on the intensity of the sound before it is amplified.

Output-referenced compression detects the level of the sound *after* it has been amplified. This type of compression is called AGCo, where the "o" refers to "output." Output-referenced compression can be used in combination with both linear amplification as well as AGCi and is nearly always used to limit the output of the hearing aid. If a hearing aid with AGCo has a volume control, turning the volume up or down will affect whether the compression is activated and how much gain is subsequently applied to the signal.

Compression Characteristics

The characteristics of a compressor are defined by the compressor's threshold of compression (kneepoint), compression ratio, and time constants (attack and release times).

Kneepoint and Compression Ratios

The kneepoint of a compressor is the level of an incoming sound that is loud enough to trigger the compressor. A common way to display the function of a compressor is using an input/output function. Figure 38.15 shows different input/output functions for output (AGCo) and input (AGCi) controlled compressors. These functions show the input on the *x*-axis and the output on the *y*-axis. These functions can be used to determine the characteristics of a specific compressor. For example, for output-controlled compression at volume 2, for a 50-dB input, the output is approximately 75 dB; for a 60-dB input, the output is approximately 85 dB; and so on. Notice that, when an output of 110 dB SPL is reached, there is no further increase in output when input increases. This point is the kneepoint of the output-controlled compression and is also the maximum output of the hearing aid (OSPL90). The kneepoint of a compression function is the point where the output curve deviates from linear. The ratio of the function is the degree of deviation. For example, if the output of the hearing aid increases by only 5 dB for every 10-dB increase in input, this would be described by a compression ratio of 2:1 (stated as 2 to 1). If the output changed by only 3.3 dB for every 10-dB increase in input, this would be a compression ratio of 3:1. In Figure 38.15 the AGCo has a kneepoint of 110 dB SPL and a ratio of 10:1. Recall that AGCo is most often used for output limiting. Thus, it typically has a very high kneepoint and a compression ratio of at least 10:1 and often higher. The AGCi, on the other hand, has a compression kneepoint of 55 dB SPL. For sound inputs up to 55 dB, the hearing aid provides linear gain. Observe that

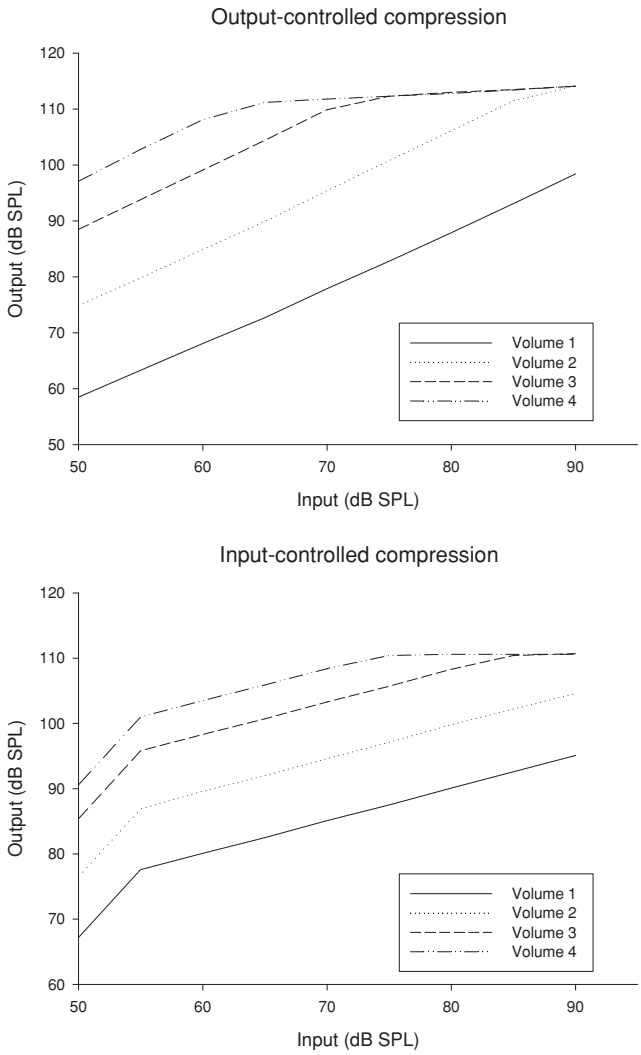


FIGURE 38.15 Input/output functions for input-referenced compression (AGCi) and output-referenced compression (AGCo).

when the input reaches 55 dB, the output of the hearing aid no longer increases by the same amount as the input, but rather by a smaller amount. Since AGCi is used for WDRC, it has a low kneepoint corresponding to soft levels of speech and low compression ratios, almost always less than 4:1 and quite often less than 2:1.

Compression Time Constants

Compression time constants have historically been part of the ANSI/IEC test battery but will not be mandatory tests in future revisions. The reason for the change is that checking time constants was a way to access the capacitor in a hearing aid and modern digital hearing aids do not have a capacitor so the test does not make sense any longer. Even though the test is not part of the quality standard, understanding time constants is important because they can make a difference in the performance of the hearing aids. Compression systems

in hearing aids have time constants that can be defined as follows.

Attack time. Using a puretone signal that changes abruptly from 55 to 90 dB SPL, attack time is the time required for the output to reach 3 dB of the steady-state value for the 90-dB input (American National Standards Institute (ANSI) S3.22-2003). Attack time is sometimes considered the time for the instrument to go "in" or "out of compression; however, for AGCi with low kneepoints, the hearing aid may not go "out" of compression for most listening situations.

Release (recovery) time. The interval between the abrupt drop from 90 to 55 dB SPL and the point where the signal has stabilized to within 4 dB of the steady-state value for the 55-dB input (ANSI S3.22-2003). In other words, release time is the time required for a circuit to respond to a decrease in the input and adjust to a lower compression characteristic (which, for some types of compression in some hearing aids, could be linear amplification).

The measures of attack time and release time are gradually being phased out. In the new IEC 118 standard (which will be consistent with future ANSI standards), these two measures will be relegated to the annex and be regarded as optional measures. Historically, attack time and release time was a quality control measure of the capacitor in the analog hearing aid. With the advent of digital technology the capacitor is no longer required. More on this issue can be found in Chapter 39.

Figure 38.16 illustrates the attack and release of a compression circuit. For most compression circuits the attack times are very fast (e.g., <10 ms). This is because the hearing aid needs to react quickly so that the input is placed appropriately within the patient's dynamic range. The release times can vary more. There are products with short release times (<10 ms) and those with long release times (>5 seconds). The release times are usually defined by the manufacturers according to their specific audiologic rationale for applying compression. The hearing aid may also use an algorithm that changes the time constants depending on the acoustic environment. This is because different compression parameters may be preferred for sound quality in some listening situations.

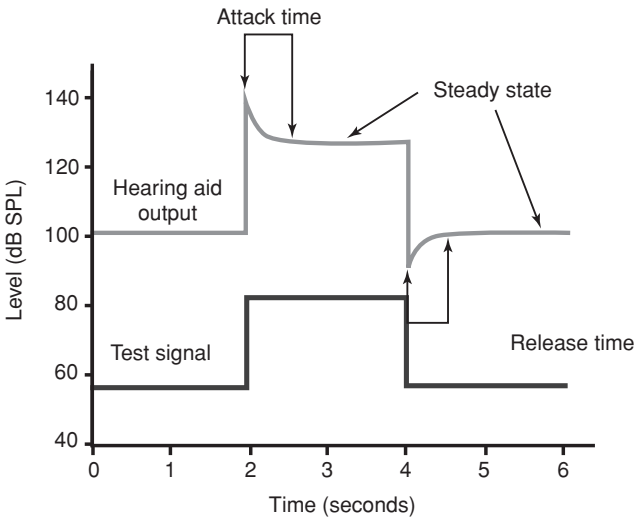


FIGURE 38.16 Schematic of the effect of attack and release of a compression circuit. [Courtesy of Harvey Dillon, personal correspondence.]

Multiple Channels

Most hearing aids today have multiple channels. This allows the input signal to be divided and processed into separate frequency bands and then recombined. In terms of multichannel compression, the compression characteristic can be changed independently for different frequency regions. These regions are separated by a crossover frequency, although there may be quite a degree of overlap between the regions. The kneepoint, compression ratio, and compression time constants can be adjusted independently with the channels. This type of compression allows the hearing aid to tailor the compressor to a hearing aid user's dynamic range that may be different in different frequency ranges. It should be noted that the terms hearing aid band and hearing aid channel are often used interchangeably. Some manufactures use the term channel to describe frequencies that are controlled by an individual compressor and the term band to describe frequencies whose amplitudes can be controlled by a single gain control.

Fitting Rationales

There are several fitting rationales for selecting the appropriate amount of gain for a hearing aid user's hearing loss. These are referred to as prescriptive gain formulas. The two most popular peer-reviewed rationales are NAL-NL2 and DSL i/o. These rationales prescribe the amount of gain required at each frequency for various input levels for a particular threshold of hearing. These prescriptions are used to set the compressor characteristics when programming the hearing aids. Manufacturers also develop rationales of their own for selecting the gain characteristics of the hearing aids, which typically do not have peer-reviewed support. It is important that once a rationale is selected, the gain is actually verified in the hearing aid user's ear at the hearing aid fitting using REM.

Expansion

Expansion is intended to improve the sound quality of hearing aids in quiet situations. It is not unusual for audiologists to be confused about the concepts of amplitude compression and expansion. Part of the reason for this is that from an audiologic viewpoint, they each seem to do something opposite of what the name implies. WDRC makes a broader range of sound intensities audible for the user. Although it is compressing the wide range of sound intensities into the limited

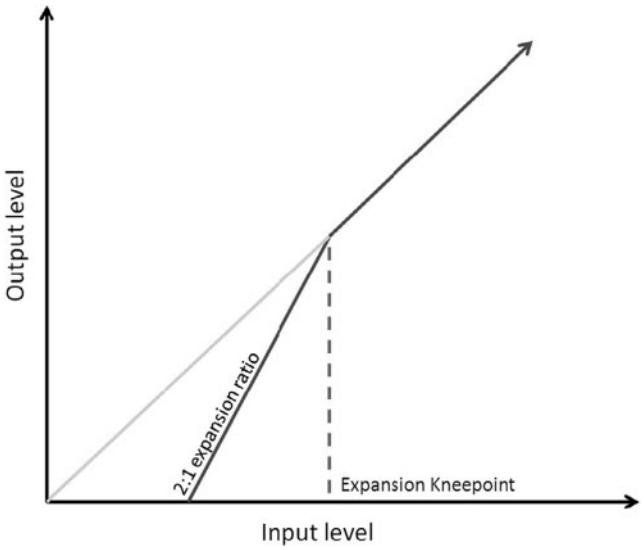


FIGURE 38.17 Input/output function for a compressor with expansion.

dynamic range of the hearing aid user, it can also be thought of as expanding the range of sounds that are made audible. Despite the confusing terminology, expansion reduces audibility for very soft sounds. Also commonly known as "squelch" or "microphone noise reduction," expansion keeps the hearing aid from overamplifying very soft sounds which are not of interest to the wearer, such as internally generated noise or very low level environmental sounds.

Expansion is part of the amplification system. As such, its characteristics are described in the same terms as compression, including kneepoints, expansion ratio, and time constants. Expansion reduces gain for sound levels below its kneepoint, whereas compression reduces gain for sound levels above its kneepoint. Expansion kneepoints are lower than the system's compression kneepoints, although this does not guarantee that soft speech will always remain audible. Figure 38.17 shows an input/output function for a hearing aid with expansion. Notice how these low inputs are in the range of linear amplification and thus receive maximum gain. Below the expansion kneepoint, gain is reduced for those inputs.

Frequency Lowering

Frequency lowering refers to signal processing algorithms that take high-frequency input signals and present these sounds to lower frequency regions. This technique has generally been recommended for hearing aid users who have "dead" regions in their cochleae. "Dead" regions refer to areas in the cochlea with loss of inner hair cell function, which, in turn, means that the corresponding auditory nerve fibers will not be stimulated (Moore, 2004). This tends to occur in frequency regions where the energy for many voiceless consonants critical for speech understanding lies (e.g., /s/, /f/, /t/). Without inner hear cells in these regions of the cochlea, any effort to provide gain cannot be successful. The best candidates for frequency lowering are those that have severe-to-profound hearing loss in which a hearing aid cannot adequately amplify the high-frequency signals or those with diagnosed "dead" regions. Dead regions can be assessed by diagnostic tests such as the TEN test (Moore et al., 2004). As of this writing today, there is controversy around using this type of signal processing for hearing aid users who do not have severe-to-profound hearing loss or "dead" regions. Some studies have indicated that it can be beneficial for speech understanding but large individual differences have been seen (Simpson et al., 2006). There are two large-scale studies using frequency compression for hearing-impaired children being undertaken today in Australia and in the United States. The results of these studies should shed more light on who is the right candidate for frequency lowering.

Two methods for accomplishing frequency lowering are available in commercial hearing aids today. They are frequency transposition (Kuk et al., 2009) and frequency compression (Glista et al., 2009). Frequency transposition uses a mixer to lower the signal by a fixed frequency value. Frequency transposition does not reduce the overall bandwidth of the hearing aid, rather it simply shifts frequencies to another region. The transposed high frequencies are laid over and coexist in the frequency region one octave below the selected start frequency. Frequency compression reduces both the frequency and the bandwidth by a preset ratio (factor) beginning at a selected cutoff frequency. So, for example, if the cutoff frequency was 2,900 Hz and the compression ratio was 4:1, the energy above this frequency would be divided by four and shifted to the area slightly higher than 2,900 Hz. The selected cutoff frequency and compression ratio both depend on the user's hearing loss and may be modified to reflect a person's listening experiences. Frequencies lower than 2,900 Hz (in this example) would be amplified as they would be normally.

Improving Signal-to-Noise Ratio

Individuals with hearing impairment not only have hearing loss, or loss of audibility, which is compensated for through amplification in hearing aids as discussed earlier in this chapter, but also have increased difficulty hearing in noise. This difficulty of hearing in noise can be quantified by measuring a listener's signal-to-noise ratio (SNR) loss (Killion, 1997). SNR loss is the increase in SNR (in dB) required by someone with a hearing loss to understand speech in noise, relative to the average SNR required for listeners with normal hearing. Two tests are commercially available to measure SNR loss, the Speech-in-Noise (SIN) test (Fikret-Pasa, 1993; Killion, 1997) and the Hearing-in-Noise Test (HINT) (Nilsson et al., 1994). There are large individual differences among hearing-impaired listeners on measures of SNR loss. A general trend is that SNR loss increases with hearing loss,

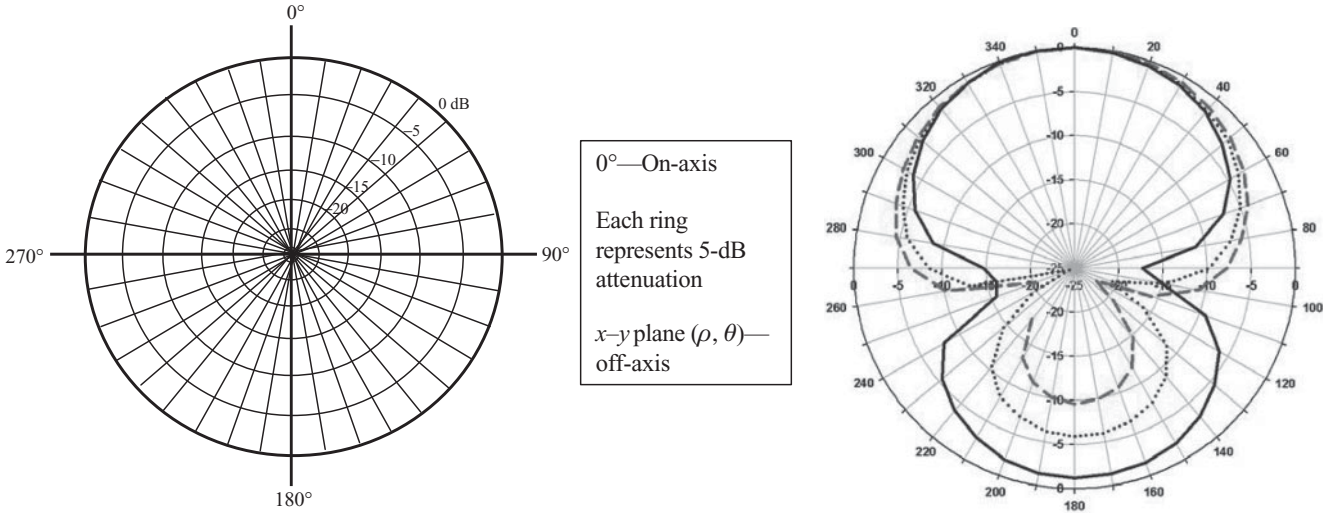


FIGURE 38.18 How to read a polar pattern. [Courtesy of Andrew Dittberner, personal correspondence.]

but the variance is quiet large and can range from no loss (normal-hearing performance in noise) to greater than 20 dB of SNR loss. To provide better hearing in noise, hearing aids incorporating directionality improve the SNR for users of hearing aids (Pumford et al., 2000). A rough estimate is that well-fit hearing aids incorporating directionality will improve the SNR for a listener by approximately 3 dB. This is enough for many hearing aid users to be able to follow a conversation in moderate levels of noise.

DIRECTIONALITY

A two-microphone method of obtaining a directional pattern is the most common technology available with modern hearing aids. Sound enters the microphones where the acoustic energy is converted to electrical energy. Following electrical conversion, the two signals are sent through an electrical network where a time delay is applied to the rear microphone signal. Finally, the two signals are subtracted to produce directivity. When both microphones are active a directional pattern is achieved. When an omnidirectional condition is desired the rear microphone is shut off usually automatically when a quiet environment is detected by the hearing aid's sound classifier.

Spatial Directionality Patterns

Fixed

Polar plots are a graphic representation of the sensitivity of the microphone for sound originating from various azimuths or angles. A polar plot is presented on a circle with the outermost circle typically representing 0-dB attenuation. Each line inside the outer circle typically represents 5 dB of attenuation. An azimuth of 0° represents sound arriving to the front of the listener whereas 180° azimuth represents sound arriving to the back of the listener. Figure 38.18 shows an empty pattern that the polar plot is graphed on and an actual polar plot. The point where there is maximum attenuation of sound is called the null. Figure 38.19 shows four polar plots. The first pattern is omnidirectional where the microphone is equally sensitive to sound from all angles. The other three designs are the cardioid, supercardioid, and hypercardioid directional patterns. The difference in the designs is mainly the location and depth of the null points. When the nulls are always at the same angle of the pattern and do not change depending on the location of noise or frequency they are called fixed directional patterns. From the polar plot a measure called the directivity index (DI) can be obtained for

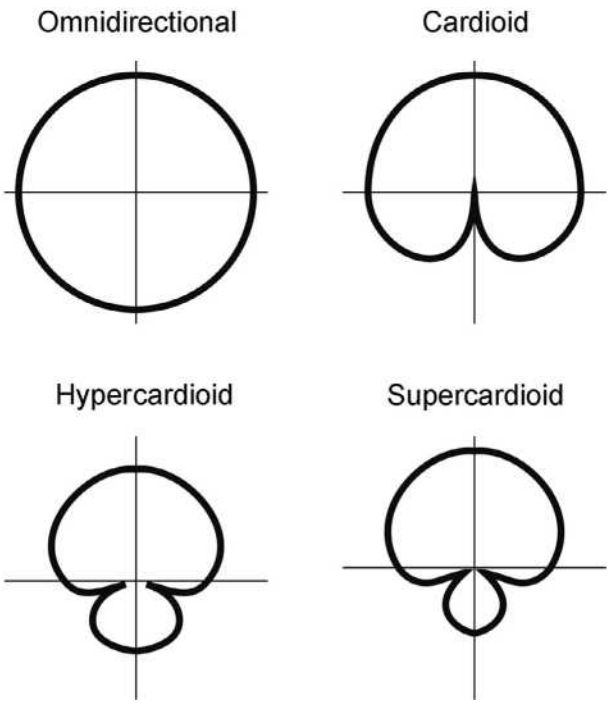


FIGURE 38.19 Four common polar patterns.

individual frequencies. The DI is a number that represents how sensitive a microphone is to sounds arriving from the front relative to sounds arriving from other directions. An omnidirectional microphone has a DI of 0 dB when measured in a test chamber. Directional microphones in hearing aids have DIs that commonly range from 2 to 5 dB measured in the test chamber. Measured in real-life situations, directional microphones in hearing aids improve the SNR of sounds arriving from the front by approximately 3 dB.

Adaptive

Adaptive directionality is a type in which the null can be moved to the angle where the most noise is detected in the environment, thus adapting the attenuation to the specific environment. This can even be done at different frequencies such that a hearing aid might have different directionality patterns depending on the frequency and location of the noise in the environment. Many hearing aids today come with adaptive directionality.

Microphone Mode Steering

Directional microphones in many hearing aids can be switched from omnidirectional to directional settings manually by the hearing aid user. Cord et al. (2004) indicated that 30% users did not switch between the settings and often did not know when to switch and/or did not want to do this manual switching in everyday life. To overcome this manual switching problem, automatic switching hearing aids were introduced where the hearing aid automatically changes from an omnidirectional setting to a directional setting depending on the environment. These types of switching algorithms depend on environmental classification systems which analyze the acoustic scene and make a decision about which microphone mode would be most beneficial. Thus, these systems are limited by the accuracy of the classification system and have no ability to determine the hearing aid user's intent in complex listening situations.

ASYMMETRICAL DIRECTIONALITY. The standard way to use directional processing in a bilateral hearing aid fitting is to program both hearing aids with a directional setting for use in a noisy environment. Another way to use directional processing is to keep one hearing aid set to omnidirectional and the other hearing aid set to directional. This seemingly unconventional way to apply directional processing can provide a better listening experience for users of hearing aids and overcomes the limitations of directional systems discussed above. Specifically, an asymmetric fitting can overcome the lack of use of manual systems and the reliance on environmental classification systems. An additional benefit is that it does not cut off a listener from his/her environment as wearing two hearing aids in directional settings can do. The user can choose to attend to whatever signal he/she may be interested in hearing. The key to asymmetrical directionality is to understand that one hearing aid in the directional setting and one in the omnidirectional setting provide the same SNR benefit for speech presented from in front of the user as using two hearing aids set in the directional settings (Bentler et al., 2004; Cord et al., 2007). Using hearing aids with the microphone modes set asymmetrically comes with the added benefit of better ease of listening (Cord et al., 2007) and better acceptance of background noise (Kim and Bryan, 2011).

Improving Comfort

A facet of successful hearing instrument use is the user's acceptance that not all amplified sounds are desirable, interesting, or pleasant. Hearing instrument users must resign themselves to hearing sounds that are not of interest to hear speech which is of interest. It is probably safe to say that no one seeks hearing help because they cannot hear their refrigerator humming or paper crinkling, yet they must accept hearing sounds like these to become successful users of amplification. A number of hearing aid technologies are aimed at reducing the drawbacks of amplification, such as the annoyance and effort associated with listening at elevated levels of background noise.

Noise Reduction

Noise reduction refers to signal processing that reduces gain in frequency areas where the SNR is estimated to be poor. It is often referred to as "digital noise reduction" which distinguishes it from spatial noise reduction schemes, such as directionality, as well as early noise reduction schemes that simply reduced gain for low-frequency sounds. These early attempts at noise reduction were based on the rationale that background noise typically has the most energy in the low frequencies.

Theoretically, noise reduction has two goals. One is to improve the SNR in noisy situations. Although it is possible to set up laboratory test conditions where a small improvement in SNR can be demonstrated, this advantage is not repeatable in more realistic test conditions and is not accepted as a benefit of noise reduction. It is not difficult to understand why noise reduction fails on this objective. To be successful, the algorithm must separate speech from noise when both signals usually have energy at the same frequencies at the same time.

Complicating matters further is that many noises have speech-like characteristics and many parts of speech have noise-like characteristics. The other aim of noise reduction is to make the experience of wearing hearing aids more acceptable and pleasant, which currently appears to be an attainable goal. Although the overall objectives of noise reduction are similar, the actual signal processing algorithms and their effects on different types of sounds can be very different both in the physical (Bentler and Chiou, 2006) and perceptual domains (Brons et al., 2013). Several examples of differences in the way sounds are affected

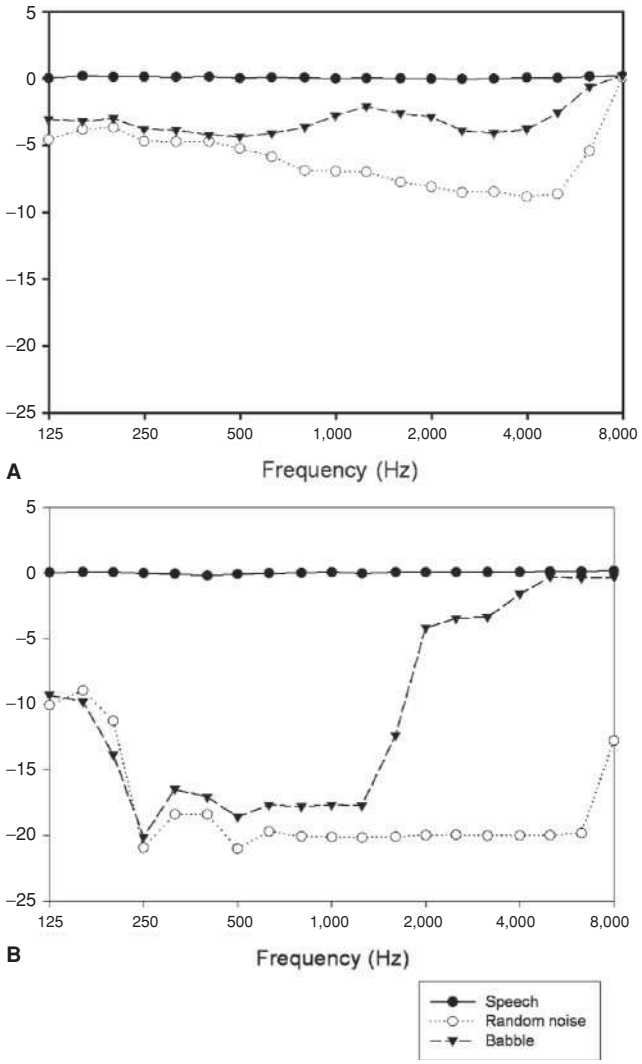


FIGURE 38.20 Example of different outcomes for two different digital noise reduction schemes.

by different noise reduction algorithms are shown in Figures 38.20A and 38.20B. From a technical perspective, noise reduction algorithms differ in terms of how many frequency channels they operate in, how quickly they engage and disengage, how they identify speech and noise in the incoming sound, and what rules they follow to reduce gain.

Identifying speech and noise, which are mixed together in the input sound, is key to how noise reduction processing works. The first digital noise reduction algorithms used *modulation* as an approximation of the SNR. With a modulation-based approach, the signal observed at the microphone is analyzed to determine whether modulations in the amplitude fluctuation (or waveform) are similar to those observed in speech. The modulated waveform, or temporal envelope of speech, contains information that is essential for the identification of the different parts of speech, such as phonemes, syllables, and words.

For modulation-based noise reduction, high modulation depth in a frequency channel is considered to be indicative of high SNR, and there will be minimal or no gain reduction applied. As the modulation depth decreases, this is considered to indicate the presence of noise and/or absence of speech, and the gain will be reduced according to rules that are specific to the particular manufacturer's algorithm. Modulation-based noise reduction works well when the wanted signal is a single talker in the presence of steady noises, like a fan or motor. However, this type of system does not work as well when the competing noise consists of other people talking, which is a common scenario. As a result, the use of modulation-based algorithms to detect and eliminate noise has obvious limitations. Even if the noise *could be* identified and removed from the environment for specific spectral regions, the speech information in that same region would be removed as well.

Increasingly, manufacturers are using a multifaceted way of identifying speech and noise portions of the sound and applying a "spectral subtraction" approach to noise reduction. This approach attempts to estimate the spectrum of the noise background and subtract it from the total signal. The noise estimate is updated in pauses between speech segments, which means that more advanced methods for identifying speech are needed than reliance on modulation of the signal. A spectral subtraction approach to digital noise reduction will typically work faster than a modulation-based approach (Kates, 2008) and may improve the ability of the algorithm to work in different types of noise backgrounds, including environments with multiple talkers.

There is no consensus on which noise reduction methods and parameter settings are the most advantageous or whether different methods may differ in terms of how suitable they are for individuals. Nevertheless, noise reduction algorithms have become widely accepted because of achieving the goals of improved listening comfort and ease of listening, and evidence is gathering in support of these benefits (Palmer et al., 2006; Bentler et al., 2008; Sarampolis et al., 2009; Wu and Stangl, 2013).

Wind Noise Reduction

Wind noise is a phenomenon that most people recognize from outdoor cell phone conversations or news correspondents trying to report from stormy areas. The wind blowing on the microphone in each of these cases causes a very loud rushing sound that can drown out the speaker's voice. It might come as a surprise to know that wind noise is also a significant problem for hearing aids. Turbulence is created when wind flows across or around any object. A microphone placed in the midst of turbulent air flow will pick up the random pressure variations and convert them to voltage changes in the same way the microphone picks up and converts the pressure variations of airborne sound waves. The wind noise picked up by the microphones is amplified and heard by

the user as a very loud and annoying noise. Depending on where the hearing aid microphones are located and how the user's head is oriented relative to the wind, this issue can be a source of great dissatisfaction. The pinna itself serves as an obstruction when wind comes from the front, creating turbulence behind the ear. Thus BTE and RIE styles, where microphone placement is behind the ear, are particularly susceptible to wind noise. Directional microphone systems are also more sensitive to wind noise than omnidirectional microphones (Chung et al., 2010).

Wind noise can be so intense that it overloads the microphone preamplifier, resulting in a signal that is distorted before it even reaches the DSP stages of the hearing aid. The ideal solution for wind noise is to keep it from entering the hearing aid microphone(s) in the first place. Some hearing aid designs incorporate wind screens for this purpose. Generally speaking, ITE-style hearing aids may also provide better protection of the microphone from wind noise. This is particularly true if the microphone is recessed, such as with a deep fitting CIC, an IIC, or the MIC styles.

Some hearing aids also have signal processing algorithms that attempt to reduce wind noise. Because wind noise contains mostly low-frequency energy, wind noise reduction algorithms target the low frequencies for gain reduction. However, as is the case with digital noise reduction, wind noise reduction algorithms differ in terms of their approach to identification of wind noise, time constants, and rules for reducing the wind noise component. These differences translate to different effects on the acoustic signal, which make each wind noise reduction system sound different.

Reducing Feedback

The amount of gain that is actually available to a hearing aid user is often limited by acoustic feedback, which is the squealing that comes about when sound from the hearing aid receiver returns to the microphone and is reamplified. There are a number of pathways by which this can occur, the most obvious of which are vents and acoustic leakage around the earmold or shell. Some additional routes by which sound can travel from the receiver back to the microphone include earmold tubing and coupling between tubing and earmold, earmold and earhook, or earhook and receiver; emission from the hearing aid shell; and structural and acoustic transmission within the hearing aid (Hellgren et al., 1999a). Although bone-anchored hearing aids (BAHAs) are not part of this chapter, it has also been demonstrated that feedback can occur as a result of skull vibration, especially if the coupling is poor. The sum total of the various transmission lines constitutes the feedback path (Figure 38.21). When the gain in the hearing aid exceeds the damping provided in the feedback path, acoustic feedback results.

The management of that annoying outcome has been to (1) remake the earmold/shell to be fuller or deeper so that there is less leakage between the mold and the wall of the ear canal, (2) reduce the size of the venting, (3) roll off the high frequencies, and more recently, (4) reduce the gain in narrow frequency bands or through notch filtering centered in those bands. Each of these solutions may be counterproductive to the intent of the fitting. Solution 1 may result in an uncomfortable fit; solution 2 may impact on the acoustic characteristics desired; and solution 3 may decrease speech intelligibility. One possible negative outcome of solution 4 is the introduction of distortion for the listener. Even when the hearing aid gain is set to a level below the oscillation point, the signal feeding back may still cause alterations, or fluctuations, in the signal that are perceptible to listeners, especially when these alterations occur at formant transitions (Cox, 1982). It has been suggested that this gain be reduced 4 to 8 dB below the feedback onset to avoid these deleterious effects (Skinner, 1988). To do so, however, undermines the goal of providing optimal gain for audibility.

Digital hearing aids have enabled more advanced approaches to feedback management. The method most similar to the previously described approaches is adaptive notch filters that can change the frequency area at which they are applied depending on where feedback occurs. Other processing schemes attempt to increase the amount of gain available for the user without changing the frequency response of the hearing aid. For example, shifting the frequency of the hearing aid output by a small amount is one method that can add a small margin of usable gain, as it reduces the correlation between the external sound and the amplified sound which re-enters the microphone. Too much of a shift in frequency, however, causes an altered sound quality that is likely to be unacceptable to hearing aid users.

Another technique for feedback reduction which aims to increase the available gain for the individual fitting is phase cancellation. This type of algorithm has become

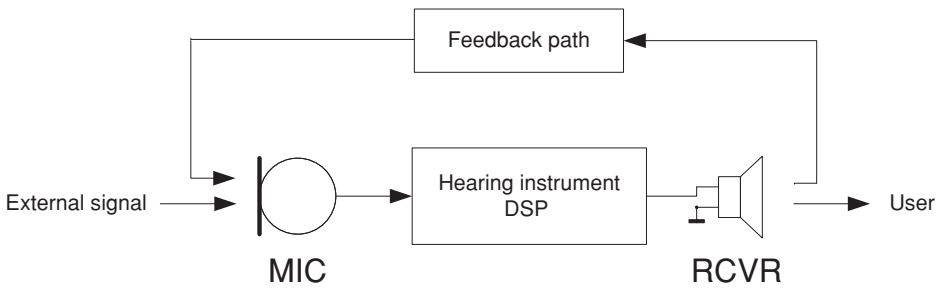


FIGURE 38.21 The feedback path describes in terms of frequency, amplitude, and phase how the amplified sound is changed as it returns to the hearing aid microphone(s).

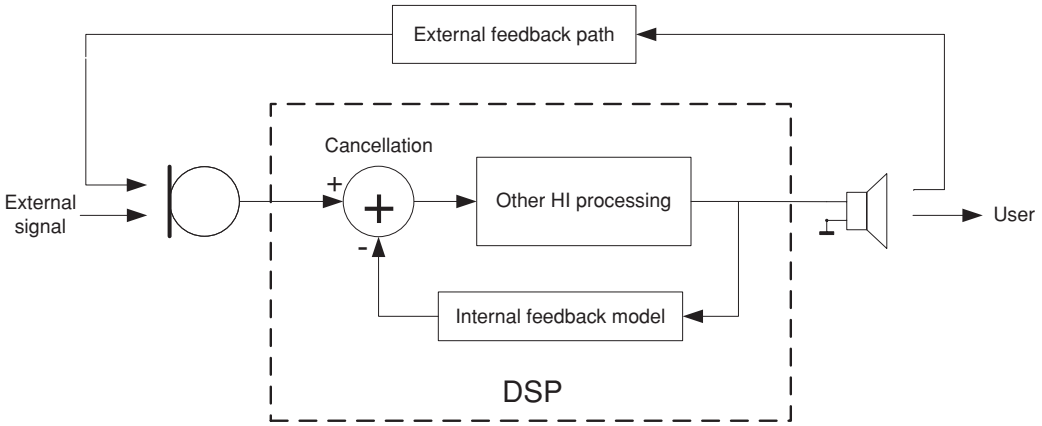


FIGURE 38.22 Feedback cancellation systems attempt to model the feedback path, create a signal that is equal but opposite in phase, and add it to the input to the hearing aid.

ubiquitous in modern digital hearing aids and is conjectured to be a contributor to improved satisfaction with hearing aids in terms of "whistling and feedback" (Kochkin, 2010). The principle behind feedback phase cancellation is for the system to analyze the feedback path, create a signal with the same frequency and amplitude characteristics but opposite in phase, and add it to the input of the hearing aid. If the added signal matches the feedback path perfectly, the feedback is completely cancelled from the input to the hearing aid (Figure 38.22). Although this sounds simple, there are significant limitations to feedback cancellation algorithms. For one thing, they must always provide an accurate model of the feedback path. The feedback path can change by 20 dB or more quite rapidly (Hellgren et al., 1999b), and the system must track these changes. The feedback path may also be complex, with multiple peaks at different frequencies.

Reverberation effects in the acoustic environment also affect the feedback path (Kates, 2008). All of these require complex computations, which take additional time and power, neither of which are in abundance in current hearing aids. As a result, the feedback cancellation algorithms are limited in how well they can react to many fast changes in the feedback path, and the potential for additional gain relative to no feedback cancellation is 10 to 15 dB (Kates, 2008). In practice, there can be large differences in the feedback cancellation algorithms from different manufacturers. Studies have demonstrated additional stable gain as a result of feedback cancellation ranging from near zero to more than 18 dB (Freed and Soli, 2006; Merks et al., 2006).

A negative consequence of these algorithms is the occurrence of *entrainment* (Merks et al., 2006). Entrainment refers to the unintentional result of the filter attempting to cancel what appears to be feedback but, in reality, is some other tonal input to the microphone (e.g., from a musical production). The listener often reports hearing either an additional tone or a modulation-type distortion during entrainment. This is an obvious detriment to sound quality. Manufacturers employ various methods for avoiding entrainment, examples of which are slower time constants for applying feedback cancellation or adding an "acoustic signature" to the output of the hearing aid to help the algorithm distinguish between feedback and nonfeedback signals in the input. The adaptive behavior of the cancellation algorithm may also be limited in terms of frequency area and extent of adaptation. No perfect solution has yet been introduced, which means that feedback cancellation algorithms are necessarily a trade-off between additional stable gain and preserved sound quality.

Personalization

Hearing aid fittings begin with some sort of prescription of settings based on user data, usually an audiogram. The main goal of fitting prescriptions is to provide amplification for optimum speech understanding while ensuring comfort for loud sounds. The presumption of this approach is that one set of hearing aid parameters will meet the listening needs of an individual in all conditions. In reality, a hearing aid user may want to enhance or diminish different aspects of the amplified sound in different situations. There is plenty of evidence that this is the case: Hearing aid users have different preferences for gain–frequency responses depending on their listening environment (Keidser et al., 1995), different volume preferences (Surr et al., 2001), as well as different preferences for directional or omnidirectional microphones in different situations (Walden et al., 2004). Studies have also shown preferences for different compression characteristics depending on acoustic environment or nonaudiologic factors such as cognitive function (see Kates, 2010 for a review).

Personalization features aim to account for individual needs and preferences in the way hearing aids work. Although many of the hearing aid features discussed in this chapter are candidates for personalization, the ones covered in this section are specifically for that purpose and may in fact interact with or control other features.

Environmental Classification

A cornerstone of personalization is to capture and use information that accounts for the unique situation of each individual hearing aid user. One hearing aid feature that contributes to personalization is environmental classification. This type of algorithm attempts to categorize and track the type of listening environments that the hearing aid user is in while wearing the devices. It provides input to data logging in the hearing aid and may also be used by decision-making algorithms to turn certain features on and off, change programs in the hearing aid, or change feature settings in other algorithms. For example, when a noisy environment is detected by the environmental classifier, the degree of noise reduction applied might be increased relative to the setting used for a quiet environment. The purpose is to adjust the hearing aid to better suit the preferences of the individual for how it should sound in a particular environment.

An obvious issue with environmental classification is that the number and characteristics of acoustic environments in the real world are infinite. Hearing aid manufacturers must break these down into a small number of manageable, idealized acoustic environments. The categories into which an environmental classifier sorts the input are defined by each manufacturer, so they are not standardized. However, all systems will at least try to identify environments that are quiet, ones that contain speech, and ones that contain noise. Some may also attempt to further characterize types of noise or to identify music.

Data Logging

Many of today's hearing aids have a feature referred to as data logging. This is more or less an electronic diary that can be used to collect data (1) on hearing aid use, (2) on program/memory and volume control use and (3) to summarize the results of the environmental classification system (e.g., percentage of time the listener was in quiet, noise, and speech in noise). Data logging is not a "processing" feature per se in that it does not treat the incoming sound. However, it can be quite valuable in patient counseling. For one thing, it provides evidence of whether or not the patient has even used the hearing aid. For patients who are using their hearing aids, comparison of what environments the hearing aids have been used in and which programs that were used can serve as a cue for instruction on appropriate program use. Data logging also plays a prominent role in trainable hearing aids that employ learning algorithms; by logging the preferred settings in different environments, the data are subsequently used for automatic switching of parameters and features.

Learning Algorithms

Anyone who has used the Internet in recent years is familiar with learning algorithms. They use information gleaned from your actions and attempt to use the information to customize your experience. For example, if you have searched for tents online, you will likely notice that advertisements for tents and other camping equipment will appear in your browser the next time you open it. Some hearing aid algorithms have been introduced that attempt to do the same sort of thing. That is, they keep track of user actions, make the assumption that these actions represent the user's preferences, and attempt to automatically apply settings that are consistent with these preferences. For example, if the hearing aid user consistently increases the volume of the hearing aid by 3 dB in a listening situation that the environmental classifier categorizes as a speech-only environment, the hearing aid will gradually begin to automatically increase gain when this environment is encountered.

Hearing aids that incorporate learning algorithms are sometimes referred to as "trainable" hearing aids. The learning algorithms currently found in hearing aids are fairly simple and crude, due in part to the limitations of environmental classification and in part to the fact that volume control is the only parameter that lends itself well to include in a commercial product for broad use. Research with trainable hearing aid concepts have demonstrated that users are capable of interacting with learning algorithms to customize other hearing aid characteristics to their preferences, such as frequency response and noise reduction (Dreschler et al., 2008). As new ways of interacting with hearing aids continue to emerge, such as via hearing aid–related apps loaded onto smartphones, it is likely that learning algorithms will play a bigger role.

Tinnitus Management

Tinnitus is a concern for many people and affects approximately 10% of the overall population, with approximately 3% to 5% of the population suffering from clinically treatable tinnitus. Most tinnitus sufferers also have some form of hearing loss. Regardless of what type of tinnitus management protocol is used, sound therapy almost always plays a vital role in the outcome. Therefore it makes sense to combine amplification for hearing loss compensation with sound therapy options for tinnitus management. Sound therapy is simply the introduction of an external sound to help reduce the contrast of the tinnitus against the background acoustic environment. Hearing aids with tinnitus features can generate sounds that are useful in tinnitus management. The simplest of these is actually to use the telecoil program in the hearing aid when there is no signal present, as this creates a low buzzing sound that may be effective for the individual. Other hearing aids are available that generate sound specifically for tinnitus management. These include different noise sounds which at a minimum can be adjusted in terms of spectrum and level. There may also be options for modulating the noise to make it sound like the ocean, rain, or other nature sound or to make the noise

level dependent on the acoustic environment. Tinnitus sufferers who wear digital wireless hearing aids can also make use of audio streaming capabilities to use other sounds from external audio sources that are effective for them. As the memory capabilities of digital hearing aids increases, other types of sounds may become available in the hearing aid itself for tinnitus management.

DIGITAL WIRELESS TECHNOLOGY

In the first quarter of 2013, the HIA started tracking the use of wireless versus non-wireless hearing instruments. These statistics show that wireless hearing aids now constitute 70% of the entire United States' hearing aid market. The phrase "wireless hearing" aids refers to digital wireless technology where wireless transmission is used to either exchange information between two hearing aids or receive information from other sound sources like a TV, MP3 player, microphone, or phone. It should be mentioned that hearing aids have utilized analog wireless technology in the form of telecoils and FMs for many years.

The first digital wireless hearing aids used ear-to-ear data transfer for convenience. When the hearing aid user adjusted the volume control on one hearing aid it would automatically adjust the other hearing aid in the same manner. This would also happen when the user changed the hearing aid memory. Digital wireless hearing aids have advanced to include data transfer for unique signal processing algorithms and connectivity to TVs, phones, and computers. Today's digital wireless hearing aids incorporate one of two types of transmission, near-field magnetic induction (NFMI) or radio frequencies (RFs). Both NFMI and RF systems offer advantages and disadvantages.

NFMI operates in a similar fashion to a telecoil using magnetic induction to transmit. The frequencies used for transmission are between 3 and 15 MHz. NFMI systems have a short range, thus making them near field, because the signal is not radiated beyond 3 to 5 feet. In NFMI, the transmission energy is kept within a localized magnetic field around the transmitting antenna (inductive neck loop). To receive signals from sound sources at greater distances, a relay device using a far-field technology must be used. Most current NFMI systems rely on a propagating RF signal using the open standard Bluetooth protocol for their far-field transmission. In operation, an NFMI system uses a device plugged into the sound source that sends the Bluetooth signal to the relay device. The relay device receives the Bluetooth signals and relays them to the hearing instruments via induction. Figure 38.23 illustrates the architecture of an NFMI-based system. The advantage of NFMI systems to the hearing aid is primarily related to battery life. NFMI has better battery life than RF systems. One other potential advantage for some patients is that the relay device can be used for other functions such as a remote control. The disadvantages of NFMI include having to wear the relay device

FIGURE 38.23 An NFMI digital wireless hearing instrument system. (Courtesy of Oticon.)

because of the short transmission range and a sound delay that is introduced because of using Bluetooth (up to 40 ms) for the far-field portion of the signal transmission. This delay can cause an echo to be heard especially when users are fit with open hearing aid designs or if the hearing aid microphones are simultaneously active and picking up the sound acoustically.

Wireless technology utilizing RF technology uses a frequency to transmit signals a minimum of 30 feet to up to several hundred depending on the antennas size and the power source. RF systems use an antenna to generate and transmit a propagated electromagnetic wave. This type of transmission is referred to as *far field.* The frequencies used in systems today include 2.4-GHz and 900-MHz ISM (Industry Science Medical) bands. The 2.4-GHz ISM band can be used internationally, whereas the use of the 900-MHz ISM band is limited to Region 2 (the Americas, Greenland, and certain eastern Pacific islands). Apart from these regulatory considerations, the choice of frequency for RF systems also impacts the size of the antennas needed for transmission, and thus the size of the devices. There is a fixed relationship between the frequency and the physical dimensions of the antenna, with a lower frequency requiring a longer antenna. Antennas in the hearing instruments must be very small. For a 900-MHz system, this means that the antennas in the transmitting devices must be much larger to compensate. Thus, the wireless accessories of this lower frequency system need to be made larger than are necessary for the higher frequency 2.4-GHz–based system. In operation, an RF system uses an adaptor to connect to sound sources and send to receiving antennas embedded in the hearing instruments.

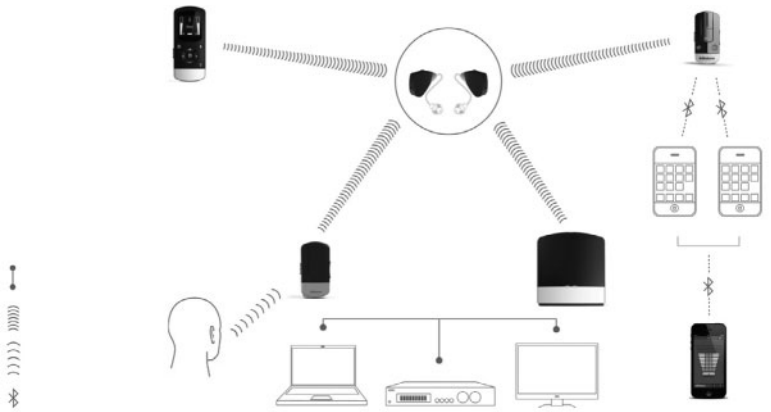


FIGURE 38.24 An RF digital wireless hearing instrument system. [Courtesy of ReSound.]

No relay device has to be worn around the neck. The architecture of an RF system is illustrated in Figure 38.24. The advantages of RF systems are the long range that the signal can be transmitted, not having to wear a relay device, and the ability to audio stream in stereo. The biggest disadvantage is battery drain because RF requires more power for a given application.

Binaural Processing

Binaural processing in hearing aids refers here to using the exchange of information between the devices to do some unique signal processing with potential benefits for the hearing aid user. In digital wireless hearing aids there are several different ways that ear-to-ear communication is being used. These include synchronizing some signal processing between the ears such as compression, noise reduction, and directionality so that both hearing aids always have the same setting; enhancing the directional patterns by using the input to all four microphones to narrow the directionality; setting different directional microphone configurations for different environments (e.g., using asymmetric directionality in some environment, directionality in others, and omnidirectionality in the rest); determining if feedback is being experienced or if it is another signal because it is arriving at both ears; streaming of sound from one hearing aid to the other; and finally enhancing cues for localization.

Connectivity and Accessories

As discussed above, digital wireless hearing aids create an entire hearing system when combined with accessories to connect the hearing aid user to other sound sources. Today the hearing aid user can be directly connected to his/her TV, phone, computer, MP3 player, and a variety of microphones. This connectivity significantly improves the SNR for the hearing aid user because the sound source is streamed directly to the ear. Connectivity has the ability to help hearing aid users hear in more environments than hearing aids alone. The future will continue to expand in this area of connectivity. Using 2.4 GHz, digital wireless hearing aids have now been introduced to the markets that directly connect to a smart phone without use of any relay device.

OTHER STYLES OF HEARING AIDS

Earlier in this chapter an overview of the common styles of hearing aids was given. There are other less common styles that will be overviewed here.

CROS/BICROS

Contralateral routing of signal (CROS) and bilateral contralateral routing of signal (BICROS) hearing aids are a special type of hearing aid used when an individual does not have any hearing in one ear that can benefit from a hearing aid. These hearing aids are designed so a microphone is worn on the ear that cannot benefit from a hearing aid and the sound picked up at this microphone is sent wirelessly or via a wire in some cases to the other ear. A CROS hearing aid is worn when one ear cannot benefit from a hearing aid and the other ear has normal hearing. In this case, the sound picked up at the "dead" ear is sent to the aid on the opposite ear with this sound sent to the eardrum. In the case of the BICROS hearing aid, the user has one ear that cannot benefit from a hearing aid and other ear that has some degree of aidable hearing loss. The sound picked up at the "dead" ear is sent to a hearing aid on the other ear where the signal is amplified and delivered to the ear.

Bone-Anchored Hearing Aids

BAHAs are surgically implanted aids that directly stimulate the cochlea through bone conduction. These aids consist of a titanium implant, an external abutment, and a sound processor. These aids are meant to bypass the external and middle ears. The titanium implant is surgically placed into the skull behind the pinna percutaneously (directly coupled to the bone). The sound processor sits behind the ear. This type of aid works by picking up sound at the microphone

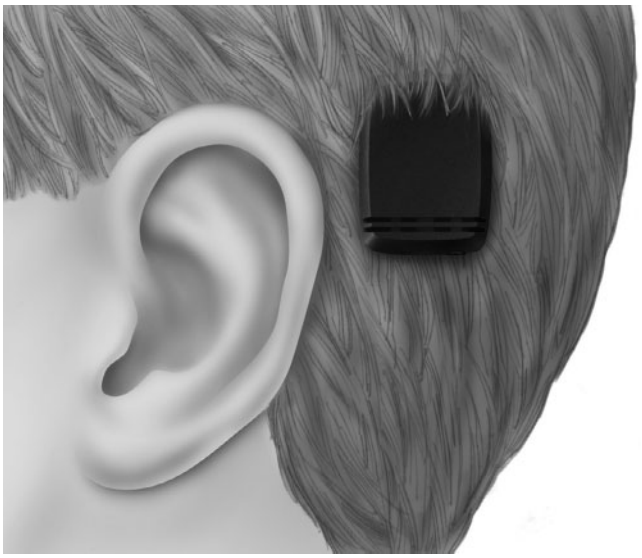


FIGURE 38.25 A bone-anchored hearing aid (BAHA). (Courtesy of Cochlear Americas © 2013.)

of the sound processor which is then transmitted to the implant. The implant vibrates within the skull and stimulates the nerve fibers of the inner ear by bone conduction. Recently, a new BAHA-type device was announced that is transcutaneous; part of the device is implanted but the other part is kept outside the skin similar to a cochlear implant. BAHA-type devices are for unilateral deafness, chronic external and/or middle ear conditions, and congenital ear malformations. Figure 38.25 shows a BAHA device.

Bone Conduction

Bone conduction hearing aids are a special kind of hearing aid used when the outer ear cannot wear hearing aids. Reasons for this might include an atresic ear, a draining ear, or any a number of problems with the ear where a hearing aid cannot be worn. In this type of device sound is sent directly to the cochlea via bone vibration, thereby bypassing the part of the ear that is diseased. Bone conduction hearing aids traditionally have used a similar type of vibrator known from bone conduction audiometry and attached to the skull with a metal or elastic band. Some manufacturers are also making use of the bone conduction principle in innovative ways, such as encasing the vibrator in a dental appliance and wearing it in the mouth or encasing it in an earmold and wearing it in an ear canal. In these cases, the microphone and sound processor might be worn on a deaf ear, thus serving the same function as a CROS hearing aid.

Extended Wear

An extended wear hearing aid is one that is placed deep in the ear canal near the tympanic member (see Figure 38.26) by an audiologist or otolaryngologist and can be worn for several months. These hearing aids were introduced to the market in 2008. The primary advantages of this type of hearing aid are cosmetic and convenience. The technology inside the device is analog rather than digital, but it is digitally programmed for a patient's hearing loss. The only nonsurgical extended wear hearing aid on the market today, the Lyric, is disposable. Once the battery wears out the aid is thrown away and a new hearing aid must be inserted in the ear canal. Candidates for extended wear hearing aids must have ear canals that can accommodate the device which limits the candidacy as some individual's ear canal size is not suited for this device. Extended wear hearing aids can cost significantly more than other types.

Body Worn

Body-worn hearing aids are the largest hearing aids. These aids are composed of a hearing aid worn on the body (usually around the neck) which is connected with cables and earmolds to the ears. Body aids can provide a wide range of gain and output. Body-worn hearing aids were the only hearing aid style available until the 1950s when BTE hearing aids were introduced. These devices are not seen in developed countries today (<1% of the market in the United States and Europe) but are still common in the developing world.

Eyeglass

Eyeglass hearing aids are a combination of eyeglasses and hearing aids. The hearing aids can be located in the frame of the glasses or can be coupled to the frame using an adapter. Tubing then extends from the frames to couple the aids to the earmolds and ear. These types of hearing aids are not very commonly seen today.

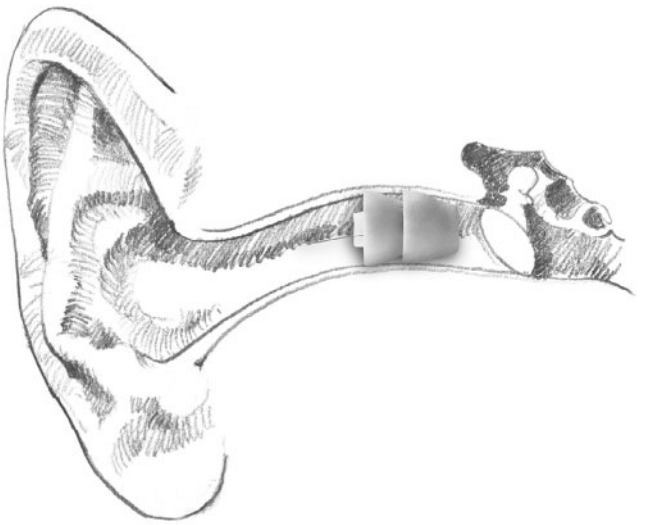


FIGURE 38.26 An extended wear hearing aid. (Courtesy of Phonak.)

SUMMARY

Modern hearing aids can improve the quality of life for patients with hearing loss. Hearing aids using digital wireless technology create hearing systems where hearing aid users can hear in a significant number of listening environments. These systems will continue to evolve in the years ahead providing new benefits to hearing aid users. This chapter has served as a review of the current state of hearing aid technology including many of the features commonly found in hearing aids. These features may have similar goals, but manufacturers use different implementation strategies that can be more or less effective. Audiologists must be critical on behalf of their patients to determine what features provide the best benefit to their patients. It is also important that the audiologist stay up-to-date on technology innovation with regard to hearing aids as technology changes quickly.

FOOD FOR THOUGHT

The Merging of Hearing Aids and Consumer Electronics

Predicting future hearing aid technology is difficult, but there are some emerging trends in the industry. Certainly, wireless hearing aids with connectivity to peripheral devices will continue to be developed and improved. This should lead to better hearing in more environments for hearing aid users. The upcoming releases of hearing aids with direct connections to smart phones will expand options to not only control hearing aids via cell phones, but also adjust the fitting of the hearing aid. Already there are cell phone applications related to hearing aids, but the future will see an increase in these. Applications could lead to greater personalization and ultimately better user satisfaction, but these also open up the controversial issue of self-fitting devices. In particular the role of the professional in these fittings will be debated.

For discussion:

1. What are the pros and cons of the convergence of hearing aids and consumer electronics?
2. How much control over the hearing aid fitting should the hearing aid user be given?
3. How can audiology professionals secure their role as the professional that fits hearing aids to the hearing aid user?

Evidence-Based Practice

Hearing aid manufacturers introduce new hearing aids to the market at an increasingly fast pace. With each introduction, new features are marketed to audiologists that promise greater end-user benefits than current hearing aids. Audiologists should use evidence-based practice when determining the appropriate hearing aid and features; however, peer-reviewed research in the area of hearing aids is often several years behind the introduction of new features in hearing aids.

For discussion:

1. How can audiologists determine what new features really make a difference to their patients?
2. How much knowledge should the audiologist have about the increasing complex algorithms found in hearing aids?

Market Penetration

It is estimated that of the approximately 34 million hearing-impaired people in the United States, only 24% of them wear hearing aids. There are many reasons given for these low penetration rates, including that many have mild hearing loss and do not have enough problems in hearing to begin to seek out help for the problem. Excluding those with milder hearing loss, there are still many people with hearing loss that need hearing aids that do not wear them.

For discussion:

1. How in the future can we help more people with hearing loss?
2. What do you think contributes to the reluctance of people with hearing loss to use hearing aids?
3. Can hearing aids that can be bought over the counter without seeing an audiologist help with this penetration problem?

How Are Hearing Aids Sold?

Hearing aids have traditionally been sold in a variety of settings from retail shops and audiologist-owned private practices to hospitals and otolaryngology practices. There is a dramatic shift, in the United States today, in how hearing aids are sold. Hearing aid manufacturers are increasingly buying shops and setting up their own retail locations for their products. In addition, big box stores like Costco and Wal-Mart are selling hearing aids.

For discussion:

1. How will the current trend in hearing aid distribution change audiology?
2. How will this trend benefit the hearing aid user?
3. How will this trend hurt the hearing aid user?

REFERENCES

American National Standards Institute. (2003) *American National Standard Method Specification of Hearing Aid Characteristics.* ANSI S3.22-1996. New York: American National Standards Institute.

Bentler RA, Chiou K. (2006) Digital noise reduction: an overview. *Trends Amplif.* 10 (2), 67–82.

Bentler RA, Egge JL, Tubs JL, Dittberner AB, Flamme GA. (2004) Quantification of directional benefit across different polar response patterns. *J Am Acad Audiol.* 15 (9), 649–659.

Bentler RA, Wu Y, Kettel J, Hurtig R. (2008) Digital noise reduction: outcomes from laboratory and field studies. *Int J Audiol.* 47, 447–460.

Brons I, Houben R, Dreschler WA. (2013) Perceptual effects of noise reduction with respect to personal preference, speech intelligibility, and listening effort. *Ear Hear.* 34 (1), 29–41.

Chung K, McKibben N, Mongeau L. (2010) Wind noise in hearing aids with directional and omnidirectional microphones: polar characteristics of custom-made hearing aids. *J Acoust Soc Am.* 127 (4), 2529–2542.

Cord MT, Surr RK, Walden BE, Dyrlund O. (2004) Relationship between laboratory measures of directional advantage and everyday success with directional microphone hearing aids. *J Am Acad Audiol.* 15 (5), 353–364.

Cord MT, Walden BE, Surr RK, Dittberner AB. (2007) Field evaluation of an asymmetric directional microphone fitting. *J Am Acad Audiol.* 18 (3), 245–256.

Cox R. (1982) Combined effects of earmold vents and suboscillatory feedback on hearing aid frequency response. *Ear Hear.* 3 (1), 12–17.

Dreschler WA, Keidser G, Convery E, Dillon H. (2008) Client-based adjustments of hearing aid gain: the effect of different control configurations. *Ear Hear.* 29 (2), 214–227.

Fikret-Pasa S. (1993) *The effect of compression ratio on speech intelligibility and quality*. Northwestern University, Ph.D. dissertation, University Mirofilms, Ann Arbor, MI.

Food and Drug Administration. (2009) Guidance for industry and FDA staff: regulatory requirements for hearing aid devices and personal sound amplification devices. Document issued on February 25, 2009.

Freed DJ, Soli SD. (2006) An objective procedure for evaluation of anti-feedback algorithms in hearing aids. *Ear Hear.* 27 (4), 382–398.

Glista D, Scollie S, Bagatto M, Seewald R, Parsa V, Johnson A. (2009) Evaluation of nonlinear frequency compression: clinical outcomes. *Int J Audiol.* 48 (9), 632–644.

Hallenbeck SA, Groth J. (2008) Thin-tube and receiver-in-canal devices: there is positive feedback on both! *Hear J.* 61 (1), 28–34.

Hearing Industries Association. (2012) Hearing Aid Industries Statistical Reporting Program summary 2012.

Hellgren J, Lunner T, Arlinger S. (1999a) System identification of feedback in hearing aids. *J Acoust Soc Am.* 106, 2821–2833.

Hellgren J, Lunner T, Arlinger S. (1999b) Variation in the feedback of hearing aids. *J Acoust Soc Am.* 105, 3481–3496.

Kates J. (2008) *Digital Hearing Aids.* San Diego, CA: Plural Publishing.

Kates J. (2010) Understanding compression: modeling the effects of dynamic range compression in hearing aids. *Int J Audiol.* 49, 395–409.

Keidser G, Dillon H, Byrne D. (1995) Candidates for multiple frequency response characteristics. *Ear Hear.* 16 (6), 562–574.

Killion MC. (1997) Hearing aids: past, present, future: moving toward normal conversations in noise. *Br J Audiol.* 31 (3), 141–148.

Kim JS, Bryan MF. (2011) The effects of asymmetric directional microphone fittings on acceptance of background noise. *Int J Audiol.* 50, 290–296.

Kochkin S. (2010) MarkeTrak VIII: consumer satisfaction with hearing aids is slowly increasing. *Hear J.* 63 (10), 19–27.

Kuk F, Keenan D, Korhonen P, Lau CC. (2009) Efficacy of linear frequency transposition on consonant identification in quiet and in noise. *J Am Acad Audiol.* 20 (8), 465–479.

Merks I, Banerjee S, Trine T. (2006) Assessing the effectiveness of feedback cancellers in hearing aids. *Hear Rev.* 13 (4), 53–57.

Moore BCJ. (2004) Dead regions in the cochlear: conceptual foundations, diagnosis, and clinical applications. *Ear Hear.* 25 (2), 98–116.

Moore BCJ, Glasberg BR, Stone MA. (2004) New version of the TEN test with calibration in dB HL. *Ear Hear.* 25, 478–487.

Mueller HG, Weber J, Hornsby BW. (2006) The effects of digital noise reduction on the acceptance of background noise. *Trends Amplif.* 10, 83–93.

Nilsson M, Soli SD, Sullivan JA. (1994) Development of the Hearing in Noise Test for the measurement of speech reception thresholds in quiet and in noise. *J Acoust Soc Am.* 95 (2), 1085–1099.

Palmer C, Bentler R, Mueller G. (2006) Amplification with digital noise reduction and the perception of annoying and aversive sounds. *Trends Amplif.* 10 (2), 95–104.

Pumford JM, Seewald RC, Scollie SC, Jenstad LM. (2000) Speech recognition with in-the-ear and behind-the-ear dual-microphone hearing instruments. *J Am Acad Audiol.* 11 (1), 23–35.

Sarampalis A, Kalluri S, Edwards B, Hafter E. (2009) Objective measures of listening effort: effects of background noise and noise reduction. *J Speech Lang Hear Res.* 52, 1230–1240.

Simpson A, Hersbach AA, McDermott HJ. (2006) Frequency-compression outcomes in listeners with steeply sloping audiograms. *Int J Audiol.* 45 (11), 281–292.

Skinner MW. (1988) Measuring for a successful fit. In: *Hearing Aid Evaluation.* Englewood Cliffs, NJ: Prentice Hall; p 285.

Surr R, Cord M, Walden B. (2001) Response of hearing aid wearers to the absence of a user-operated volume control. *Hear J.* 54 (4), 32–36.

Walden B, Surr R, Cord M, Dyrlund O. (2004) Predicting hearing aid microphone preference in everyday listening. *J Am Acad Audiol.* 15, 365–396.

Wu Y, Stangl E. (2013) The effect of hearing aid signal processing schemes on acceptable noise levels: perception and prediction. *Ear Hear.* 34 (3), 333–341.

CHAPTER 39

Troubleshooting and Testing Hearing Aids

William Cole and Marshall Chasin

INTRODUCTION

Many clients try hearing aids with great reluctance because of social stigma and the high cost associated with the devices. This reluctance is likely to be reinforced when common problems are not minimized or prevented during the prescription and fitting process. In a survey of hearing aid owners in the United States, one in six reported that they did not use their hearing aids at all (Kochkin, 2000). Common complaints included poor fit, occlusion, feedback, wax buildup, poor service, and sweaty ears. This chapter offers prevention and troubleshooting tips for hearing aid problems common among adult clients.

LOOKING FOR PATTERNS

A detailed and up-to-date case history and background are necessary for each client. This is especially important for new clients. Even changes in weight or occupation may affect hearing aid functioning. The clinician needs to determine the client's current level of knowledge and skill regarding the care and use of his/her hearing aids. A reported problem with a hearing aid may not be due to a technically related issue but, in fact, may be caused by a lack of knowledge on the client's part and may possibly be resolved with training and counseling.

The problem can result from many possible sources. The clinician needs to determine the nature of the problem: Physical (pain in the ear); technical (hearing aid static, cutting out); acoustic (feedback); anatomic (occlusion, feedback); psychologic (adaptation to hearing aid); or emotional (anxiety/fear about the hearing aid and stigma). Remember that a client's complaint may have multiple underlying causes. Sometimes the problem is straightforward; other times, a client must be questioned closely over a series of visits. Table 39.1 lists a number of questions to guide the clinician in determining the underlying cause of a reported problem.

Patterns may point to the underlying cause of a problem. Problems may occur only in the evening, or when it is humid, or in certain locations. A client may complain that his/her hearing aid goes dead most afternoons or evenings, especially on a hot or rainy day, but usually works again the next morning. This pattern is typical for clients with persistent, dry flaky wax or skin. After several hours, the increasing levels of humidity can cause any dry wax or skin to expand enough to block the receiver tube. Overnight, the wax or skin dries out and shrinks, once again allowing sound to pass through. This pattern is also typical of drops of moisture condensing inside the hearing aid on a hot, humid day, which may cause a short circuit until the humidity level is low enough for the moisture to evaporate.

Another client may complain that whistling or feedback is heard only when he/she is visiting a particular person or location. It is possible that the hearing aid is fine and that what the client is hearing is the feedback of another person's hearing aid or simply some other high-pitched sound.

THE PROCESS OF HEARING AID TROUBLESHOOTING

Table 39.2 provides an overview of the various steps in effective troubleshooting of reported hearing aid problems. It is meant to serve as a step-by-step guide to identifying and solving reported problems.

Performing a Visual Inspection

Fit in the Ear

Before proceeding with any other step, the clinician should observe the hearing aid while it is still in the client's ear. It is often possible to see common problems such as improper placement or loose fit. If the hearing aid is not properly placed, the client may experience pain or feedback. Clients

TABLE 39.1

Patterns: Determining the Underlying Cause of a Problem

1. What is the problem?
2. How often does it happen?
3. How long does it last?
4. When did it start?
5. Is there a pattern?

TABLE 39.2

The Steps Used in Troubleshooting Reported Hearing Aid Problems

1. Look for patterns
2. Inspect the hearing aid in the client's ear
3. Disinfect the hearing aid before handling
4. Inspect hearing aid components:
   - Microphone
   - Receiver
   - Volume control
   - Program buttons and switches
   - Battery, battery door, and battery contacts
   - Ear hooks and tubing
   - Vents
5. Listen to the hearing aid
6. Perform American National Standards Institute tests on the hearing aid

require counseling and practice in the insertion of hearing aids. Clients should be counseled to recognize the signs of an improperly placed hearing aid, such as feedback, discomfort, or a decrease in sound quality or volume. If manual dexterity is an issue, a geriatric handle can be added to the aid or mold, or a caretaker can be trained to properly insert the hearing aid. For a full discussion on fit-related issues, see the section later in this chapter titled "Proper Fit."

Disinfection

Virtually every hearing aid has some kind of bacterial or fungal growth, and most have a combination of several microorganisms. Handling multiple hearing aids risks passing bacteria, molds, and fungus such as *Staphylococcus* and *Candida* between clients. Associated diseases and infections include pneumonia, meningitis, and diphtheria.

The clinician must wash his/her hands using an established infection control protocol before handling any hearing aid. The hearing aid must be thoroughly disinfected with a germicidal wipe. Ultraviolet light of a specific frequency (253.7 nm) is highly effective in infection control and is used in devices such as the Dry & Store chambers. The following list indicates the most common microorganisms found on hearing aids and earmolds (Bankaitis and Kemp, 2003):

- *Staphylococcus* (various)
- Diphtheroids
- *Pseudomonas* (various)
- *Acinetobacter lwoffi*
- *Enterobacter cloacae*
- *Lactobacillus*
- *Aspergillus flavus*
- *Candida parapsilosis*

Inspecting the Hearing Aid and Component Parts

For a review of the basic anatomy of a hearing aid, consult Chapter 38, Hearing Aid Technology. The following sections list procedures for examining different parts of a hearing aid.

Hearing Aid Shell or Earmold

Examine the hearing aid shell or the earmold for cracks or damage. Reshelling or a new mold may be indicated.

Microphone

Check the microphone for debris. If there is a wind screen or wind hood, check to see if it is blocked. Look for signs of exposure to hair sprays or fine dust. Note that there may be moisture present even if water condensation is not visible (see section on moisture). Debris may be removed gently with a suction tool. Blocked or damaged wind screens and wind hoods should be replaced. Behind-the-ear microphone covers should be routinely replaced as specified by the manufacturer.

Receiver

Inspect the receiver. Remove any wax guard, and use an otoscope to look all the way down the receiver tube. To improve depth perception, back your eye off the otoscope view finder several inches and look down the otoscope view finder with both eyes. The receiver tube should be clear and the receiver clearly visible. Gently remove any debris, if possible. A vacuum chamber or suction tool can be used to remove small amounts of deeply seated wax, debris, or moisture (see the following section on moisture).

For in-the-ear hearing aids, the receiver is particularly susceptible to damage by wax and moisture. Vigorous cleaning may also cause damage, dislodging the receiver tube and redirecting amplified output into the hearing aid cavity rather than into the ear canal. The presence of a basket-style wax guard can discourage mechanical damage from overzealous cleaning as well as prevent wax from reaching the receiver.

Effects of Moisture on a Hearing Aid

Much like a tropical jungle, the ear canal is hot, humid, and dirty. Humidity is increased because of reduced ventilation caused by wearing hearing aids. In addition, other sources can increase ear canal humidity, such as physical exertion, sweating, high humidity in the environment, and otitis externa (Gray et al., 2005). Behind-the-ear hearing aids are not directly exposed to the heat, humidity, and wax found inside the ear canal; however, they are exposed to sweat from the scalp and head.

The microphone and receiver must remain clear to pick up and transmit sounds accurately but can easily be clogged

or damaged by dirt, wax, sprays, humidity, and sweat. The microphone diaphragm vibrates according to the frequency and volume of the incoming sound and can be significantly dampened by molecules of moisture. Because a wet diaphragm cannot vibrate fast enough to clearly transmit high frequencies, sound quality is compromised.

Moisture and sweat can also cause distortion, intermittent failure, faulty buttons and switches, corrosion of metal contacts and electronics, reduced battery life, and blocked vents, filters, and tubing. The regular use of a desiccant (such as a Dri-Aid kit or Dry & Store) and protective coverings for behind-the-ear hearing aids (such as sweatbands or SuperSeals) can significantly reduce the effect of sweat and humidity on hearing aids, both reducing the need for repair and lengthening service life.

Volume Control

Examine the volume control if there is one. It should turn freely and not be too loose or too tight. A stiff volume control is often remedied by application of a contact cleaner. Remember that even if a volume control is present, it may be deactivated or may act as an on/off switch even when deactivated. A listening check will confirm the current functioning of a volume control.

Program Buttons and Switches

Examine program buttons, switches, or other controls on the hearing aid. Buttons and switches should move freely. Look for a buildup of debris or corrosion around the controls. A small brush will often remove most debris or dirt, and the careful use of a contact cleaner can loosen stiff buttons or switches. During the listening check (discussed later), listen to see if the buttons or switches are functioning correctly.

Battery, Battery Door, and Contacts

The battery contacts should grip the battery snugly but not too tightly. Look for scratches on the battery caused by tight contacts. A battery door should open smoothly and not be too tight or loose. Check for cracks or breaks in the plastic of the battery door. The hinge of in-the-ear battery doors can break fairly easily, requiring replacement. With some practice, battery doors are easy to replace, although care must be taken that the correct replacement door is used. For some makes of hearing aids, there are right-hand and left-hand battery doors that cannot be interchanged. Also, battery doors may change from model to model within the same company. If in doubt, order the required battery door based on the serial number of the hearing aid.

When removing or replacing battery doors, always take great care not to damage the hinge pin. If the metal hinge pin is broken or dislodged, the hearing aid must be sent to the manufacturer for repair. For behind-the-ear and open-fit hearing aids, the hearing aid may need to be sent to the manufacturer if the battery door needs replacing.

Take a look at the battery and battery contacts for signs of corrosion. Use a cotton swab and contact cleaner to clean dirty contacts, if needed. Occasionally, the battery may be pushed into the internal cavity of the hearing aid rather than placed into the battery door. Typically, this happens with clients with poor vision or when caretakers or friends try to change the battery. Removing the battery can solve the problem; however, it is possible that the internal wiring or circuit may have been damaged. Unless a test of the hearing aid reveals that it is functioning within specifications, the hearing aid should be sent to the company for repair. For more information, see "Batteries" section later in this chapter.

Ear Hooks (Behind-the-Ear Hearing Aids Only)

For behind-the-ear hearing aids, inspect the ear hook; loose ear hooks should be replaced. Check the tubing for any debris or moisture. If testing shows low gain compared to the manufacturer's specifications, retest the hearing aid without the ear hook. Ear hooks with filters are particularly susceptible to partial or complete blockage by debris, wax, or moisture and should be replaced as needed. Care should be taken to avoid over-tightening when replacing an ear hook. Some ear hooks cannot be replaced in-house and must be sent to the manufacturer for replacement.

Tubing (Behind-the-Ear Hearing Aids Only)

If the tubing is hardened or cracked, it should be replaced because it can allow sound to cycle back to the microphone, causing feedback.

For open-fit hearing aids, inspect the tubing and dome for blockage or damage. Because the tubing for these hearing aids is so thin, wax blockage is the most common problem and should be cleared with the tool provided by the manufacturer for this purpose. Open-fit tubing and tips should be changed regularly as recommended by the manufacturer. For those models with the receiver in the ear, there is generally a wax guard system that can be changed at the receiver.

Vents

A vent is a passage through a hearing aid or mold that allows heat to escape from the ear canal, equalizes atmospheric pressure to the ear drum, and allows excess amplification at various frequencies to escape from the ear canal.

Check to see if the vent is occluded with wax or other debris. A plugged vent may cause sweaty ears, uncomfortable pressure, or occlusion. If a client complains that feedback started suddenly and an examination shows that the hearing aid has a large open vent, a vent plug may have been

previously used but has since fallen out. A well-fitting vent plug should not fall out. When a vent plug is used, it should still provide a release of pressure if possible.

Performing a Listening Check

When listening directly to a hearing aid, use a custom listening ear piece for the best sound quality. Disinfect the flexible coupler of the listening piece (as described in the section earlier on infection control) before and after listening to a hearing aid.

With high-power hearing aids, always turn the volume to the lowest setting and do not insert the listening ear piece deep in the ear. Ensure a tight fit between the listening ear piece and the hearing aid to avoid painful feedback. Always remember that the sound pressure levels (SPLs) generated by power hearing aids are capable of causing damage to the clinician's inner ear.

Before doing a listening check, remove any wax guards, microphone protectors, ear hooks, or tubing. This will ensure that you are testing the hearing aid circuit. Always use a fresh battery to rule out battery-related issues. When performing a listening check of a hearing aid, listen for the following:

- Clear and consistent sound
- Smooth increase/decrease of sound when operating the volume control (if activated)
- Obvious distortion
- Cutting in and out
- Static or cutting out when operating the volume control
- Static or cutting out when operating the toggle switches or push buttons
- The hearing aid's frequency response, using the Ling six sound test (Estabrooks and Birkenshaw-Fleming, 2003)
  - /m/ Low and middle frequencies
  - /oo/ Low frequencies
  - /ah/ First and second formants, middle frequencies
  - /ee/ First formant low frequency, second formant higher frequency
  - /sh/ Middle and high frequencies
  - /s/ High frequencies

A listening check may indicate that a hearing aid is functioning well, but it cannot replace a formalized electroacoustic test, which determines whether the hearing aid is functioning according to manufacturer's specifications.

Troubleshooting with Hearing Aid Analyzers

A typical hearing aid analyzer is shown schematically in Figure 39.1. A signal generator [6] provides test signals to a loudspeaker [1]. The level and spectrum of the signal is measured and controlled by a reference microphone [5] in conjunction with the signal generator. The output of the hearing aid [2] is coupled to a measuring microphone [3] and measuring system [7] by a coupler [4]. To reduce the impact of ambient noise on the intended stimuli or on the operation of the hearing aid, the sound source and hearing aid may be enclosed in a sound-isolating test chamber [8] that is lined with sound-absorbing material [9] to improve sound-field uniformity. In most cases, the stimulus is acoustic, but it may also be magnetic in cases where the performance of a telephone coil is to be tested.

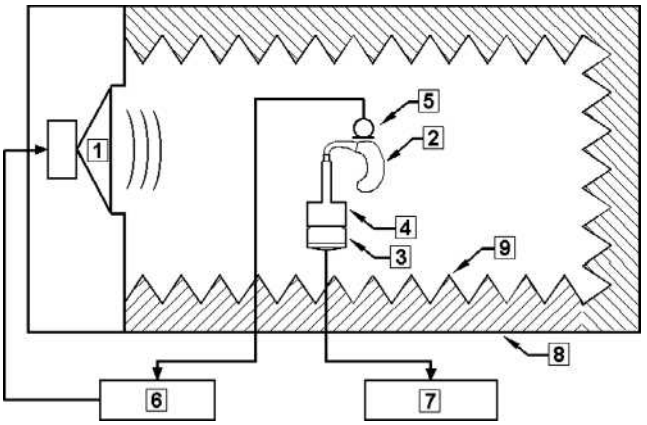


FIGURE 39.1 Schematic representation of a hearing aid analyzer. Acoustic test signals from loudspeaker [*1*] are generated and controlled by signal generator/control system [*6*] in conjunction with reference microphone [*5*]. Output of the hearing aid [*2*] is coupled to a measuring microphone [*3*] and measuring system [*7*] by a coupler [*4*]. Sound-isolating enclosure [*8*] is lined with sound-absorbing material [*9*] to reduce standing waves.

Test Signals

A variety of test signals are available in the modern hearing aid analyzer. These include steady puretones, steady pseudo-random noise, modulated puretones, modulated noise, and real speech or music. It must always be remembered that, when testing hearing aids with compression or any other adaptive processing features, the results obtained will be valid only for the test signal used. Although it may be tempting and convenient to generalize performance for complex signals from measurements done with simpler signals, the error in doing so will be hearing aid dependent, and this error increases as the difference in signals increases (Henning and Bentler, 2005; Scollie and Seewald, 2002).

In the configuration shown in Figure 39.1, the test signal is controlled by placing a small calibrated microphone very close to the hearing aid microphone port(s) and using the measured SPL to control the signal to the loudspeaker. This is known as the pressure method and is the preferred method in ANSI/ASA S3.22 (ANSI S3.22, 2009). Alternately, a substitution method may be used to control the test signal. In this case, an equalization (leveling) step is performed prior to the test. In this step, the microphone is removed from the coupler and is used to measure the sound

field near the hearing aid microphone port while a known electrical signal is applied to the loudspeaker. For greatest accuracy, all objects should be positioned in the test chamber just as they will be during the test, and a dummy microphone should be installed in the coupler. After the equalization step, the dummy microphone and the coupler microphone are interchanged for subsequent tests.

Couplers

The coupler that connects the hearing aid to the measuring microphone also serves as an acoustic load on the hearing aid. This acoustic load has a strong influence on the measured output of the hearing aid, and several have been standardized for hearing aid testing. The most common has a volume of 2 cm^3 and is frequently referred to as a 2-cc coupler. The American National Standard Method for Coupler Calibration of Earphones (ANSI S3.7, 1995) defines this coupler and provides several variations to accommodate different hearing aid configurations. ANSI/ASA S3.22 (ANSI S3.22, 2009) specifies which of these variations is to be used with different hearing aid types.

In-the-ear and in-the-canal devices, including deep-insertion hearing aids, are to be tested in a type HA-1 coupler (Figure 39.2), which has a direct entrance to the 2-cm^3 cavity. The acoustic coupling between the sound outlet of the hearing aid and the coupler entrance must be made airtight by using an appropriate sealant.

Hearing aids that employ a button-type receiver are to be tested using the type HA-2 coupler (ANSI S3.22, 2009). This coupler (Figure 39.3) has the entrance to the cavity through an earmold substitute having a 3-mm diameter sound bore that is 18-mm long. All hearing aids that couple to the ear by means of a length of tubing are to be tested using type HA-2 with entrance through a tube. ANSI/ASA S3.22 permits two variations on the tubing used in this coupler. The first is intended for most postauricular (behind-the-ear) hearing aids and consists of a rigid tube with a 2-mm inside diameter and a length of 25 mm between the earmold substitute and the tip of the ear hook (Figure 39.4). The second variation (type HA-4) is like the first except that both the earmold substitute and the connecting tubing have a 1.93-mm diameter sound bore, creating a uniform sound path with a length of 43 mm. This was originally intended for use with eyeglass hearing aids, which used skeleton-type earmolds and a continuous length of no. 13 tubing (inside diameter of 1.93 mm).

A third variation of the HA-2 coupler is permitted by ANSI/ASA S3.22 for testing modular in-the-ear hearing aids. In this variation, designated the HA-3 coupler, the tubing connects directly from the hearing aid receiver outlet to the entrance to the cavity. This tubing is required to have an inside diameter of 1.93 mm and a length from the receiver case to the cavity entrance of 10 mm.

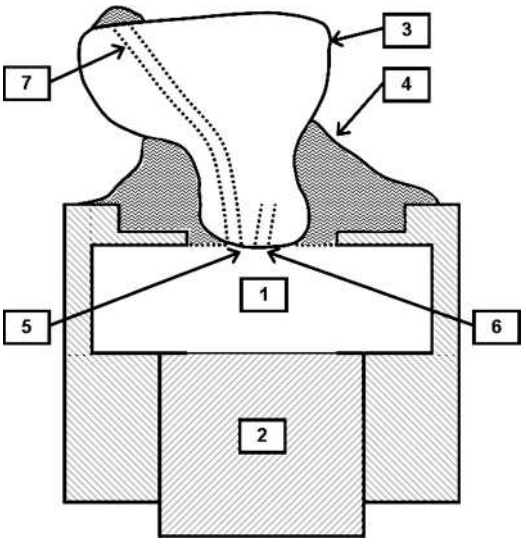


FIGURE 39.2 The HA-1 hearing aid coupler. The coupler microphone [*2*] is concentric with the cavity [*1*], which has a diameter between 18 and 21 mm and a volume of 2.0 cm^3 ± 1%. Hearing aid [*3*] is sealed to the cavity with putty [*4*], such that the tip [*5*] is even with the cavity wall and the sound outlet [*6*] is approximately centered in the opening. Vent [*7*] is sealed at the faceplate.

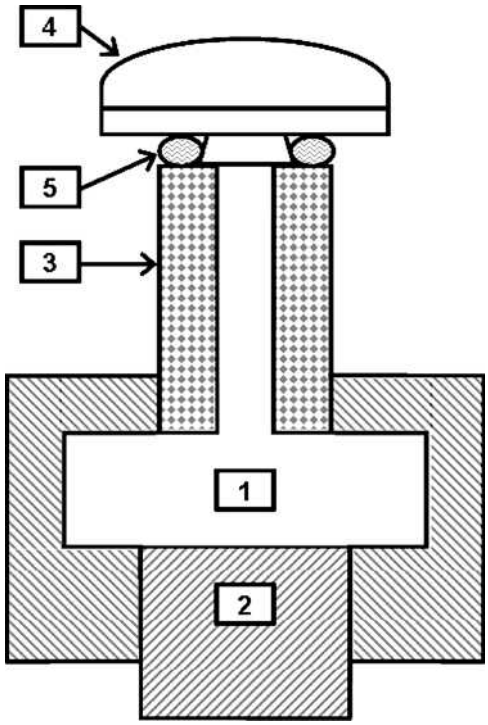


FIGURE 39.3 The HA-2 hearing aid coupler. The coupler microphone [*2*] is concentric with the cavity [*1*], which has a diameter between 18 and 21 mm and a volume of 2.0 cm^3 ± 1%. Entrance to the cavity is through an earmold substitute [*3*] having a 3-mm diameter sound bore with a length of 18 mm. The earphone [*4*] is sealed to the earmold substitute with a suitable sealing mechanism [*5*].

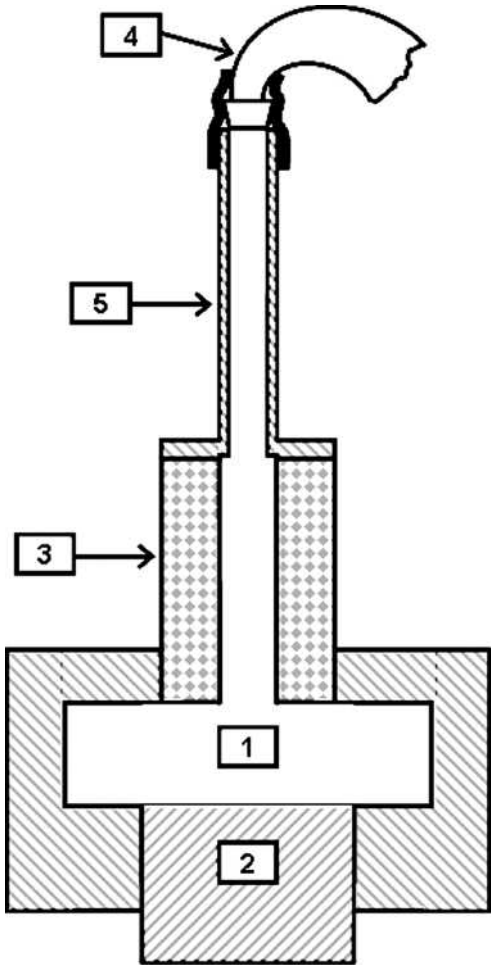


FIGURE 39.4 The HA-2 coupler with entrance through a rigid tube. The coupler microphone [*2*] is concentric with the cavity [*1*], which has a diameter between 18 and 21 mm and a volume of 2.0 $cm^3 \pm 1\%$. Entrance to the cavity is through an earmold substitute [*3*] having a 3-mm diameter sound bore with a length of 18 mm. Coupling from the tip of the hearing aid hook [*4*] to the earmold substitute is via a rigid tube [*5*] with a 2-mm inner diameter and a length of 25 mm.

The coupler to be used for postauricular hearing aids with the receiver in the ear canal is not explicitly indicated in ANSI S3.22, but the HA-1 coupler is intended to be used with hearing aids having ear tips (ANSI S3.7, 1995), and this is the logical choice for reporting test results for these devices. The tip and coupling system used should be of average size and should be stated.

Vented or Open Fittings

There are no standardized couplers (see the following "Ear Simulators" section) or test methods capable of characterizing hearing aids operating with large vents or in open ear canals. "Open-fit" hearing aids must be tested using one of the closed coupler configurations previously described. Postauricular hearing aids with thin coupling tubes are often provided with a standard ear hook; thus, they can be tested using the HA-2 coupler. If this is not the case, the open end of the thin tube must be sealed to the entrance of the HA-1 coupler using an adapter or an appropriate sealant. The exact configuration should be specified by the manufacturer, and any required adapters should be available from the manufacturer for test purposes.

Ear Simulators

The 2-cc coupler does not accurately represent the acoustic impedance or resonances of a real ear, and its usefulness as a test load is confined to the 200- to 5,000-Hz range. Ear simulators are designed to more closely approximate the acoustic impedance and resonance characteristics of an average of ears over a wide frequency range. Their use is required when realistic estimates of performance of deep insertion, vented and open fittings, and feedback suppression systems are desired. In some cases, a pinna may be part of the simulator, and the combination may be part of an acoustic manikin.

Simulators are more complex, more expensive, and more difficult to maintain than the 2-cc coupler, and this has confined their use to the laboratory. The Zwislocki coupler (Zwislocki, 1971), which is no longer commercially available, is one such ear simulator. Standards for occluded ear simulators (OES) are ANSI/ASA S3.25 (ANSI S3.25, 2009) and IEC 60318-4 (2010).

AMERICAN NATIONAL STANDARDS INSTITUTE HEARING AID TESTS

In recent years, there has been a concerted effort to harmonize ANSI and IEC standards relating to hearing aids by one organization directly adopting all or part of a standard produced by the other. As a result, there are now few differences between parallel standards and those differences tend to be small.

There are five standards published by ANSI that relate to the testing of hearing aids. These are described here.

First is ANSI/ASA S3.22, Specification of Hearing Aid Characteristics (ANSI S3.22, 2009). This standard describes methods of measuring a number of hearing aid characteristics and provides allowable tolerances for those that are deemed important for the maintenance of product uniformity and compliance with published specifications. The parallel IEC standard is IEC 60118-7 which is currently under revision and has largely been harmonized with ANSI/ASA S3.22.

The use of portions of the 2003 version of this standard is mandated by the Food and Drug Administration (FDA). As of this writing, the use of the 2009 revision has not been mandated by the FDA. This standard also contains procedures for many other tests that are not required by the FDA. The differences between the 2003 and 2009 versions are small, the most significant of which are the inclusion of

a definition of compression, expansion, compression ratio, and expansion ratio, the allowance of the use of a broadband noise signal for gain measurement if the result does not differ from the gain measured with a puretone by more than 1 dB, and the removal of tolerances on input–output and attack–release time tests for AGC instruments and their relocation to an informative annex.

All hearing aid manufacturers are required to include in the product brochure or other labeling that accompanies the hearing aid the technical data listed in Table 39.3 with

TABLE 39.3

Data Obtained from ANSI S3.22-2003 that Must be Provided by the Hearing Aid Manufacturer in a Product Brochure or in Other Labeling that Accompanies a Hearing Aid

Requirement	Description of Required Data
Abbreviations	HFA: High-frequency average–the average of values at 1,000, 1,600, and 2,500 Hz SPA: Special-purpose average–the average of values at three frequencies specified by the hearing aid manufacturer that are at one-third octave frequencies separated by two-thirds octave RTS: Reference test setting–setting of the gain control (i.e., volume control, master or overall gain control) required to produce an HFA gain within ±1.5 dB of the HFA-OSPL90 minus 77 dB for a 60-dB input sound pressure level (SPL) or, if the full-on HFA gain for a 60-dB input SPL is less than the HFA OSPL90 minus 77 dB, the full-on setting of the gain control AGC: Automatic gain control–means for controlling gain as a function of signal level; it includes various forms of compression
OSPL90 curve	Coupler SPL as a function of frequency for a 90-dB input SPL and gain control at full on
HFA-OSPL90	The average of the OSPL90 values at the HFA or SPA frequencies
HFA full-on gain (HFA-FOG)	The average of the full-on gain at the HFA or SPA frequencies
Reference test gain (RTG)	The average of the gain at the HFA or SPA frequencies for a 60-dB input SPL, with gain control at RTS
Frequency response curve	The coupler SPL as a function of frequency for a 60-dB input SPL, with gain control at RTS
Frequency range	The range between the lowest and the highest frequency at which the frequency response curve is 20 dB below its HFA or SPA value
Total harmonic distortion (THD)	The ratio of sum of the powers of all the harmonics to the power of the fundamental
Equivalent input noise (EIN)	The SPL of an external noise source at the input that would result in the same coupler SPL as that caused by all the internal noise sources in the hearing aid
Battery current	The electrical current drawn from the battery when the input SPL is 65 dB at 1,000 Hz and the gain control is at RTS
Induction coil sensitivity (HFA-SPLITS)	For hearing aids with an inductive input coil (telecoil), the average of the coupler SPL at the HFA or SPA frequencies when the hearing aid, with gain control at RTS, is appropriately positioned on a telephone magnetic field simulator (TMFS)
Input–output curve	For hearing aids with AGC, the coupler SPL as a function of the input SPL at one or more of 250, 500, 1,000, 2,000, 4,000 Hz, with the gain control at RTS
Attack time	For hearing aids with AGC, the time between an abrupt change from 55- to 90-dB input SPL and the time when the coupler SPL has stabilized to within 3 dB of the steady value for a 90-dB input SPL, at one or more of 250, 500, 1,000, 2,000, or 4,000 Hz, with the gain control at RTS
Release time	For hearing aids with AGC, the time between an abrupt change from 90- to 55-dB input SPL and the time when the coupler SPL has stabilized to within 4 dB of the steady value for a 55-dB input SPL, at one or more of 250, 500, 1,000, 2,000, or 4,000 Hz, with the gain control at RTS

TABLE 39.4

Food and Drug Administration–Mandated ANSI S3.22-2003 Tests and their Parameters and Tolerances

Test	Gain Setting	AGC	Input	Frequency	Measure or Calculate	Tolerance
OSPL90 curve	Full on	Min	90 dB SPL	200–5,000 Hz	Coupler SPL	Unspecified
Maximum OSPL90	Full on	Min	90 dB SPL	Frequency of maximum	Maximum of OSPL90 curve	+3 dB
HFA- or SPA-OSPL90	Full on	Min	90 dB SPL	HFA or SPA	Average coupler SPL at HFA or SPA frequencies	±4 dB
HFA or SPA full-on gain (HFA- or SPA-FOG)	Full on	Min	50 dB SPL	HFA or SPA	Average gain at HFA or SPA frequencies	±5 dB
Reference test gain (RTG)	RTS	Min	60 dB SPL	HFA or SPA	Average gain at HFA or SPA frequencies	Unspecified
Frequency range	RTS	Min	60 dB SPL	From the lowest frequency (f_1) to the highest frequency (f_2) at which the frequency response curve is 20 dB below its HFA or SPA average		Unspecified
Frequency response curve	RTS	Min	60 dB SPL	From the higher of f_1 or 200 Hz to the lower of f_2 or 5,000 Hz; wider range may be shown	Coupler SPL or gain	±4 dB from the lesser of 1.25 f_1 or 200 Hz to 2 kHz ±6 dB from 2 kHz to the lesser of 4 kHz or 0.8 f_2
Total harmonic distortion (THD)	RTS	Min	70 dB SPL 65 dB SPL	500, 800, or half the lower two SPA frequencies 1,600 or half the highest SPA frequency		+3%
Equivalent input noise (EIN)	RTS	Min	OFF and 50 dB SPL	(Coupler SPL with no input)–(HFA or SPA gain with a 50-dB input SPL)		+3 dB
Battery current	RTS	Min	65 dB SPL	1,000 Hz	Battery current	+20%
SPL for an inductive telephone simulator (SPLITS)	RTS	Min	TMFS	200–5,000 Hz	Coupler SPL. Orient aid on TMFS for maximum output. Place BTE as flat as possible on test surface. ITE and ITC with faceplate as close as possible and parallel to test surface	Unspecified
HFA or SPA SPLITS	RTS	Min	TMFS	HFA or SPA	Average SPLITS values at the HFA or SPA frequencies	±6 dB
Input–output curves	RTS	Max	50–90 dB SPL in 5-dB steps	One or more of 250, 500, 1,000, 2,000, 4,000 Hz	Coupler SPL versus input SPL	±5 dB at 50- and 90-dB input SPL when matched at 70-dB input SPL
Attack time	RTS	Max	Step from 55 to 90 dB SPL	Same frequencies used for input-output curves	Time from input step until coupler SPL settles within 3 dB of its steady value for 90-dB input SPL	±5 ms or 50%, whichever is greater
Release time	RTS	Max	Step from 90 to 55 dB SPL	Same frequencies used for input-output curves	Time from input step until coupler SPL settles within 4 dB of its steady value for 55-dB input SPL	±5 ms or 50%, whichever is greater

AGC, automatic gain control; Min, minimum; Max, maximum; SPL, sound pressure level; RTS, reference test setting; HFA, high-frequency average; SPA, special-purpose average; TMFS, telephone magnetic field simulator; BTE, behind-the-ear; ITE, in-the-ear; ITC, in-the-canal.

measurement conditions and tolerances as given in Table 39.4. When troubleshooting a hearing aid, these required data are the benchmarks against which measured performance should be verified. It should be noted that, when verifying compliance with manufacturers' specifications, the indicated tolerance plus measuring equipment accuracy must be added to the value listed by the manufacturer. For example, if the measurement equipment accuracy is ±1 dB and the tolerance given in the table for a particular test is ±4 dB, then a measured value within ±5 dB of the value listed by the manufacturer would be considered to be within specification.

When interpreting the results of the tests included in this ANSI standard, it is important to remember the following points:

1. ANSI S3.22 is a quality control standard. The data apply only for the puretone signals and measurement conditions, hearing aid settings, and configuration employed when they were generated. They do not predict performance for other signals, conditions, settings, or configurations.
2. Measurements are defined only for the frequency range 200 to 5,000 Hz. A wider range may be shown for informational purposes.
3. Attack and release times include any processing delay through the hearing aid. For digital hearing aids, this will account for some fixed portion of the reported times, typically between 3 and 10 ms. Given the allowed tolerances on these quantities, this is unlikely to be significant.
4. Attack and release times are very dependent on the hearing aid settings and the test protocol. Do not assume they represent times likely to be experienced in actual use.

The second standard is ANSI/ASA S3.35, Method of Measurement of Performance Characteristics of Hearing Aids under Simulated Real-Ear Working Conditions (ANSI S3.35, 2010). This standard provides terminology and techniques for the precise determination of simulated insertion gain, three-dimensional directional response, and directivity index using a suitable manikin and ear simulator. It gives requirements for the test space (typically a large anechoic chamber) and the test equipment. Hearing aids must be placed in a linear, nonadaptive mode of operation. This is a voluntary standard, and its use is not mandated by any government regulation. The parallel IEC standard is IEC 60118-8: Methods of measurement of performance characteristics of hearing aids under simulated in situ working conditions.

The third standard is ANSI/ASA S3.42 Part 1, Testing Hearing Aids with a Broad-Band Noise Signal (ANSI S3.42, 1992). This standard defines the spectrum of a broadband noise test signal and specifies analysis methods for obtaining the steady-state output and gain of hearing aids using this signal. It should be noted that the specified spectrum of the test signal is that of the peaks of speech, not the long-term average speech spectrum (LTASS). As such, it has considerably more high-frequency content than is found in speech-weighted noise based on the LTASS (see Figure 39.5 for a representation of the S3.42 Part 1 noise spectrum and LTASS). As noted in the standard, the steady-state gain and output obtained using this standard are not representative of the gain or output for real speech signals processed by compression hearing aids (Henning and Bentler, 2005; Scollie and Seewald, 2002). This is a voluntary standard, and its use is not mandated by any government regulation. The parallel IEC standard is IEC 60118-0: Measurement of electroacoustical characteristics, which is currently under revision.

The fourth standard is ANSI/ASA S3.42/Part2 (ANSI S3.42, 2012), Methods for Characterizing Signal Processing in Hearing Aids with a Speech-Like Signal. This is a direct adoption of IEC 60118-15 which specifies an International

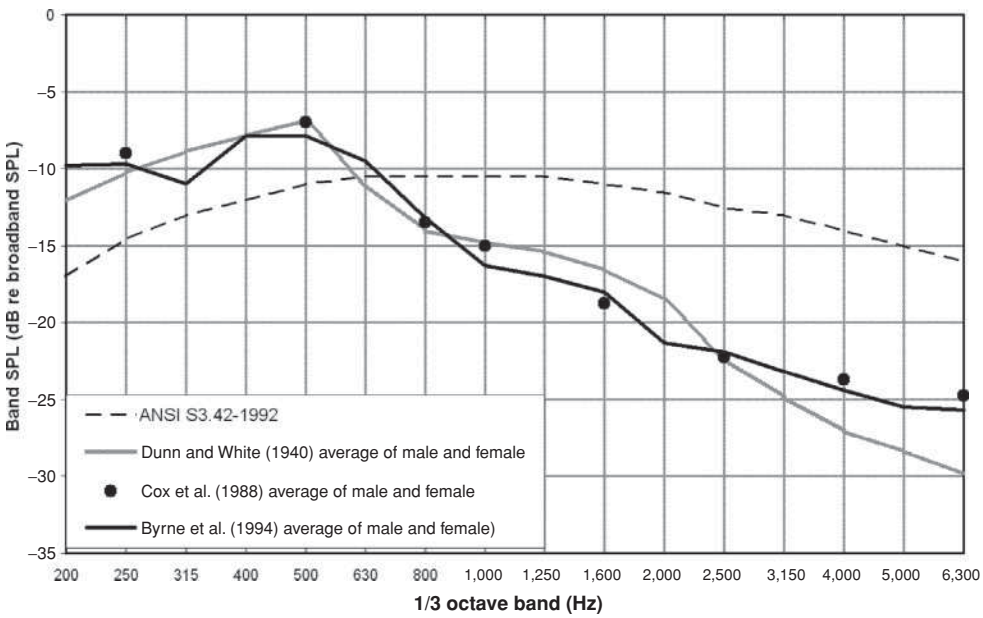


FIGURE 39.5 The ANSI S3.42-1992 noise spectrum and selected long-term average speech spectra.

Speech Test Signal (ISTS) to be used as the input signal for all tests. This signal was derived by combining segments of recorded speech from six female talkers speaking different languages. The hearing aid output in an ear simulator or 2-cc coupler is analyzed in one-third octave bands and the 30th, 65th, and 99th percentiles of the distribution of SPL in each band are calculated. This is then converted to a coupler or estimated real-ear gain for the three percentiles and the LTASS.

Finally, the fifth standard is ANSI/ASA S3.46, Methods of Measurement of Real-Ear Performance Characteristics of Hearing Aids (ANSI S3.46, 2013). This standard defines terms used in real-ear measurements and provides guidance on procedures for both closed and vented fittings, sources of error, and essential reporting and equipment requirements. A definition and procedure for measuring the real-ear to coupler difference (RECD) and an annex describing sources of error in its measurement and application are among the changes in this revision. This is a voluntary standard, and its use is not mandated by any government regulation.

Troubleshooting Using ANSI/ASA S3.22 Tests

Most commercial hearing aid analyzers provide automated test sequences that make it easy to run the FDA-mandated hearing aid tests of ANSI/ASA S3.22. Although these tests do not represent performance at "use" settings, it is good practice to run them for comparison with manufacturers' data when a hearing aid is first received, when it has been repaired, or when a malfunction is suspected.

The tests of ANSI/ASA S3.22 are run with all adaptive features disabled and three different hearing aid setups (for more in-depth discussion of some of the hearing aid parameters discussed in the following list, the reader is referred to Chapter 38):

- The output sound pressure level with a 90-dB input (OSPL90) and full-on tests are run with any automatic gain control (AGC) function set for minimum effect (i.e., its most linear operation) and the maximum output and gain set to their highest values. Most programming software provide a setting that accomplishes this. If this is not the case, setting the AGC function for minimum effect may be accomplished by disabling the AGC (if possible), by setting the compression threshold to its highest setting, and/or by setting the compression ratio as close to 1.0 as possible, while still keeping maximum output and gain at their highest values.
- All other tests are run with the gain control at the reference test setting. Most programming software provide a setting for this. If this is not the case, most commercial hearing aid test systems provide automated assistance in making this adjustment.
- Attack and release times and input–output curve tests are performed with the AGC function set to have maximum effect. Some programming software may provide a setting that does this. If not, setting the AGC function for maximum effect may be accomplished by setting low-level gain as high as possible and setting high-level gain and maximum output as low as possible. This will typically result in a very low compression threshold, a very flat input–output curve above this threshold, and maximum attack and release times. These results should be interpreted as representing the extremes of what is attainable, not what is typical in use.

Manufacturers are required to indicate the control or software settings or provide test programs used for all tests. These settings must be used when verifying performance against test strips or specification sheets.

Before running the ANSI/ASA S3.22 tests, clinicians should do the following:

1. Install a fresh battery, or use the battery substitute in the analyzer.
2. For behind-the-ear aids, ensure that the plastic tubing on the HA-2 coupler is flexible, free from splits, and of the correct length and diameter. The tubing between the tip of the ear hook and the entrance to the earmold simulator should be 2 mm in diameter and 25 mm in length. Some analyzers include all of this tubing within the HA-2 coupler, using a very short piece of earmold tubing only to seal the tip of the ear hook to the internal tubing. Others include varying amounts of the 2-mm section within the coupler, with the remainder (usually 15 to 25 mm) made up of flexible tubing added by the user. In these cases, both the internal diameter and the total length must be as per the ANSI guidelines.
3. For behind-the-ear aids, ensure that the ear hook and dampers are as specified in the manufacturer's test data and that they are free of obstructions.
4. In-the-ear, in-the-canal, completely-in-the-canal, and other deep-insertion hearing aids should be well sealed to the HA-1 coupler with their tip flush with the entrance to the 2-cc cavity.
5. Any wax guards or microphone screens specified by the manufacturer must be in place and free of obstructions. Any not specified by the manufacturer should be removed.
6. Vents must be plugged at the faceplate (custom aids) or external (behind-the-ear) end.
7. The hearing aid should be set to the omnidirectional mode, its widest frequency response range, greatest high-frequency average (HFA) OSPL90 or special-purpose average (SPA) OSPL90, and, if possible, greatest HFA or SPA full-on gain. The HFA frequencies are 1,000, 1,600, and 2,500 Hz; the SPA frequencies are specified by the manufacturer and are one-third octave frequencies separated by two-thirds octave. Any AGC function should be set to have minimum effect, and any adaptive features should be disabled. Settings or a program

TABLE 39.5

Deviations from Manufacturers' Specifications and their Possible Cause and Remedy

ANSI Test	Result	Possible Cause–*Remediation*
OSPL90 curve	Large peaks or notches in the low to mid-frequencies [see Figures 39.6 and 39.7]	Open vent–*close at faceplate end* Poor seal of hearing aid tip to HA-1 coupler–*reseal* Cracked tubing on HA-2 coupler–*replace* Defective ear hook–*replace*
OSPL90 curve	Curve is very jagged	Aid is not set to test program–*correct settings*
OSPL90 curve, maximum OSPL90, HFA-OSPL90	Curve is well below manufacturer's reported results Maximum and HFA-OSPL90 are below tolerance	Defective battery–*try new battery, different batch* Restricted airflow to zinc air battery–*clean air holes or grooves in battery compartment* Wrong or blocked ear hook–*replace/clean ear hook* Blocked wax guard–*clean or replace* Blocked receiver tube–*clean or repair* Defective receiver–*repair*
HFA-FOG	HFA-FOG is below tolerance, but OSPL90 tests are OK	Blocked microphone port(s)–*clean, replace filters* Defective microphone–*repair*
Frequency response curve	Curve is below tolerance at some frequencies, but OSPL90 tests are OK	Aid not in omnidirectional mode–*change settings* Blocked microphone port(s)–*clean, replace filters* Defective microphone–*repair*
Frequency response curve	Curve has sharp peaks at one or two frequencies	Feedback–*check seal to coupler and vent closure* Cracked tubing on HA-2 coupler–*replace* Defective hook–*replace* Internal feedback–*repair*
Total harmonic distortion	Levels are above allowed tolerance	Defective battery–*try new battery, different batch* Restricted airflow to zinc air battery–*clean air holes or grooves in battery compartment* Defective receiver–*repair* Defect in circuit–*repair*
Equivalent input noise	Levels are above allowed tolerance	Noise in the test environment–*repeat test in quiet* Blocked microphone port(s)–*clean, replace filters* Defective microphone–*repair* Specifications may be with expansion enabled–*check settings used by manufacturer for this test*
Attack and release times	Values are beyond tolerance limits	Noise in the test environment–*repeat test in quiet* Test settings do not match those specified by the manufacturer for these tests–*correct settings* Defect in circuit–*repair*

ANSI, American National Standards Institute; HFA, high-frequency average; FOG, full-on gain.

to achieve these conditions should be provided by the manufacturer.

Table 39.5 lists some potential deviations from manufacturers' specifications and their possible cause and remedy.

It is important to make sure that any given test has been run properly, with the hearing aid set exactly as specified, before attempting to draw any conclusions from the test results. Failing to close vents or improper sealing of the hearing aid or earpiece to the HA-1 coupler can result in curves like those in Figures 39.6 and 39.7. Please note that both curves are output SPL curves versus the typically displayed gain curves when illustrating the effects of venting on hearing aid performance.

Some "rules of thumb" for interpreting ANSI/ASA S3.22 test results are as follows:

- OSPL90 tests generally provide information about the output components of the hearing aid (i.e., receiver, hook, dampers, wax guards) or the power source.

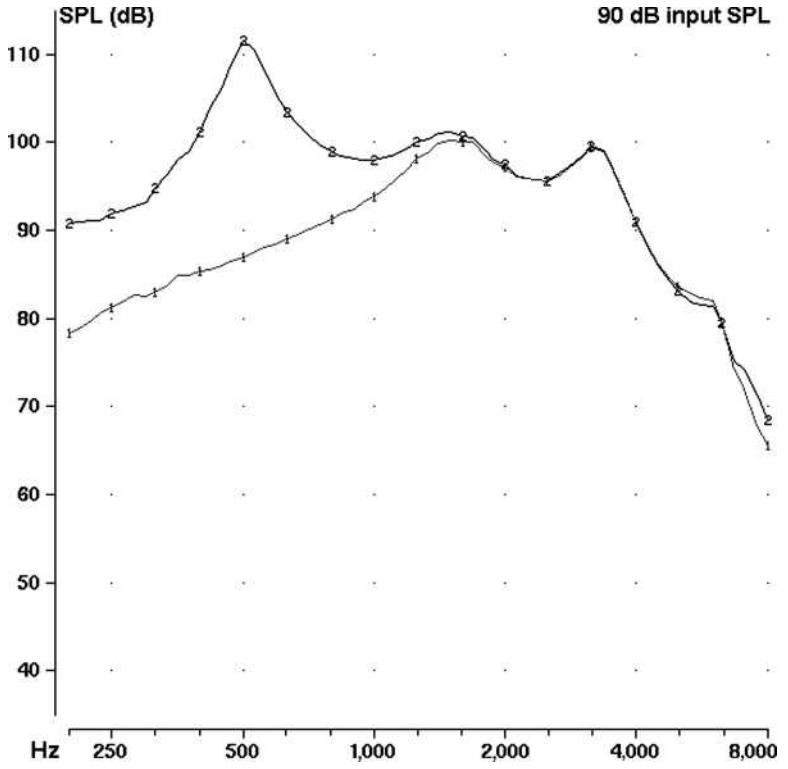


FIGURE 39.6 Effect of an open vent on an OSPL90 curve for an in-the-ear hearing aid. The lower curve (*1*) is the correct curve, obtained with the vents sealed at the faceplate (outer) end; the upper curve (*2*) is obtained with the vent open; note that the low-frequency sound enters directly through the open vent unamplified, yielding more low-frequency energy in the output than if the vent was plugged. In addition, the open vent, in conjunction with the 2-cc cavity, forms a resonator for sounds entering directly through the vent, resulting in the boost observed at 500 Hz. This effect depends on vent dimensions, the hearing aid, and its settings.

- Gain tests generally provide information about both the output and the input components of the hearing aid (i.e., microphones, wind filters).
- Distortion tests generally provide information about the receiver or power source.
- Equivalent input noise tests will generally provide information about the microphone(s).
- AGC tests may detect faulty components in analog circuits, but in digital hearing aids, these characteristics are controlled by software. Consequently, for digital circuits,

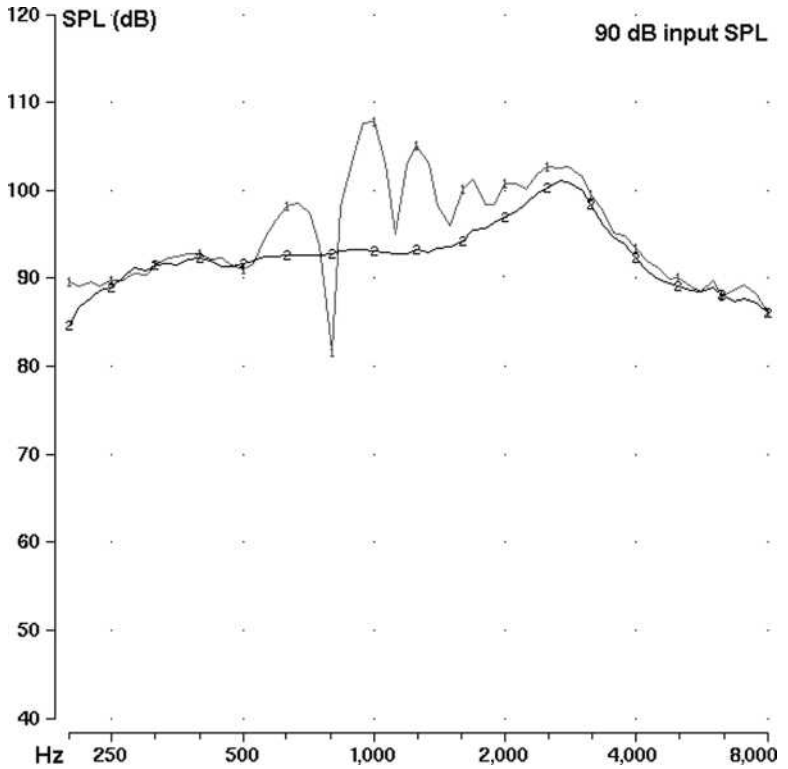


FIGURE 39.7 Effect of a poor seal to the coupler on the OSPL90 curve for an in-the-canal hearing aid. The smooth curve (*2*) is obtained with a good seal; the irregular curve (*1*) is obtained with a poor seal to the coupler. The magnitude of the effect depends on the hearing aid and its settings.

it is extremely unlikely that these tests will be failed by themselves. Failures in digital circuits are more likely to impact many or all of the tests.

Troubleshooting When ANSI/ASA S3.22 Cannot Be Used

Sometimes data sheets or test strips are not available, programming software is not at hand, programming connectors are broken, cables cannot be readily obtained, or the hook is not the one used for the ANSI tests. In these cases, test results cannot be directly compared with ANSI specifications to determine if the hearing aid itself is functioning properly. The hearing aid analyzer then becomes a useful tool for probing the hearing aid to determine if it is performing in an acceptable fashion.

Perhaps the simplest deviation from the ANSI test conditions is the use of an ear hook different from the one used to generate the specifications. Running the ANSI tests with a different hook, especially one with different dampers, is likely to change the peaks in the OSPL90 and frequency response curves and the numerical data derived from them. However, the data should still indicate whether the hearing aid is performing reasonably close to expectations. If the ear hook is damaged or permanently blocked and a replacement is not readily available, it is still possible to determine if the hearing aid itself is functioning by replacing the hook with a length of earmold tubing that has the same length as the hook (this gets added to any tubing that would normally be attached to the coupler). Figure 39.8 shows frequency response curves for a behind-the-ear aid with the proper damped ear hook (lower curve) and with the hook replaced with earmold tubing having the same length as the ear hook but without dampers (upper curve). Although the peaks in the response curve are no longer damped, running an ANSI test battery in this case will still provide data that may be compared to the ANSI specifications to help decide if the hearing aid is functioning properly and simply needs a new hook or if it has more serious problems and needs to be sent for repair.

If it is not possible to disable adaptive features as required for the ANSI tests or if it is simply desired to test the hearing aid at its "use" settings, consideration must be given to the response of the adaptive features to the test signal being used. Adaptive features are most likely to be activated by signals that are tonal or unchanging, and this activation may occur some time after the signal is applied. Running an ANSI test battery in this situation may produce erratic OSPL90 and frequency response curves. Tests of distortion or attack/release time are unlikely to be reliable because the relatively long duration of these tests will likely give the adaptive features time to react.

USE OF ACOUSTIC STIMULI TO ASSESS NONADAPTIVE HEARING AID CHARACTERISTICS

Tests Using Speech-Like Test Signals

There are a number of ways to extract useful information about the condition of the hearing aid without disabling

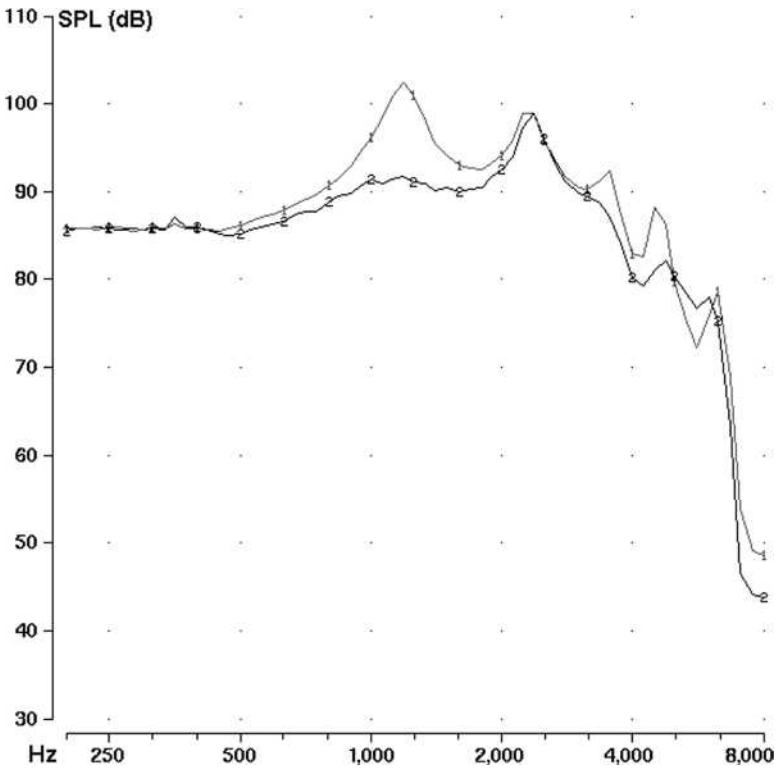


FIGURE 39.8 Effect of ear hook on frequency response curves. The lower curve (*2*) was obtained with the manufacturer-specified damped ear hook. The upper curve (*1*) was obtained with the hook replaced with earmold tubing having the same length as the ear hook and no dampers.

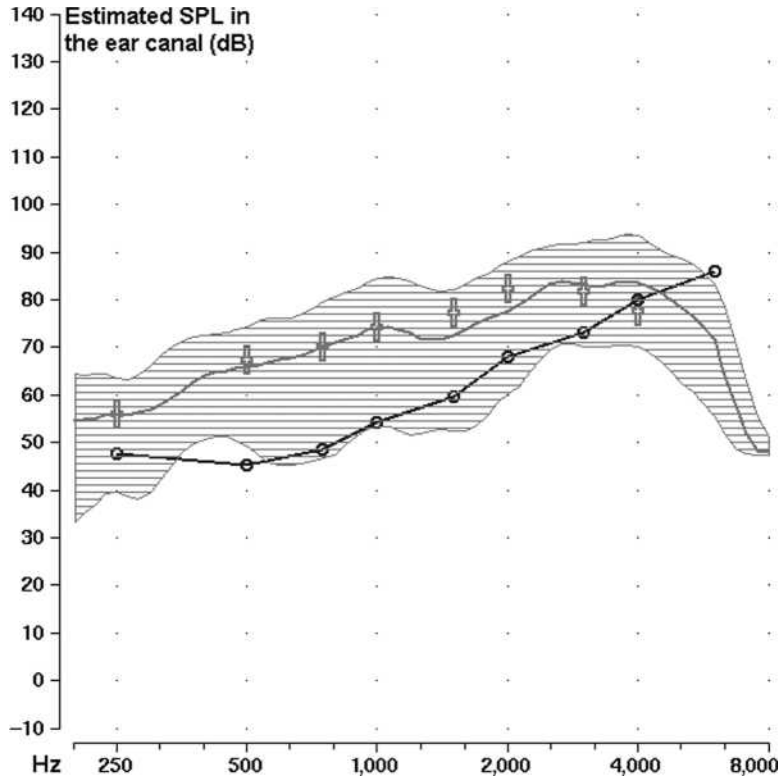


FIGURE 39.9 Using speech to check hearing aid operation with adaptive features enabled. The *hatched area* is the amplified speech region bounded by the peaks of speech at the top and the valleys at the bottom. The *heavy curve* is the long-term average speech spectrum (LTASS), the *circles* are the hearing threshold converted to sound pressure level (SPL), and the *elongated + marks* are the NAL-NL1 real-ear targets for the amplified LTASS. The input was real speech at 65 dB SPL. Ear canal SPL has been estimated by adding an average real-ear to coupler difference (RECD) to the coupler SPL. Noise reduction, feedback suppression, and adaptive directional features are enabled.

adaptive features. The method with the least likelihood of inadvertently triggering noise reduction, feedback suppression, or adaptive directional features uses speech-like signals and determines gain or output. These may be real speech signals or speech-like signals such as the ISTS (ANSI S3.42, 2012), modulated noise, or International Collegium of Rehabilitative Audiology (ICRA) noise, a signal derived by digital manipulation of real speech (Dreschler et al., 2001). The amplified speech-like signal may be compared to the patient's hearing thresholds or to targets for amplified speech generated from the threshold data. Fitting methods such as Desired Sensation Level (DSL) and National Acoustics Laboratories—Nonlinear (NAL-NL1, NAL-NL2) are based on amplifying speech to some desired level and yield targets for the amplified LTASS. Figure 39.9 shows the estimated ear canal SPL produced by a hearing aid amplifying real speech at 65 dB input SPL. The measured coupler SPL has been converted to ear canal SPL by adding the RECD so that it may be compared with the SPL threshold and real-ear NAL-NL1 speech targets. Noise reduction is set for maximum effect and feedback suppression, and adaptive directional features are enabled. In Figure 39.9, the hatched area is the amplified speech region bounded by the peaks of speech at the top and the valleys at the bottom. The circles are the hearing thresholds converted to SPL and the elongated + marks are the NAL-NL1 targets for the amplified LTASS. Most of the speech region is above threshold, and the LTASS of the amplified speech is close to the NAL-NL1 targets, indicating that this hearing aid is providing adequate amplification for speech at this level. The test may be repeated at other levels to ensure that expansion does not reduce gain for low-level speech signals and that compression keeps loud speech well below levels that might cause discomfort.

It must be emphasized that, when testing hearing aids with compression or any other adaptive processing features, the results obtained will be valid only for the test signal used (Henning and Bentler, 2005; Scollie and Seewald, 2002). Tests using speech-like test signals provide the most reliable indication that audibility goals are being met. If speech-like test signals are not available, the tests described in the following sections may provide estimates of electroacoustic parameters useful in deciding whether a hearing aid is performing as expected.

Tests Using Short-Duration Broadband Noise

If speech-like test signals are not available, tests that use short-duration broadband noise signals may be used to inspect the operation of the hearing aid. Since compression systems typically have millisecond attack times, whereas adaptive features usually have an onset time of several seconds, such signals will frequently show the operation of compression, free of the confounding effects of adaptive features. Figure 39.10 shows the gain obtained with a pink noise signal of 2 seconds in duration presented at (top to

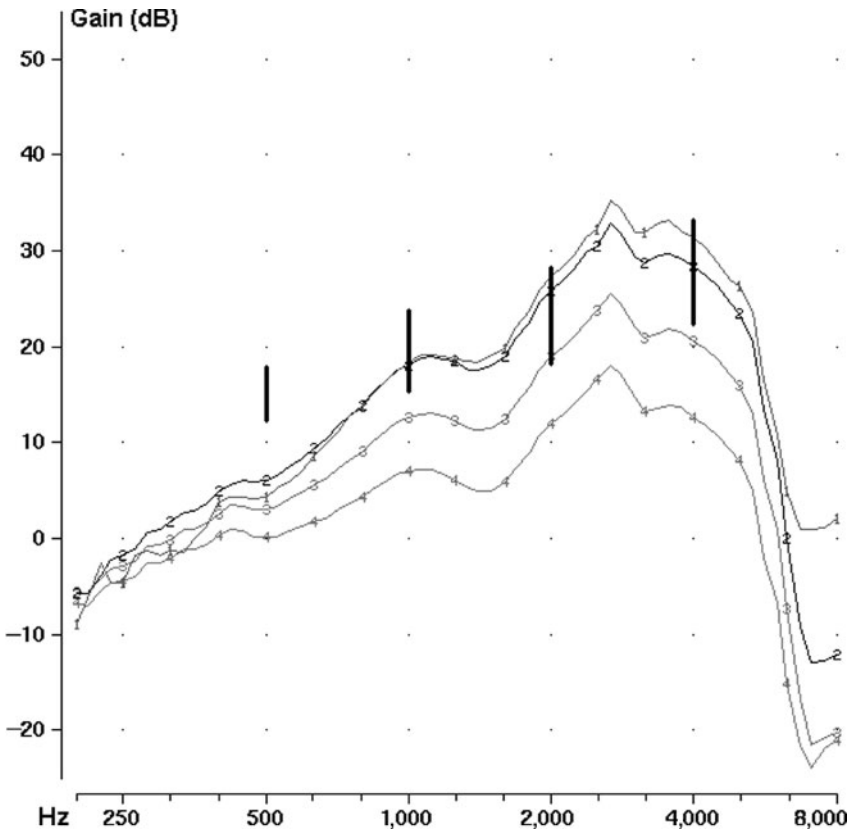


FIGURE 39.10 Using short-duration noise to check hearing aid operation. *Curves* are the coupler gain for pink noise presented for 2 seconds at [*top to bottom*] 45-, 60-, 75-, and 90-dB sound pressure level [SPL] for the same hearing aid and same settings used in Figure 39.9. The *vertical lines* at 500, 1,000, 2,000, and 4,000 Hz indicate one-third to one-half the hearing loss for which this hearing aid was programmed.

bottom) 45, 60, 75, and 90 dB SPL for the same hearing aid and same settings used in Figure 39.9.

- Observe that the gain curves are free from sharp peaks or abrupt dips, and they resemble curves for similar hearing aids.
- The gain for 45 dB input SPL is 4, 18, 27, and 31 dB at 500, 1,000, 2,000, and 4,000 Hz, respectively. The half-gain rule would indicate that this hearing aid might be appropriate for a hearing loss of about 35 at 1 kHz, 55 at 2 kHz, and 60 at 4 kHz.
- The 45- and 60-dB curves are closely spaced, indicating a compression threshold below 60 dB SPL.
- The 60-, 70-, and 90-dB curves are separated by about 7 to 8 dB over much of the useful frequency range. This indicates wide dynamic range compression (WDRC) with a compression ratio (input change/output change) of about 2.
- The 90-dB gain curve can be used to give a rough estimate of the OSPL90 of the hearing aid by adding 90 dB to the gain. This yields an estimated OSPL90 at 500 Hz, 1, 2, and 4 kHz of 90, 97, 102, and 103 dB, respectively, with a peak of about 108 dB at about 2.7 kHz.

These tests provide a good deal of information about this hearing aid and may be used to judge if it is functioning as intended. Refer back to Table 39.5 for a listing of possible causes and remediation of irregular response curves, low gain, and low maximum output.

Tests Using Short-Duration Tones

A test that can shed some light on hearing aid operation when it is not possible to change its programming is an input–output test. This test uses tones that are increased in 5-dB steps every few hundred milliseconds. The levels are maintained long enough to show the operation of compression but change frequently enough not to be attacked by adaptive features. Figure 39.11 shows input–output curves at various frequencies for the same hearing aid and same settings used in Figures 39.9 and 39.10. In each panel, the horizontal axis is the input SPL, and the vertical axis is the output SPL.

1. For a 90 dB input SPL, the output at 500, 1,000, 2,000, and 4,000 Hz is 90, 94, 99, and 99 dB SPL, respectively.
2. The gain is the difference between the output SPL and the input SPL. For a 45 dB input SPL, the gain at 500, 1,000, 2,000, and 4,000 Hz is 5, 18, 28, and 25 dB, respectively.
3. The compression ratio is obtained by dividing a change in input SPL (e.g., 10 dB) by the corresponding change in output SPL. A compression ratio of 1 (a diagonal line) is linear amplification, less than 1 indicates expansion, and greater than 1 indicates compression. The 250-Hz panel shows a compression ratio of about 0.5 (expansion) below 50 dB SPL and about 1 (linear) above about 60 dB SPL input. The 2,000-Hz panel shows a compression ratio of about 2 above 50 dB SPL.

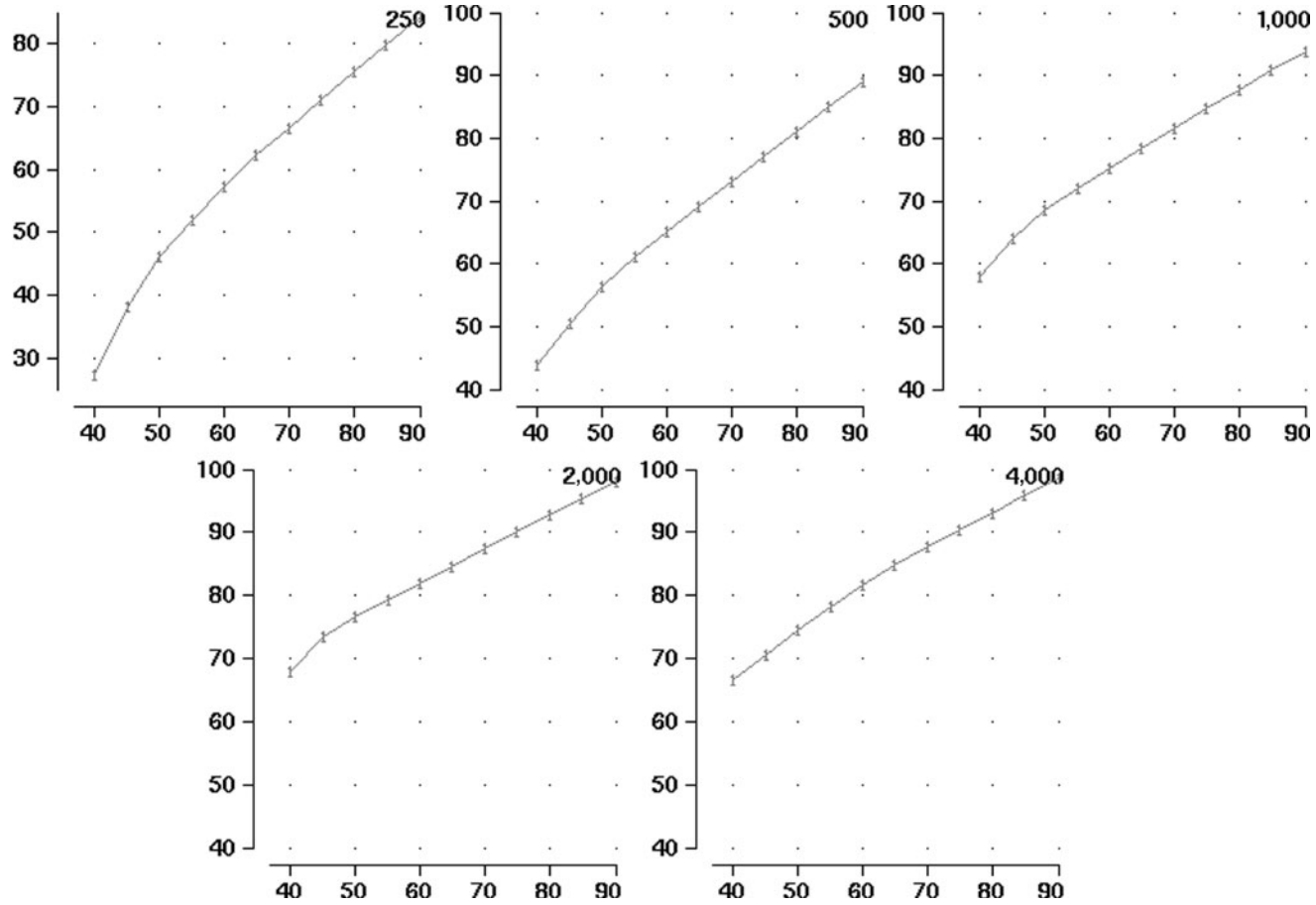


FIGURE 39.11 Using short-duration tones to generate input–output curves to check hearing aid operation. Curves are coupler sound pressure level (SPL; *vertical*) for varying input SPL (*horizontal*). The hearing aid and settings are the same as those for Figure 39.9.

4. The lowest input SPL at which the compression ratio exceeds 1 is often referred to as the "compression threshold" or "kneepoint." The compression threshold at 500, 1,000, 2,000, and 4,000 Hz is about 55, 50, 45, and ≤40 dB SPL, respectively.

High-level short-duration tone bursts are sometimes used in real-ear measurement equipment to determine the maximum output capabilities of a hearing aid without causing discomfort to the patient or risking further hearing damage. These tone bursts are typically long enough to activate compression limiting but short enough to avoid triggering adaptive features. If such a signal is available in a hearing aid analyzer, it may be employed to estimate the OSPL90 of the hearing aid without the need to disable noise reduction, feedback suppression, or adaptive directional systems. Figure 39.12 shows the 2-cc coupler SPL in response to a series of 90-dB tone bursts at one-third octave intervals for the same hearing aid and same settings used in Figures 39.9 to 38.11. The estimated OSPL90 at 500, 1,000, 2,000, and 4,000 Hz is 86, 93, 98, and 100 dB, respectively.

Short-duration tone tests may be expected to produce somewhat different estimates of gain, maximum output, compression threshold, and compression ratio than those obtained using brief broadband noise signals because the two signals are likely to be treated differently by compression systems. However, either is sufficiently accurate when the goal is simply to determine whether a hearing aid is functioning as expected and it is not possible or desired to change settings.

VERIFYING DIGITAL FEATURES USING HEARING AID ANALYZERS

The proliferation of digital technology in hearing aids has led to the introduction of features designed to address issues beyond amplification. These features may be significant factors in the selection and successful use of a hearing instrument, and their proper operation should not be taken for granted. These features are disabled for the standard tests of ANSI/ASA S3.22, but most hearing aid analyzers can be used to verify and document the functioning

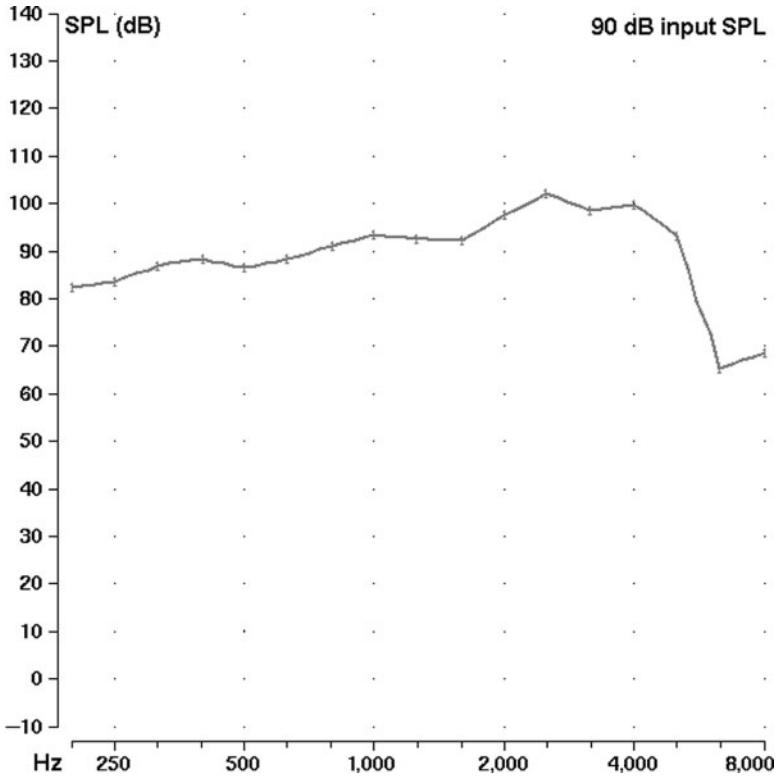


FIGURE 39.12 Using tone bursts to check maximum output with adaptive features enabled. Tone bursts were 128 ms in duration presented at 90-dB sound pressure level (SPL) at one-third octave frequencies. The hearing aid and settings are the same as those for Figure 39.9.

of noise reduction, adaptive directional microphones, and feedback suppression features. The following tests have been found to work on many current hearing aids but may not be applicable to all hearing aids and may not work with future hearing aids.

Functional Check of Adaptive Noise Reduction

Most noise reduction algorithms operate by reducing gain in frequency bands in which the signals do not exhibit the modulation characteristics of speech (Chung, 2004a). A steady broadband noise signal may be used to verify the functioning of such systems. In Figure 39.13, the upper curve (1) shows the gain for a steady 60-dB SPL pink noise signal when it is first applied, whereas the lower curve (2) shows the gain when it has stabilized 10 seconds later. The noise reduction algorithm has reduced the gain by about 12 dB at 1 kHz. This test also provides some idea of the speed with which the hearing aid responds to the onset of noise. To avoid the confounding effects of adaptive directional response, it may be necessary to set the aid to omnidirectional mode before performing this test.

The noise reduction algorithm should not reduce the gain for a speech signal. This can be verified if the hearing aid analyzer provides speech-like test signals. That is, there should be no change in output for these signals when the noise reduction feature is toggled between minimum (or off) and maximum reduction.

Functional Check of Directional Hearing Aids

Accurately testing directional hearing aids requires a large test space and specialized equipment (ANSI S3.35, 2010). However, it is possible to use a hearing aid analyzer to determine if the directional feature is functioning as expected. Note, however, that such tests performed in a small test chamber will not produce results that can be compared with published specifications. They will generally show less directionality, especially at lower frequencies.

It is usually recommended that the hearing aid be set for linear operation with a fixed directional pattern and to switch off any adaptive noise reduction and feedback suppression algorithms before attempting to measure directional performance. This is because gain or output for sounds from different directions is usually determined by making measurements from different directions at different times. Compression or adaptive features may change the gain or output of the hearing aid between successive measurements, resulting in erroneous measures of directional performance.

To test directional function, the hearing aid is set as indicated earlier, and a gain or output curve for a 50- to 65-dB broadband signal (e.g., pink noise) is obtained, with the hearing aid oriented so that its direction of maximum sensitivity is toward the loudspeaker (within 45°). Next, the hearing aid is oriented so that the direction of maximum sensitivity is away from the loudspeaker, and the test

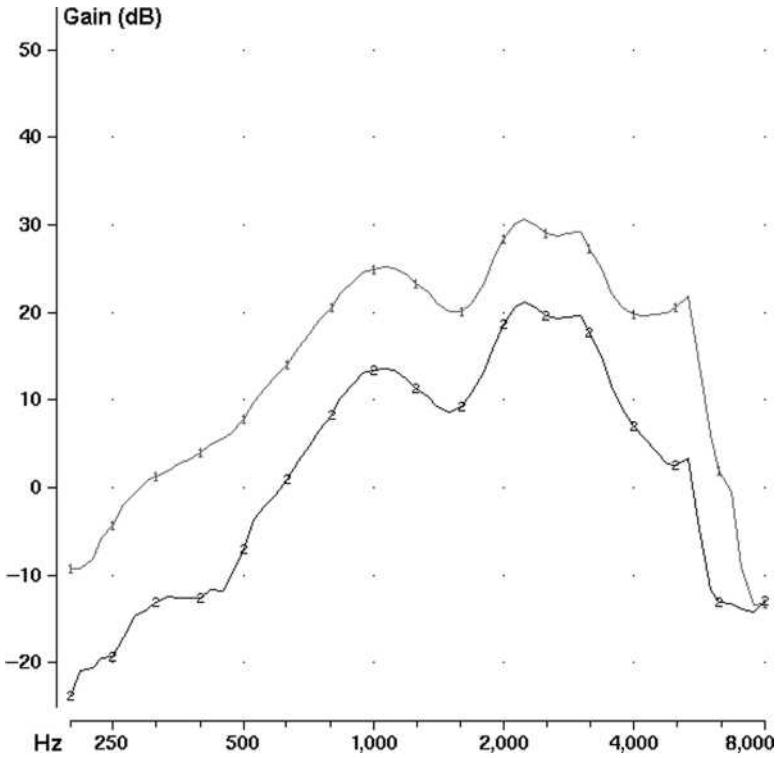


FIGURE 39.13 Functional check of adaptive noise reduction feature. The upper curve (*1*) shows the gain for a steady 60-dB sound pressure level (SPL) pink noise signal when it is first applied, whereas the lower curve (*2*) shows the gain when it has stabilized 10 seconds later.

is repeated. The second curve should lie below the first, at least in the mid-frequencies, with the separation between the curves being an indication that the directional feature is functioning. This is a functional test only, and the curve separation obtained in this way is not expected to correlate with standard measures of directional performance. As an additional check, the tests should be repeated with the hearing aid in omnidirectional mode. In this case, the two curves should be nearly coincident.

Some hearing aid analyzers provide special signals for testing directional function in a test chamber that has two speakers (Smriga, 2004). In this case, the test can be performed without setting the hearing aid for linear operation or switching off adaptive algorithms. This scheme delivers two separate signals, each containing over 500 different tones, from two directions simultaneously. Digital analysis separates the coupler signal into two frequency response curves, one for each direction, and the separation between these curves shows the functioning of the directional microphone(s). The operation of compression or noise reduction impacts both response curves, but their separation remains unaffected. Such a test may also reveal the signal level at which change is initiated and the time required for change to occur. The end result of such a test is shown in Figure 39.14. In each panel, the bold curve is the response from a source in the front hemisphere, whereas the lighter curve is the response from a source in the rear hemisphere obtained using two simultaneous pink noise signals. Panel A shows an omnidirectional response immediately after signal presentation, whereas panel B shows the directional response after the signals have been presented for 20 seconds. This test may be modified by adding speech to the sound source in the front hemisphere to test hearing aids that change their directional response pattern as a function of signal-to-noise ratio rather than noise level.

A hearing aid may fail to show any appreciable directional function using these tests for the following reasons:

1. The hearing aid has been oriented so that gain is about the same for signals from both directions tested. Some directional patterns have rear or side lobes with gain comparable to the front lobe at some angles. This can be checked by changing the orientation used for the test.
2. The hearing aid is not set for directional operation. This may result from failure of a programming or directional switch, failure to enable the feature in the programming software, an automated decision within the programming software, or a failure of hardware or software.
3. Blockage of the microphone ports on the hearing aid.
4. Microphone drift in two-microphone directional systems. These systems rely on well-matched microphones for their directional performance. Microphone sensitivity changes with temperature, humidity, and time, all of which can degrade directional performance. Some hearing aids self-correct for these changes, but those that do not can cease to be directional hearing aids.
5. Miswired or improperly assembled microphones in the hearing aid.

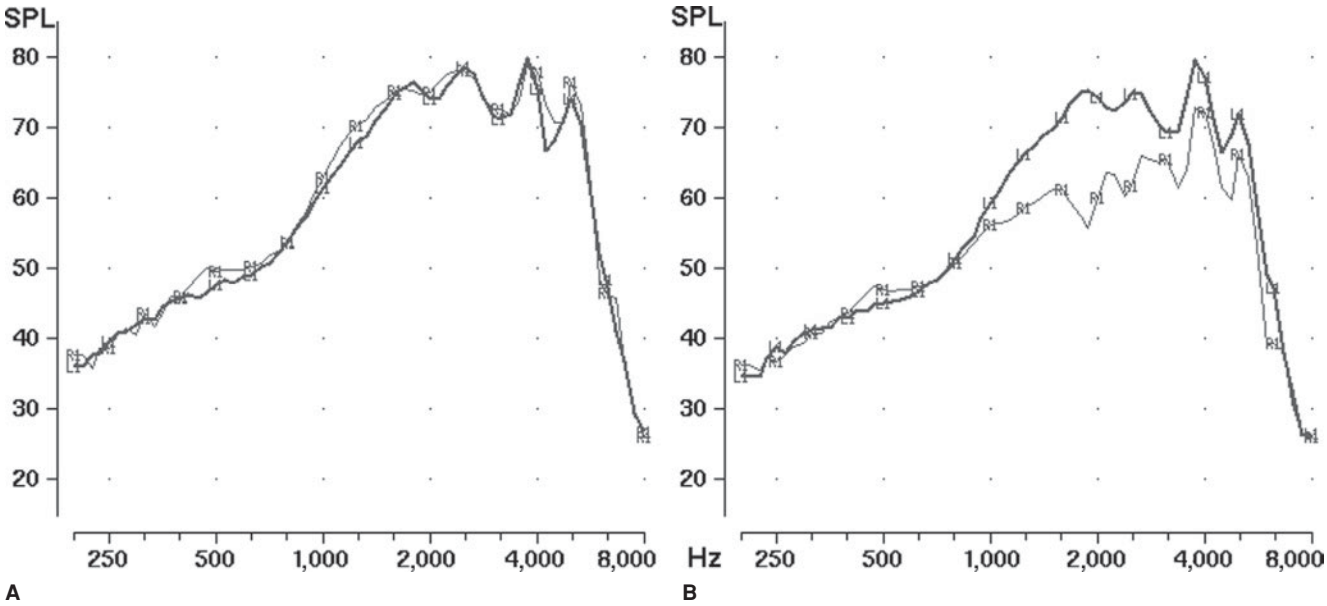


FIGURE 39.14 Functional check of adaptive directional feature. In each panel, the *bold curve* is the response from a source in the front hemisphere, whereas the *lighter curve* is the response from a source in the rear hemisphere obtained using two simultaneous pink noise signals. [**A**] An omnidirectional response immediately after signal presentation. [**B**] The directional response after the signals have been presented for 20 seconds.

Functional Check of Feedback Suppression Systems

Feedback occurs when the gain through the hearing aid (the forward path) plus the attenuation from the ear canal back to the hearing aid microphone (the feedback path) is greater than 0 dB, with a phase shift that is an integer multiple of 360°. The sum of the gain through the forward path and the attenuation (negative gain) through the feedback path is termed the open loop gain.

Feedback suppression systems operate by reducing the gain of the forward path at frequencies where the open loop gain would otherwise exceed 0 dB. This may be accomplished either through filters in the forward path or by subtracting from the microphone signal an estimate of the signal in the feedback path (called phase cancellation). In some systems, no action is taken to change the forward path gain until feedback, in the form of a persistent tonal signal, is detected. Freed and Soli (2005) have classed these as "detectors."

Other systems can measure the attenuation of the feedback path during the fitting process by internally applying a known electrical test signal to the hearing aid receiver and measuring the resulting signal at the hearing aid microphone. Freed and Soli (2005) have classed these as "initialized" systems. In the case of forward path filters, this information is used to reduce (or limit) the gain in frequency regions where the open loop gain would otherwise exceed 0 dB. In the case of phase cancellers, the same test signal being applied to the hearing aid receiver is also applied to a digital filter, which is automatically adjusted until its output approximates the signal being measured by the hearing aid microphone. The digital filter then effectively becomes a simulation of the feedback path. In operation, the output of this filter is subtracted from the microphone signal, canceling that portion of the microphone signal that is due to the feedback path. To avoid the initialization step and to accommodate changing feedback paths, some phase cancellers employ an adaptive digital filter that is continuously adjusted to minimize the difference between its output and the microphone signal, using ambient sound rather than an internally generated signal.

A number of laboratory measures have been proposed to characterize feedback suppression systems (Freed and Soli, 2005), but the test described here is intended only to determine whether the suppression system is working. It is based on a test described previously by Smriga (2004) and has been found to work on current hearing aids using different types of suppression systems. The difficulty in performing a test of a feedback suppression system in the test chamber of a hearing aid analyzer is getting feedback to occur on demand. Smriga (2004) described a way to do this in analyzers that provide a headphone for listening to the output of the hearing aid in the coupler. In the steps that follow, this method is used to induce feedback in the presence of a speech-like test signal. A speech-like signal is used to avoid engaging adaptive directional or noise reduction features and to ensure that changes in forward path gain caused by compression and/or expansion are representative of those achieved in actual use. Variations of this test are possible using a low-level noise signal or no input signal at all, but

FIGURE 39.15 Setup for inducing controlled feedback in the test chamber. The monitor headphone (*1*), which is normally used to listen to the output of the hearing aid in the coupler, is placed near the hearing aid. The monitor gain is adjusted to induce feedback.

it may be necessary to disable adaptive features and expansion, and the results may not represent real-use experience.

1. Program the hearing aid for normal use. Place the hearing aid in the test chamber as if for a standard hearing aid test.
2. Connect the monitor headphone, normally used for listening to the output of the hearing aid, and place it in the test chamber near the hearing aid (Figure 39.15).
3. Present a speech-like signal at 60 dB SPL and display the coupler SPL.
4. Adjust the headphone volume control until one or more peaks appear in the response curve (Figure 39.16).
5. Enable the feedback suppression and observe that the peaks are removed from the response curves. Observe also that there has been no significant loss of output at the frequencies of the peaks. This result may not be observed for some hearing aids that require an initialization step before enabling feedback suppression. In this case, perform initialization after step 4 but with the sound source turned off. The sound source should be switched on before proceeding to step 5.

PROPER FIT

In a survey of hearing aid owners in the United States, the third most common reason for not using hearing aids was fit and comfort (Kochkin, 2000). Clients may complain that the hearing aids "are too big," "fall out of my ears," "hurt my ears," or "are uncomfortable." An accurate impression and proper fit in the ear are critical for effective hearing aid use.

Proper fit is often more critical and also more difficult to achieve in those with severe or profound hearing losses and for those fit with completely-in-the-canal hearing aids. In some cases, two or three remakes of the hearing aid shell or mold may be required before a good fit is achieved. Depending on the material used and the degree of hearing loss, new molds may be required every 3 to 6 months, whereas others may not need to be remade for a year or more.

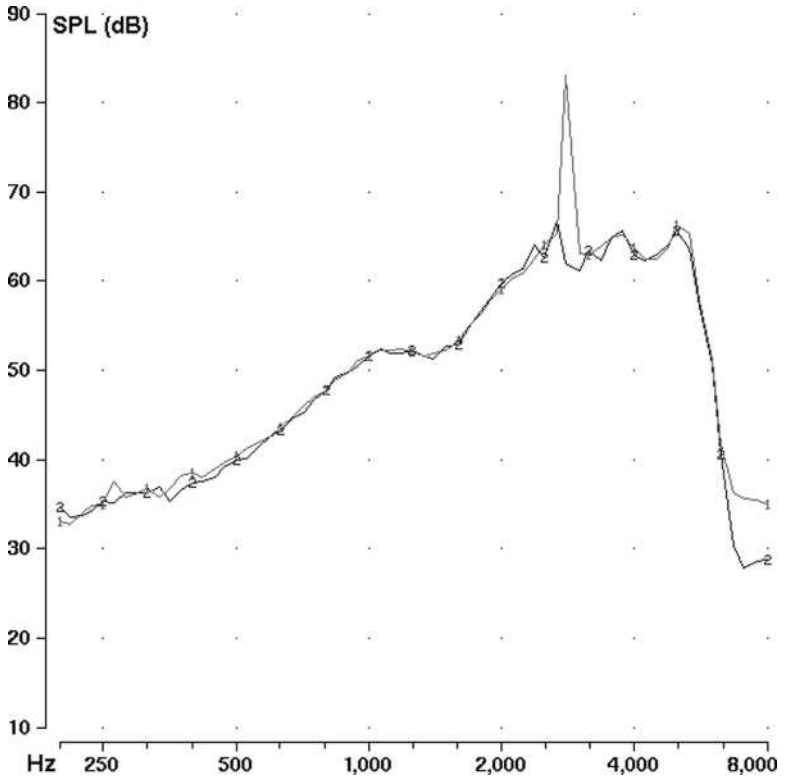


FIGURE 39.16 Functional check of adaptive feedback suppression. Shown is the long-term average speech spectrum for speech at 60-dB sound pressure level (SPL) obtained with the setup of Figure 39.15. *Curve 1* was obtained by adjusting the gain of the monitor headphones, with feedback suppression disabled, until feedback occurred. *Curve 2* was obtained after enabling feedback suppression without changing the gain.

Pain

Hearing aids and earmolds should never cause pain or aching even after a full day of use. Clients should be cautioned to remove the hearing aid if there is pain and return as soon as possible to ensure a proper fit. Whereas new users are more likely to give up when there is pain, some long-term users cannot manage without their hearing aids and will persist in wearing a painful hearing aid until there is a visible red, swollen sore that may take weeks to heal.

If proper placement in the ear is observed, it should be determined whether the pain experienced happens immediately or after a few hours of use. The ear needs to be inspected. Is a sore or red spot present in the helix, concha, or canal? Is the pain in one particular spot (indicating a possible pressure sore) or all over (indicating that the aid may be slightly too large and needs to be reduced in general by a small amount)? If the client experiences pain almost immediately or if the pain is felt in a widespread area, a complete remake of the shell or mold from a new impression is indicated. However, if the pain occurs only after a few hours or only in one particular spot, then grinding and buffing the shell or mold is worth doing. Note that, with open-fit behind-the-ear hearing aids, pain may be caused by inappropriate tubing size or dome size.

If a sore is present, it is important to advise the client to leave the hearing aid out until all soreness, redness, or swelling is gone. Pressure sores can take days or weeks to heal completely and will not heal unless the hearing aid is not worn at all during the recovery period.

Excessive Hair in the Ear

Excessive hair growth in the concha or ear canal may cause chronic fit problems. The client may choose to keep the ear hair trimmed or switch to a behind-the-ear or open-fit model, if appropriate.

Change in Client's Weight

A change in the client's weight can affect the fit of a hearing aid. The shape and size of the ear can change with a weight increase or decrease of 10 pounds or more. In the case of weight loss, the hearing aid may become too loose and fall out of the ear or cause feedback. With significant weight gain, the hearing aid may become too tight and cause pain or sores. Reshelling for in-the-ear hearing aids or making a new mold in the case of behind-the-ear hearing aids usually alleviates the problem.

Effect of Climate

Hearing aids made from an impression in one season or climate may cause problems in another. To determine whether this may be a problem for a particular client, ask if rings and wrist watches are tighter in the summer and looser in the winter. This can be an issue for clients traveling to different climates. For behind-the-ear hearing aids, some clients may require a winter mold and a summer mold. For in-the-ear hearing aids, a shell made from an impression taken in the winter may feel tight in the summer but may avoid feedback or a loose fit in the winter. Table 39.6 lists various seasonal and climate problems.

TABLE 39.6

Seasonal and Climate Problems

Summer issues

- High humidity can cause condensation in in-the-ear hearing aids, resulting in a short circuit
- High humidity can cause condensation in the tubing of behind-the-ear hearing aids, resulting in a blockage
- Client's ear tissues may swell (fluid retention), making fit snug or painful
- High temperatures (>122°F or 50°C) (common in cars on a hot day) may impair batteries

Winter issues

- Dry air due to heating may shorten battery life
- Dry air due to heating may increase static electricity, which could damage the hearing aid circuit
- Cold or dry air may shrink client ear tissues, making the fit too loose and/or causing feedback
- Low temperatures (< –14°F or –10°C) may impair batteries

Grinding and Buffing Shells and Molds

A hearing aid should be reshelled if the shell is quite thin or if there are cracks, holes, or signs of extensive grinding or previous application of coatings. However, sending a hearing aid, or mold, out for a remake means that the client will be without the hearing aid for several days or more. If a remake of the shell or mold is not required, grinding and buffing can be done in the office while the client waits. A Dremel-type tool with a flex-shaft is ideal for this kind of fine, delicate grinding and buffing. If one or two adjustments do not resolve the problem, it is advisable to take a new impression and have the shell or mold remade.

There is a wide variety of materials available for earmolds such as acrylic, silicone, and polyvinyl chloride (PVC). The hardness (or Shore rating) of a material will determine the appropriate bit type and speed for grinding and buffing. Shore ratings and material descriptions are available from companies that make molds. For hard materials such as acrylic, a small bit and a medium speed are best. Hard

materials are more difficult to smooth and need to be buffed well. For soft materials such as PVC, a medium bit and a higher speed work well and reduce the amount of shredding. Super-soft materials such as silicone require a larger bit and higher speed, and the mold must be braced to reduce vibration and increase the effectiveness of the grinding; softer materials, however, do not require buffing. If a large amount must be removed, soft materials such as PVC and silicone can be cut or trimmed before grinding, but care must be taken to ensure that there are no sharp edges.

OCCLUSION AND AMPCLUSION

"My own voice is too loud," "It sounds like I am shouting," and "Everything is hollow and echoing" are complaints often heard from clients trying new hearing aids. Sometimes the subjective discomfort is so pronounced that the client refuses to wear the hearing aids.

Shell-related occlusion is a common side effect of placing an object into the ear canal, particularly with binaural fittings. When talking, the voice projects primarily through the mouth, but some of the energy normally escapes through the ears. With the ears "plugged," however, a client's voice may resonate more within the head.

Ampclusion occurs when certain frequencies are over- or underamplified by the hearing aids, causing a hollowness or echoing effect. Some of the common causes of shell-related occlusion and ampclusion include

- Too little venting in the hearing aid
- Inappropriate length of hearing aid in the canal
- Physical blockage of canals (e.g., wax, blocked vent)
- Inappropriate gain for hearing loss in low frequencies ("ampclusion")

Adapting to Initial Shell-Related Occlusion

Some shell-related occlusion effect should be expected when trying hearing aids for the first time. Clients should be counseled that their own voice and sounds, in general, will sound different at first. Mild cases of shell-related occlusion can disappear within a few minutes, although a few days of regular use are often needed as the client adapts to the new sound.

Persistent Occlusion: Shell-Related or Ampclusion?

When occlusion persists, the underlying cause must be determined. If the sense of occlusion is less when the hearing aids are turned off while still in the ear (by opening the battery door), then the problem is ampclusion (over- or underamplification provided by the hearing aids). If the occlusion effect is worse with the hearing aid off, then the problem is either underamplification (ampclusion) or shell-related occlusion. If the occlusion is unaffected by the hearing aids being on or off, then shell-related occlusion is the problem.

One needs to check whether the occlusion is present with just one hearing aid or with two. If the occlusion is primarily present in one ear, adjustments may be needed only in that ear. If the occlusion is observed only when both hearing aids are in place but not with either one alone, then the occlusion may be due to an increase in the resonance of sound within the head or underventing, both of which result in overamplification of low frequencies. Although the origin is shell related, if shell-related solutions do not solve the problem, then it should be treated as ampclusion because of overamplification of the low frequencies.

Nonacoustic Occlusion due to Cranial Nerve Stimulation

The trigeminal nerve (cranial nerve [CN] V), vagus nerve (CN X), facial nerve (CN VII), glossopharyngeal nerve (CN IX), and many intermediary branches of nerves course through and around the external auditory canal, the tympanic plexus at the eardrum, and around the middle ear and Eustachian tube. Stimulation of any of these can trigger reflexes that may occasionally cause nonacoustic occlusion when fitting hearing aids. The vagus and trigeminal reflexes in particular may contribute to occlusion in some people; however, there is currently no easy way to determine whether this may be an underlying cause. Until a clinically feasible method for predicting and testing for cranial nerve stimulation is developed, this should be treated as a shell-related problem.

Resolving Shell-Related Occlusion

During the assessment and prescribing process, the optimal venting for the hearing loss can be determined (Table 39.7)

TABLE 39.7

Optimal Venting to Minimize Occlusion

Hearing Loss at 500 Hz	Optimal Vent Size
<20 dB HL	Open fit (>3 mm)
20–30 dB HL	2–3 mm
30–40 dB HL	1.5–2 mm
>50 dB HL	<1 mm
With CICs	Reverse horn vent

Source: Adapted from Kuk F. (2005) Developing a hierarchy to manage the "own voice" problem. Session at American Academy of Audiology Conference, Washington, DC, April 2005.
HL, hearing level; CIC, completely-in-the-canal.

to minimize shell-related occlusion. Shell-related occlusion may result from inappropriate venting in the hearing aid, too long a canal in the hearing aid, physical blockage of the hearing aid, or pressure on nerves in the ear canal, which, in turn, requires physical alteration of the hearing aid shell or earmold. An open-fit behind-the-ear hearing aid may solve occlusion for clients whose hearing loss is in the appropriate range.

Very deep insertion in the canal may also significantly reduce the occlusion effect, but the effective depth is 4 mm beyond the second bend, which is usually physically uncomfortable for the client (Pirzanski, 1998). The difficulty in inserting and removing these hearing aids, combined with physical discomfort, limits the clinical usefulness of very deep fits in the canal for reducing occlusion.

Resolving Ampclusion

Ampclusion may be resolved by adjusting low-frequency gain according to a client's perception of his/her own voice. If the problem is worse when the client speaks louder, overamplification is the issue, and low frequencies should be reduced. If the problem is worse when the client speaks softer, underamplification is the issue, and low frequencies should be increased.

FEEDBACK

Although many clients will persist in wearing a hearing aid that causes pain, most will refuse to wear one that squeals or buzzes. Feedback may occur with every head or jaw movement or may be occasional such as when putting on a hat or hugging family or friends.

Clients should be advised that there are times when feedback is expected and normal, such as when inserting or removing a hearing aid with both the power and volume on, or when the microphone is covered, such as by a hand, hat, or telephone. Some clients use feedback to confirm that the hearing aid is working and that the battery is good and to judge when the hearing aid has been correctly inserted based on when the feedback stops.

Understanding Feedback

A hearing aid is an amplifying system with a microphone, an amplifier, and a speaker (receiver). If the sound leaves the receiver and re-enters the microphone, it becomes reamplified and creates an oscillation. Low levels of acoustic feedback may not cause actual squealing but can disrupt frequency response and reduce speech clarity by creating an echo sensation. Feedback can progress into self-sustaining oscillation, which is the well-known, embarrassing whistle. When feedback drives the hearing aid into saturation, it generates multiple intense oscillations that are clearly audible and usually extremely uncomfortable for the wearer.

Although both high- and low-frequency sounds can easily escape from the hearing aid through venting or poor fit, the shorter wavelengths of the high-frequency sounds allow them to more easily reflect off surfaces such as the pinna and concha, increasing the likelihood of re-entering the hearing aid at the microphone. High-frequency sounds exiting the receiver are also more likely to be in phase with the incoming sounds at the microphone.

In hearing aids with greater high-frequency gain, the escaping sound is more likely to be louder than the initiating sound when it finds its way back to the microphone. In addition, higher gain hearing aids may produce oscillations of multiple frequencies that result in a faster onset of acoustic feedback. Because peaks in the real-ear frequency response often occur at high frequencies and such peaks are often associated with rapid phase changes, it is clear how these factors interact to produce the oscillation of feedback.

Because high-frequency hearing loss is most common, clients may not hear the acoustic feedback even though it is audible to others around them.

Is It Really Feedback?

In some cases, it may be difficult to determine whether a problem experienced by a client is feedback. Clients will use a bewildering variety of terms and phrases to describe the problem, and it is not often clear what the actual issue is. Clients may describe a squealing and whistling that appear to indicate the presence of feedback or a buzz or other odd sound that could be an issue with the telephone coil in the hearing aid or the presence of a real sound in their environment. The various possibilities must be investigated to determine the underlying cause. One way to determine if feedback is the problem is to induce feedback by covering the hearing aid while it is in the client's ear. If the client reports that this is the sound he/she has heard, then feedback has been confirmed.

Patterns may help identify the source of feedback-related issues. If a client hears the disturbing sound in question only when he/she smiles or bends over, then feedback is likely the issue. However, if the sound in question is only heard at certain times or in certain locations, then the client may be hearing an unfamiliar sound in the environment. Main sources of feedback include

- Improper fit in the ear
- Effect of impression materials
- Anatomy of the ear
- Improper placement in the ear
- Cracked or loose tubing in earmold
- Orientation of sound bore results in aiming sounds at the canal wall
- Presence of wax in the ear canal
- Reduced tympanic membrane compliance

- Large venting with high gain
- Low kneepoint (WDRC circuit)
- Inappropriate style of hearing aid for the degree of hearing loss
- Distance between the microphone and the receiver

Improper Fit

It is important to ensure the best physical fit possible in the ear before resorting to the application of software feedback managers. The more severe the hearing loss, the more amplification is usually prescribed and the more critical the fit will be in preventing feedback. With a new hearing aid or mold, it is worth the investment of time, energy, and resources in the first few months to achieve optimal fit and minimize future problems.

Feedback can be a major issue with older hearing aids, as well. Over time, the ear often changes slightly, and there may also be some shrinkage in the hearing aid shell or mold, depending on the properties of the material used and the chemistry of the ear canal skin. Materials such as PVC can shrink significantly over 2 or 3 years. The fit of a hearing aid tends to become less precise after 2 or 3 years, usually getting looser in the ear over time. The resulting gaps between the hearing aid and the ear canal can allow sound to leak out of the ear and cause feedback. In some cases, the hearing aid becomes so loose that the shifting in the ear can easily be seen with normal jaw movement. One easy test is to apply a soft expanding material, such as Comply Soft Wraps, around the canal portion of the hearing aid. If the feedback is eliminated by the presence of the Comply Soft Wrap, then poor fit is likely the primary cause of the feedback, and a recoating or reshelling of the hearing aid often corrects the problem. If the hearing aid is 5 years old or older, it may be preferable to purchase a new hearing aid since repair and reshelling costs are higher for hearing aids over 5 years of age. With behind-the-ear hearing aids, a new earmold should be made if the fit is loose in the ear.

A significant change in the client's weight can also affect the fit and cause feedback. See earlier "Proper Fit" section.

Effect of Impression Materials

The type of impression material used for taking impressions can affect the ultimate fit in the ear. Standard-viscosity silicone tends to give a more accurate impression of the ear, resulting in a better fit and less feedback. When using injector guns for impression taking, keep in mind that many guns do not have enough power to push standard-viscosity silicone; therefore, a standard syringe may be required. Low-viscosity materials reduce stretching of the ear tissue and can make a more comfortable though less accurate fit, increasing the chance of feedback in the hearing aid. If feedback is a problem for a hearing aid made from a low-viscosity impression material, a reshelling from a medium- or high-viscosity material impression is worth trying.

Open-Jaw versus Closed-Jaw Impressions

Hearing aids and molds made from closed-jaw impressions or impressions taken while the client is chewing may lack a proper acoustic seal and result in retention and feedback problems. Hearing aids and molds made from open-jaw impressions tend to have better anatomic definition of the ear, a more secure fit, less feedback, and better comfort (Chasin et al., 1997). Open-jaw impressions should be done one ear at a time, with a bite block as far back as possible on the same side and the longest axis kept vertical to maximize the openness of the jaw.

Anatomy of the Ear

Sometimes the cause of intermittent feedback can be traced to jaw movement. Look for patterns such as feedback that occurs after a meal or only later in the day. In some clients, the temporomandibular joint (TMJ) may significantly amplify the effect of jaw movement while talking and eating, resulting in the hearing aid shifting in the ear (Oliviera et al., 2005). Sometimes, the cumulative effect of chewing over several meals is needed before the jaw movement displaces the hearing aid enough to create feedback. In this case, the feedback tends to be experienced only later in the day. This effect is often greater in one ear than the other. A very straight ear canal tends to result in poor retention of the hearing aid as well, and jaw movement may shift the hearing aid out of the ear canal in some cases.

There is currently no way to predict whether the anatomy of a client's ear will have a negative effect on the fit of a hearing aid. For experienced users, ask if feedback has ever been a problem with previous hearing aids. If the problem is chronic ("I've always had problems with hearing aids in my right ear"), then the anatomy of the client's ear may be the root cause, and an open-jaw impression is recommended. For some clients, adding a canal or helix lock to in-the-canal or completely-in-the-canal hearing aids may solve the problem by improved retention.

Improper Placement in the Ear

Improper placement in the ear can cause feedback. See "Performing a Visual Inspection" section earlier in this chapter.

Cracked or Loose Tubing in Earmold

Cracked or loose tubing in earmolds allows sound to escape freely from the ear causing feedback. See "Inspecting the Hearing Aid and Component Parts" section earlier in this chapter.

Orientation of the Sound Bore

The orientation of the sound bore of the mold or hearing aid shell is also important. If the sound bore is pointed toward the wall of the ear canal rather than toward the eardrum, some of the sound may be reflected back toward the receiver, increasing the chance of feedback.

Presence of Wax in the Canal

The presence of a significant amount of wax in the ear can cause feedback. The sound from the receiver tube of the hearing aid can be deflected by the wax back toward the hearing aid. The sound can exit the ear via the vent and cause feedback. This can be exacerbated by an imperfect fit in the ear canal or improper placement of the hearing aid in the ear. In many cases, removal of the wax reduces or eliminates the feedback. If there is a significant amount of wax present, it should be removed before other causes of feedback are investigated and before other solutions (such as reshelling or running a software feedback manager) are attempted.

Open-fit behind-the-ear hearing aids are particularly susceptible to blockage by wax because of the small diameter of the tubing. Manufacturers provide a tool to keep the tubing clear.

Venting with High Gain

If acoustic feedback is a problem, a vent may need to be reduced or closed completely; however, this may result in an increase in occlusion and humidity in the ear canals.

One way to retain some venting in a high-gain hearing aid is to choose a hearing aid with a smooth real-ear response or enough channels to achieve a smooth response. Feedback is less likely to occur if the peaks of the frequency response are minimized.

The large venting of open-fit behind-the-ear hearing aids allows high frequencies to escape easily, increasing the risk of feedback. More occlusive ear pieces are available from some companies for use with open-fit tubing that restrict the escape of high frequencies from the ear canal. Software feedback management can also be used to reduce feedback (see "Software Feedback Reduction Managers" section later in this chapter).

Feedback in Wide Dynamic Range Compression Circuits

Hearing aids using WDRC or other compression schemes that provide more gain for low-level input signals and less for higher level signals are more prone to feedback than linear hearing aids (Olsen et al., 2001). This is especially true when the input levels to the hearing aid are low. A hearing aid may be in feedback for quiet situations because of the relatively high gain, but the feedback may cease in noisier environments because of the lower amounts of gain (Chung, 2004b). Feedback in quieter locations can be partially reduced by increasing the threshold kneepoint, by increasing the amount of expansion, or by a binaural fitting (through loudness summation, thus reducing the amount of amplification required).

Inappropriate Hearing Aid Style for the Degree of Hearing Loss

Sometimes a person with a severe or profound hearing loss in the high frequencies is fit with an in-the-canal or completely-in-the-canal hearing aid. This can be an issue for persons with very straight ear canals or poor retention of the hearing aid. Feedback can be reduced or eliminated by increasing the distance between the microphone and the receiver by increasing the canal length or switching from an in-the-ear model to a behind-the-ear model. Moving to a larger hearing instrument may increase the surface contact in the ear, thereby providing a more efficient seal. A behind-the-ear model also allows for use of softer materials, such as PVC or silicone, that can reduce feedback by providing a more snug fit in the ear.

Feedback can occur when open-fit behind-the-ear hearing aids are used with a severe hearing loss. A hybrid open fit with the slim tube attached to a custom-made earpiece may be effective in this case. The increasing effectiveness of software feedback managers may make the open-fit behind-the-ear hearing aid more useable for severe hearing losses (see earlier "Venting with High Gain" section; see also "Software Feedback Reduction Managers" section later in this chapter).

Distance between the Microphone and the Receiver

If feedback persists with a behind-the-ear model, it is possible to route the signal contralaterally between two behind-the-ear hearing aids. In this configuration, the sound from the right hearing aid is routed to the left ear and vice versa. This can be accomplished by either a wire or a wireless (e.g., Wi-Fi) routing. The distance between the microphone and the receiver will be significantly increased and often eliminates feedback completely.

Body aids work on the same principle of maximizing the distance between the microphone and the receiver, with the microphone typically being worn at chest level. However, body aids are being phased out and are difficult to purchase.

The use of a frequency modulation (FM) system will also reduce or eliminate feedback, again because of the increase in distance between the microphone and the receiver.

Malfunctioning Battery

Batteries occasionally cause unusual problems with hearing aids, including feedback. Some hearing aid circuits will experience more feedback as the battery is about to expire. If no obvious cause for the feedback can be found, try a new battery from a different batch and/or brand.

Improper Mounting of Microphone and Receiver

In the course of normal operation, receivers generate a small amount of vibration. Any vibration transferred to the shell of the hearing aid may generate a small amount of sound that is sensed by the microphone, potentially causing feedback. If proper fit and venting have been ensured, persistent feedback may be due to the microphone or receiver not being mounted optimally to minimize vibration. In this case, the hearing aid would have to be returned to the manufacturer for verification.

Software Feedback Reduction Managers

Software feedback reduction managers should be the last resort for addressing persistent feedback problems. When running the feedback manager in a manufacturer's software, one needs to be aware of what the program is doing. There are many different methods employed within software feedback managers. Feedback management programs typically work by gain reduction, notch filtering, or phase shifting. It is well worth some investigation to determine the specifications of any software feedback manager before using it.

Gain reduction can eliminate feedback by reducing the gain in the high frequencies, but such a broad range of reduction can also decrease the ability to clearly understand speech, particularly for unfamiliar voices, for foreign accents, or in competing noise. For this reason, the gain reduction method of feedback reduction should be a last resort.

Notch filters work by gain reduction, but the frequencies reduced are limited to those known to cause feedback. This can be implemented at the hearing aid fitting, and there are some models of hearing aids that attempt to "seek out and destroy" the offending feedback signal. To date, this adaptive approach has met with limited success. A very narrow notch filter can reduce feedback without greatly altering the final output from the hearing aid. However, if the feedback occurs at multiple frequencies, several notch filters or a much wider notch filter may be required, and the resulting gain may be significantly lower than that required for the level of hearing loss.

Phase shifting or phase inversion, also known as "feedback cancellation," eliminates or reduces acoustic feedback without significantly reducing the prescribed gain of the hearing aid across the frequencies. This preserves the ability to hear speech and other sounds as clearly as possible. In phase shifting, the phase of any detected feedback is mirrored in a 180° phase shift, resulting in destructive interference. The goal is to achieve an exact 180° shift within an extremely short time to achieve near or complete cancellation of the feedback signal.

Feedback can build to saturation within 200 ms, so the ideal feedback-canceling system must be able to negate audible oscillations in real time. Some digital hearing aids incorporate high-speed real-time feedback cancellation systems that claim to completely stop feedback. As of yet, however, there are no independent, large-scale studies to substantiate these claims.

EAR WAX

An existing hearing loss can be exacerbated by the presence of ear wax. Ear wax can significantly reduce the transmission of sound by blocking the ear canal, blocking the sound from exiting the hearing aid, or causing damage to internal components of the hearing aid. Hearing aid manufacturers report that the majority of all hearing aid repairs are due to damage from ear wax.

What Is Ear Wax?

Ear wax is a normal product of the ear. Ear wax is primarily composed of keratin (derived from dead skin) with a mixture of cerumen (secretions from the ceruminous and pilosebaceous glands), sweat, dust, and other debris. The amount and consistency of ear wax vary from person to person. Ear wax can vary in color from yellow to orange or reddish-brown to dark brown or almost black. It may be nearly liquid or thick, sticky or dry, or soft or hard. Wax type is genetically inherited, although the appearance of wax may vary from time to time in the same person. Cerumen type has been used by anthropologists to track human migratory patterns, such as those of the Inuit. There are two main types, wet and dry. Dry flaky wax is common in persons of Asian descent and Native Americans (Overfield, 1985). Dry wax contains by weight about 20% lipid. Wet wax is common in people of Western European descent (Caucasians) and people of African descent (Overfield, 1985) and consists of approximately 50% lipid (Burkhart et al., 2000). Wet wax can be either soft or hard, with hard wax being more likely to be impacted.

Why Do We Have Ear Wax?

Various hypotheses have been advanced as to the purpose of ear wax. It has been proposed that wax provides protection against foreign objects, assists in cleaning the ear canal, acts as a lubricant, acts as an antibacterial and antifungal agent, and promotes a healthy immune response.

Debris is removed from the ear canal by a "conveyor belt" process of epithelial migration that is aided by jaw movement. Cells of the tympanic membrane migrate outward from the umbo (at a rate equivalent to that of fingernail growth) to the walls of the ear canal. The speed of cell migration accelerates as the cells move outward to the entrance of the ear canal. The cerumen in the canal is also carried outward, taking with it any dirt, dust, and particulate matter that may have gathered in the canal. Jaw movement tends to dislodge any debris attached to the walls of the ear canal, although vigorous chewing may actually stimulate wax production in some people.

Wax can also act as a lubricant, preventing drying and itching of the skin in the ear canal (asteatosis). In wet-type cerumen, the lubricating effect is due to the presence of cholesterol, squalene, long-chain fatty acids, and alcohols produced by the sebaceous glands (Harvey, 1989).

Studies have found that cerumen can provide protection against some strains of bacteria. Chai and Chai (1980), among others, have found cerumen to be effective in reducing the viability of a wide range of bacteria (sometimes by up to 99%), including *Haemophilus influenzae, Staphylococcus aureus,* and many variants of *Escherichia coli.* These antimicrobial properties are due to the presence of saturated fatty acids, lysozymes, and the relatively low pH of cerumen, which is typically around 6.1 in normal ear canals (Roland and Marple, 1997). Sirigu et al. (1997) also showed evidence of an antibody-mediated local immune response in the ear canal associated with the production and presence of cerumen, whereas Fairey et al. (1985) found that too little ear wax increases the risk of infection.

Removal of Ear Wax

If wax is hard and impacted in the ear canal, it may cause damage to the skin as it is removed and thus should be first softened. Wax removal is often more difficult for older people because their wax tends to be drier and harder. Ear wax can be softened by applying a few drops of mineral oil, baby oil, or glycerin in the ear for several days in a row. Oil should be administered at night time so that it can be absorbed into the wax and skin overnight. If oil is administered in the morning, the oil will likely get into the hearing aid when inserted and possibly disable the hearing aid.

Over-the-counter drops claiming to soften wax are available, most commonly with carbamide peroxide as the active ingredient. Other common active ingredients found in commercial wax removal preparations are triethanolamine oleate and docusate sodium. However, a recent study (Roland et al., 2004) found that triethanolamine oleate and carbamide peroxide were no more effective than a placebo (an isotonic salt solution) in aiding the removal of cerumen from occluded ear canals in an office setting. Docusate sodium and sodium bicarbonate have also been studied with conflicting results.

Once the ear wax has been softened, it can be removed with a minimum of discomfort. A standard protocol should be developed that includes obtaining informed consent and proper safety measures. The removal of wax may cause minor trauma to the ear canal, resulting in small amounts of blood. Since blood is a bodily fluid capable of transmitting various diseases, appropriate infection control measures should always be taken during cerumen removal.

Syringing with Water

Syringing with water can be done by a client at home, by a trained audiologist, by a family doctor, or by another qualified person. Water pressure may, however, push the wax deeper into the canal (possibly touching the eardrum), whereas significant amounts of water may remain in the ear canal after syringing. When hydrogen peroxide (H_2O_2) is used, oxygen bubbles off, leaving water in the ear canal. A problem with wet, warm ear canals is that they make good incubators for growth of bacteria. In these instances, the ear canal may be flushed with isopropyl alcohol to displace the water and dry the skin but should be used sparingly to avoid excessive drying and itching.

Plastic Scoops

Small, flexible plastic scoops are commonly used by audiologists trained in wax removal. A good hands-free magnifier and light source are required. The basic technique is to gently scoop built-up wax from the canal. Care must be taken to minimize discomfort or trauma to the ear canal and to avoid contact with the tympanic membrane. This method is not recommended if wax is deeply impacted. Hairs in the ear canal may be embedded in the wax and can leave small amounts of blood in the canal when they are pulled out with the wax.

Suction

Suction is an effective way to remove wax and debris; however, there is a risk of damage to the ear canal and/or tympanic membrane. This method can be uncomfortable for the client, both physically because of the suction and acoustically because of the high SPLs. Suction should be used only by a qualified practitioner such as an otolaryngologist.

Cotton Swabs

Using cotton swabs to clean the ears is not recommended. Swabs tend to push wax deeper in the canal and may stimulate the production of more wax. Swabs irritate the skin of the ear canal and may damage the ear drum.

Ear Candling

Ear candling or coning is an ineffective and potentially dangerous method of cleaning the ears. A hollow candle

is placed at the entrance of the ear canal and lit, supposedly sucking out ear wax. Despite many claims that ear candling is effective for wax removal, it has been proven that the substances appearing within the cone originate from the melted candle, not from the ears (Seely et al., 1996). The suction supposedly created by the candle's flame is insufficient to remove wax and there is a substantial risk of burns, infection, obstruction of the ear canal, and perforation of the eardrum (Seely et al., 1996). Ear candling is not recommended at any time, and federal health warnings have been issued (FDA Important Alert #77-01; FDA, 1998).

Cleaning Hearing Aids

Hearing aids should be cleaned regularly as a preventive measure. A thorough cleaning every 6 months is usually sufficient to reduce repairs due to wax damage. Some clients require deep cleaning of their hearing aids every month or even more frequently, whereas others may never have a problem with wax.

A vacuum chamber with a suction tip for cleaning hearing aids is essential for any hearing care practice. The vacuum chamber loosens and removes small particles of dust and wax, whereas the suction tip removes more recalcitrant debris. Care must be used when using a suction tip because the receiver can be easily damaged.

Prevention: The Use of Wax Guards

Wax guards are the first line of defense against wax damage in a hearing aid. Different kinds of wax guards have been developed, including covers, metal springs, vented plastic plugs, and vented plastic baskets. One of the most effective is the vented plastic basket type, which is also the simplest for clients to change on their own. When clients cannot change the wax guard themselves, encourage them to bring their hearing aids in for regular cleaning and to change the wax guards.

HEARING AIDS AND TELEPHONES

The speech signal exiting from the receiver of an ordinary telephone is already slightly amplified to about 70 dB SPL, which may be sufficient for people with a mild hearing loss. For people with a severe hearing loss, using a telephone can be a frustrating experience because of a lack of amplification and visual cues and/or poor word recognition.

Feedback is also a common complaint for clients using a telephone. Feedback is generated when a hearing aid microphone is covered by the telephone receiver.

Foam Pads

For hearing aids without telecoils or acoustic telephone programs, feedback can be reduced or eliminated by adding a foam pad to a telephone receiver to increase its distance from the hearing aid microphone.

Telecoils

Hearing aids with telecoils can eliminate feedback caused when using a telephone by turning the hearing aid microphone off and allowing the telecoil to pick up the magnetic signal from the telephone receiver instead. The Hearing Aid Compatibility Act of 1988 required "essential" telephones made in the United States or imported into the United States from August 1989 onward to be compatible with telecoils. The act defines "essential" phones as "coin-operated telephones, telephones provided for emergency use, and other telephones frequently needed for use by persons using . . . hearing aids"; this definition includes workplace telephones, telephones in hospitals and nursing homes, and telephones in hotel and motel rooms. For more information on telecoils, please refer to Chapter 38, Hearing Aid Technology.

Clients using telecoils may still run into problems using the telephone. Clients may have trouble operating the program button on their hearing aids, or the pressure of the telephone receiver can toggle the program button accidentally. With behind-the-ear hearing aids, a client must be shown how to hold the telephone to the hearing aid rather than to the ear for the telecoil to maximally pick up the magnetic signal. Also, there may be instances in which an older telephone model with insufficient electromagnetic leakage is used and, in turn, may result in a weak or nonexistent signal when used with the telecoil of a hearing aid.

Autocoils

Hearing aids with autocoils are switched into the telecoil mode automatically whenever a magnetic field is detected. The magnetic field must be very close to the hearing aid to trigger the autocoil. A typical problem with autocoils is that clients may not hold the phone close enough to trigger the autocoil. Counseling the client to hold the phone right up to the hearing aid should solve the problem. The initial feedback the client may hear should disappear quickly as the autocoil switches modes and the microphone is turned off.

A client may have a phone that does not have a strong enough magnetic field to trigger the autocoil. Adding a magnet to the telephone receiver may solve the problem, and a larger magnet or a second magnet may be added if required.

Magnetic Interference and Telecoils

When a hearing aid is in the telecoil mode, any strong or nearby electromagnetic signal may be detected and amplified, producing a buzzing sound. Common sources of electromagnetic interference include fluorescent lights, microwave ovens, televisions, tube-type computer monitors, power lines, and electrical transformers.

Because the strength of an electromagnetic field often varies considerably with small changes of position, it is sometimes possible to minimize interference by moving the head a few inches to one side. In some places, effective telephone communication with a telecoil is simply not possible given the strength of the interference.

Cell Phones

Historically, cell phones were not required under US law to be compatible with hearing aids; however, Section 255 of the US Telecommunications Act of 1996 requires a phased improvement of compatibility according to ANSI PC63.19, (2011) in new cell phones sold after 2005.

For hearing aids with telecoils, a special neckloop may be plugged into the headset jack of the cell phone to transmit the signal to one or both of the hearing aids.

Bluetooth wireless technology can bridge the gap between hearing aids and cell phones. Hearing aids with Bluetooth can automatically receive wireless signals from Bluetooth-enabled cell phones. FM systems with Bluetooth can wirelessly forward signals from a Bluetooth-enabled cell phone to a hearing aid with an FM receiver. The future may see new technologies being used to communicate with cell phones.

Modified Telephone Use

Modifications have been made to telephones to make them more accessible to the hearing aid user. There are many amplified telephones with built-in volume boost controls and high-quality speaker phones that allow for binaural listening. A direct connection from a telephone to a hearing aid via direct audio input (DAI) is also possible if the telephone has an output or headphone jack.

Frequency-Modulated Systems

FM systems provide another way to access the telephone via a hearing aid as well as allow for binaural listening. Binaural listening on a telephone can significantly improve clarity and efficacy of communication for many people with hearing loss. With the correct connector cord, any telephone with an output or headphone jack can be routed to the auxiliary input of an FM transmitter. Many FM systems can be set to automatically switch into the telephone mode when the phone rings.

Acoustic Telephone Programs

Hearing aids without a telecoil may allow for an acoustic telephone program. Because telephones only transmit information at 3,000 Hz and below, amplification of the higher frequencies in the hearing aid can be reduced sufficiently to eliminate feedback. Significant reduction of higher frequencies may, however, impair effective speech discrimination. Therefore, hearing aids should have two or more programs with the primary program left unchanged.

Alternatives to the Telephone

Some people with severe hearing loss or poor word recognition cannot use the telephone at all. Many text-based alternatives are available including voice carry over (VCO) telephones, e-mail, teletype devices, Blackberry communicators, pagers, and fax machines. The Blackberry and pagers usually have a vibration option to alert the owner to incoming messages.

BATTERIES

Clients may be shocked to learn that a battery will only last days or weeks—typically 150 hours of use. It might be useful to mention to a client that a battery would also only last 150 hours in a flashlight or radio as well. Chips for high-end hearing aids have become more and more sophisticated, incorporating more sound-processing features such as noise reduction, speech enhancement, adaptive directionality, and feedback cancellation, which, in turn, put a higher demand on the battery. However, even though the demands placed on batteries have increased, batteries have improved in strength and current drain, thus keeping overall battery life fairly constant.

The current standard is zinc air batteries. These are significantly more efficient than mercury or silver batteries and can be safely disposed of, unlike the mercury-based batteries.

How Do Batteries Work?

Zinc air batteries require oxygen to produce energy. Since they contain tiny air holes, environmental factors such as humidity can affect battery life. The batteries come with tabs to cover the air holes and may require several minutes to fully activate after the tab is removed.

Testing Batteries

When using a battery tester, count to three slowly and look for any sign of decrease in power. A battery that initially appears to be at full power may begin to fade after several seconds.

Most hearing aids have a low-battery warning; however, clients may not hear the warning beep if the hearing aid is blocked with wax or if the battery dies before emitting the warning signal. Clients should be advised to check for bad batteries and to recognize any low-battery warning given by their hearing aids. Most clients would benefit from purchasing their own battery tester.

Corrosion

Clients who sweat a lot or who live in a hot humid climate can experience chronic sweat-induced corrosion, a dark residue forming on the battery that has been described as "rust." This corrosion occurs when sweat enters the battery compartment and bridges the positive and negative terminals of the battery. Salty sweat serves as an electrolyte that promotes ionic conduction, causing the battery to corrode. The corrosion is rapid, and severe "rusting" may be observed in a matter of minutes in some individuals. This problem is mostly associated with behind-the-ear hearing aids because the location of the aid allows for easy sweat access. A protective covering over the hearing aid, such as sweatbands or SuperSeals, or a waterproof or water-resistant hearing aid, such as the Rionet line, may solve the problem.

Clients who are regularly exposed to certain chemicals, such as high levels of chlorine in an indoor pool, may experience chronic chemical corrosion of their batteries. If the hearing aid must be worn in this environment, the use of a waterproof or water-resistant behind-the-ear model, such as the Rionet series, may be required.

Malfunctioning Battery

Batteries occasionally cause unusual problems with hearing aids. Some hearing aid circuits will experience increased distortion or feedback when the battery is about to expire. If no obvious cause for a problem can be found, a new battery from a different batch and brand may eliminate the problem.

Maximizing the Life of Batteries

Hearing aid batteries should be stored at room temperature and will operate best within the humidity range of 50% to 60%. When humidity is above 60%, clients may use a dry-aid kit for both their hearing aid and batteries. Batteries should also not be kept in locations where they may short out against metal objects.

Replacing the tab on the positive side of the battery may be beneficial if the typical battery life is longer than 10 days.

A tight-fitting battery compartment in a hearing aid that is made airtight by debris buildup can result in shortened battery life. Several manufacturers now have water-resistant and waterproof hearing aids to avoid this problem by using a ventilation window covered by a membrane on the battery compartment, which allows air to pass freely but repels liquids. In addition, nano-coating of hearing aid circuitry is now widespread in the hearing aid industry.

PREVENTION

The importance of preventing common problems before they happen cannot be overstated. A positive, trouble-free experience for the client will reinforce the audiologist's message that hearing aids are supposed to make life easier and improve communication and the quality of life. Many of the common problems that discourage hearing aid use can be minimized or avoided during the prescription and fitting process. Table 39.8 lists a number of ways in which common problems with hearing aids can be prevented.

TABLE 39.8

Prevention of Common Problems

- Evaluation of listening needs and prescription
  - Assess listening needs and physical abilities to ensure optimum prescription
  - Choose style and venting to minimize occlusion, sweat, and pressure
  - Include directional microphones whenever possible
  - Choose a circuit with fast real-time feedback cancellation whenever possible
  - Order appropriate wax guards
  - Take deep, accurate open-jaw ear impressions
  - Add canal or helix lock to overcome poor retention in the ear
  - Set adaptation levels as appropriate for each client
- Expectations and training
  - Discuss expectations with the client
  - Provide thorough training to the client during the initial fitting
  - Recommend regular cleaning for both the hearing aids and the client's ears
  - Provide Dri-Aid kit, battery tester, and appropriate accessories
  - Recommend the use of devices such as Dry & Store
  - Provide documentation and written instructions for new users
  - Provide aural rehabilitation (orientation classes, counseling, reading materials, etc.)
- Follow-up
  - Insist on timely and thorough follow-ups within the first months
  - Set up a callback system to see how clients are doing during the first few days
  - Encourage clients to call if they are having any trouble

CONCLUSIONS

This chapter discussed many troubleshooting tips for hearing aids, as well as proper hearing aid assessment protocols. These included both formal and informal assessment of the hearing aids. Formal testing involves the use of hearing aid analyzers, different couplers, various test stimuli, different measures for testing digital hearing aids and advanced

functions, and the appropriate ANSI standard. Informal assessment focuses on visual assessment of the hearing aid and its components, the ear itself, and the fitting of the hearing aid to the ear.

As with a hearing assessment, a detailed up-to-date case history and background are necessary for each client. This is especially important for new clients. Even changes in weight or occupation may affect hearing aid functioning. The strategies, tips, and testing protocols in this chapter will assist you in helping your clients receive optimal benefit from their personal amplification.

FOOD FOR THOUGHT

1. There appears to be a harmonizing of the hearing aid test standards internationally. Will this trend continue and will it affect other areas of the hearing aid industry? For example, in the United States the FDA has approved the use of personal sound amplification products (PSAPs). Will these (or similar standards) be applied to the manufacture and testing of these products?
2. The standards mentioned in this chapter are reporting standards—a delineation of how to test hearing aids, tolerances of the tests, and how to report the data on specification sheets. To date there are no performance standards—how a hearing aid should perform, rather how a hearing aid does perform. What are your views about the future development of performance standards?
3. In the most recent suggestions by ANSI/ASA there is a recommendation to remove tests such as attack time and release time from the standard and place them in an informative (but optional) annex. Yet, data such as attack and release times can affect the ultimate hearing aid fitting. Why do you think that attack time and release time tests will be relegated to the annex and what other measures might follow in their path in the future?

IN MEMORIAM

This chapter is dedicated to the memory of Moneca Price, whose sudden and unexpected death during the final revisions of the sixth edition of this handbook shocked and saddened all those who knew her. Although much of this has been updated in this current edition, the spirit of Moneca will not be forgotten.

REFERENCES

ANSI PC63.19. (2011) *American National Standard for Methods of Measurement of Compatibility between Wireless Communications Devices and Hearing Aids.* ANSI PC63.19-2011. New York: Institute of Electrical and Electronics Engineers.

ANSI S3.7. (1995) *American National Standard Method for Coupler Calibration of Earphones.* ANSI/ASA S3.7-1995 (R2008). New York: American National Standards Institute.

ANSI S3.22. (2009) *American National Standard Specification of Hearing Aid Characteristics.* ANSI/ASA S3.22-2009, Revision of ANSI S3.22-2003. New York: American National Standards Institute.

ANSI S3.25. (2009) *American National Standard for an Occluded Ear Simulator.* ANSI/ASA S3.25-2009. New York: American National Standards Institute.

ANSI S3.35. (2010) *American National Standard Method of Measurement of Performance Characteristics of Hearing Aids under Simulated Real-Ear Working Conditions.* ANSI/ASA S3.35-2010. New York: American National Standards Institute.

ANSI S3.42. (1992) *American National Standard Testing Hearing Aids with a Broad-Band Noise Signal.* ANSI/ASA S3.42-1992/Part1 (R2012). New York: American National Standards Institute.

ANSI S3.42. (2012) *American National Standard Methods for Characterizing Signal Processing in Hearing Aids with a Speech-Like Signal.* ANSI/ASA S3.42-2012/Part2/IEC 60118-15:2011. New York: American National Standards Institute.

ANSI S3.46. (2013) *American National Standard Method Methods of Measurement of Real-Ear Performance Characteristics of Hearing Aids.* ANSI S3.46 - 2013. New York: American National Standards Institute.

Bankaitis AU, Kemp RJ. (2003) *Infection Control in the Hearing Aid Clinic.* St. Louis, MO: Auban.

Burkhart CN, Burkhart CG, Williams S, Andrews PC, Adappa V, Arbogast J. (2000) In pursuit of ceruminolytic agents: a study of ear wax composition. *Am J Otol.* 21, 157–160.

Byrne D, Dillon H, Tran K. (1994) An international comparison of long-term average speech spectra. *J Acoust Soc Am.* 96, 2108–2120.

Chai TJ, Chai TC. (1980) Bactericidal activity of cerumen. *Antimicrob Agents Chemother.* 18, 638–641.

Chasin M, Pirzanski C, Hayes D, Mueller G. (1997) The real ear occluded response (REOR) as a clinical predictor. *Hear Rev.* 4, 22–26.

Chung K. (2004a) Challenges and recent developments in hearing aids. Part I. *Trends Amplif.* 8, 83–124.

Chung K. (2004b) Challenges and recent developments in hearing aids. Part II. Feedback and occlusion effect reduction strategies, laser shell manufacturing processes, and other signal processing technologies. *Trends Amplif.* 8, 125–164.

Cox RM, Moore JN. (1988) Composite speech spectrum for hearing aid gain prescriptions. *J Speech Hear Res.* 31, 102–107.

Dreschler WA, Vershuure H, Ludvigsen C, Westermann S. (2001) ICRA noises: artificial noise signals with speech-like spectral and temporal properties for hearing instrument assessment. *Audiology.* 40, 148–157.

Dunn HK, White SD. (1940) Statistical measurements on conversational speech. *J Acoust Soc Am.* 11, 278–288.

Estabrooks W, Birkenshaw-Fleming L. (2003) *Songs for Listening! Songs for Life!* Washington, DC: A.G. Bell Association for the Deaf and Hard of Hearing.

Fairey A, Freer CB, Machin D. (1985) Ear wax and otitis media in children. *Br Med J (Clin Res Ed).* 291, 387–388.

FDA. (1998) *FDA Important Alert #77-01.* Government of the United States of America.

Freed D, Soli S. (2005) An objective procedure for evaluation of adaptive antifeedback algorithms in hearing aids. *Ear Hear.* 27, 382–398.

Gray RF, Sharma A, Vowler SL. (2005) Relative humidity of the external auditory canal in normal and abnormal ears, and its pathogenic effect. *Clin Otolaryngol.* 30, 105–111.

Harvey DJ. (1989) Identification of long-chain fatty acids and alcohols from human cerumen by the use of picolinyl and nicotinate esters. *Biomed Environ Mass Spectrom.* 18, 719–723.

Henning R, Bentler R. (2005) Compression-dependent differences in hearing aid gain between speech and nonspeech input signals. *Ear Hear.* 26, 409–422.

IEC 60318-4. (2010) *Occluded-Ear Simulator for the Measurement of Earphones Coupled to the Ear by Ear Inserts.* IEC 60318-4, 2010. Geneva: International Electrotechnical Commission.

Kochkin S. (2000) MarkeTrak V: why my hearing aids are in the drawer: the consumer's perspective. *Hear J.* 53, 34–42.

Kuk F. (2005) Developing a hierarchy to manage the "own voice" problem. Session at American Academy of Audiology Conference, Washington, DC, April 2005.

Oliviera R, Babcock M, Venem M, Hoeker G, Parish B, Vasant K. (2005) The dynamic ear canal and its implications: the problem may be the ear, not the impression. *Hear Rev.* 12, 18–19, 82.

Olsen L, Musch H, Stuck C. (2001) Digital solutions for feedback control. *Hear Rev.* 8, 44–49.

Overfield T. (1985) *Biologic Variation in Health and Illness: Race, Age, and Sex Differences.* Menlo Park, CA: Addison-Wesley Publishing.

Pirzanski C. (1998) Diminishing the occlusion effect: clinician/manufacturer factors. *Hear J.* 51, 66–78.

Roland PS, Eaton DA, Gross RD, Wall GM, Conroy PJ, Garadi R, et al. (2004) Randomized, placebo-controlled evaluation of Cerumenex and Murine earwax removal products. *Arch Otolaryngol Head Neck Surg.* 130, 1175–1177.

Roland PS, Marple BF. (1997) Disorders of the external auditory canal. *J Am Acad Audiol.* 8, 367–378.

Scollie S, Seewald R. (2002) Evaluation of electroacoustic test signals. *Ear Hear.* 23, 477–487.

Seely DR, Quigley SM, Langman AW. (1996) Ear candles–efficacy and safety. *Laryngoscope.* 106, 1226–1229.

Sirigu P, Perra MT, Ferreli C, Maxia C, Turno F. (1997) Local immune response in the skin of the external auditory meatus: an immunohistochemical study. *Microsc Res Tech.* 38, 329–334.

Smriga D. (2004) How to measure and demonstrate four key digital hearing aid performance measures. *Hear Rev.* 11, 26–31.

Zwislocki J. (1971) *An Ear-Like Coupler for Earphone Calibration.* Report LSC-S-9, Laboratory of Sensory Communication, Syracuse University, April 1971. Syracuse, NY: Syracuse University.

CHAPTER 40

Hearing Aid Fitting for Children: Selection, Fitting, Verification, and Validation

Susan Scollie

INTRODUCTION

Audiologists who provide hearing aids for children serve infants, toddlers, preschoolers, children, teens, and their families. This wide-ranging population has specific needs for listening, language acquisition, and the physical fit of hearing aids. This chapter will outline the key differences for the pediatric population and introduce best practices for hearing aid fitting and follow-up by audiologists.

PEDIATRIC VERSUS ADULT HEARING AID FITTING

Recently, the American Academy of Audiology issued a Pediatric Amplification Guideline (AAA, 2013). This document stresses nine ways in which children and adults differ in their needs for listening and hearing aid use. Specifically, the nature of childhood hearing loss is unique: It is more likely to be variable between ears, between children, and over time within a child, and more likely to be associated with other health conditions. Also, children's hearing must be tested using different methods and their small but growing ear canals change the assessment and the hearing aid fitting over time. Further, children's listening needs frequently include the use of aided listening as a support for speech and language acquisition and development. This limits their ability to use top-down processing and increases their need for level, signal-to-noise ratio (SNR), and bandwidth of the signal. Finally, children's hearing aids may require that a caregiver initiate and/or monitor device use, meaning that both caregivers and children are the recipients of our care. All of these realities impact our protocols and service delivery to the pediatric population, particularly in infancy.

Clinical practice in pediatric amplification follows a sequence of events as shown in Figure 40.1. Not shown in Figure 40.1 are the many feedback paths that occur throughout, including the important link between assessing outcomes, defining next steps, and revisions to device selection, physical fit, or signal processing decisions. Because there are no "typical" children, it should be a fluid family-centered and child-centered practice, with revision of decisions in response to changes. The steps illustrated in Figure 40.1 will serve as an overall organization for this chapter.

Identifying Hearing Loss

Many children who have permanent congenital hearing losses are now identified in infancy because of universal newborn hearing screening programs and associated early intervention initiatives (JCIH, 2007, 2013). In this service route, children are referred for a full audiologic assessment based on a screening result, and hearing assessment takes place in infancy. For infants, the main tools for hearing assessment are auditory-evoked potentials. Both the frequency-specific auditory brainstem response (FS-ABR) and the auditory steady-state response (ASSR) are used to predict the behavioral audiogram. Corrections are applied to achieve a more accurate prediction of hearing thresholds. This occurs *after* testing in some FS-ABR protocols (Figure 40.2) or *as part of the test setup* in ASSR and other ABR protocols. The corrections generate an estimated audiogram which is then used

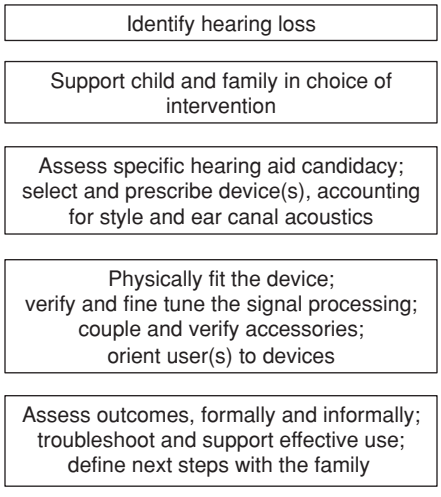


FIGURE 40.1 Components of the clinical pathway for pediatric amplification.

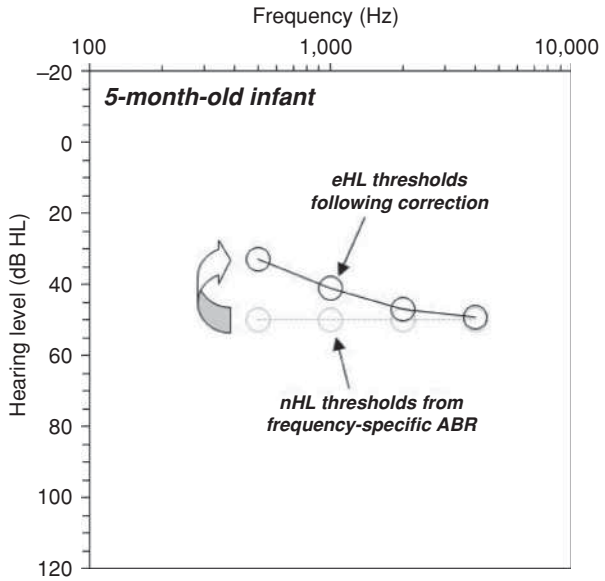


FIGURE 40.2 Estimated audiogram from frequency-specific auditory brainstem responses (FS-ABRs) for a 5-month-old infant. Values are shown for both uncorrected (*light*) and corrected (*dark*) threshold estimates.

as the basis for further discussions with families, including whether or not the infant may be a candidate for hearing aid use, and subsequent calculation of prescribed hearing aid gain and output. Accuracy of these corrections is therefore an important precursor to accurate infant hearing aid fitting.

Assessment of hearing loss continues past early infancy, transitioning from electrophysiological methods to behavioral testing once conditioned procedures are possible. For older infants and young children assessment of hearing uses behavioral methods (visual reinforcement audiometry, conditioned play audiometry, or standard procedures). In the early months and years of life, behavioral measures of threshold may be best described as minimum auditory response levels (MRL), which are up to 25 dB above true threshold in children with normal cochlear sensitivity (Sabo et al., 2003). Infants and young children may cease responding prior to test completion on a given day, so repeated assessments may be required before behavioral estimates of thresholds are available, via air and bone, for each ear. Obtaining high-quality assessment of a child's ear-specific and frequency-specific hearing before making candidacy decisions is important because children have higher incidence of asymmetrical, progressive, and conductive/mixed hearing losses (Gravel, 2002; Pittman and Stelmachowicz, 2003; Tharpe, 2008). Specifically, the results of sound field threshold testing are not considered sufficient information for hearing aid fitting, because they are not ear specific. It is possible to perform audiometry with insert or TDH headphones, thus obtaining ear-specific threshold data. In general, insert phones are preferred, because their lighter weight permits easy head movement. Also, they prevent collapse of the external ear canal, provide better attenuation of room noise, and reduce the need for masking in air conduction audiometry because of increased interaural attenuation (Clemis et al., 1986; Wright and Frank, 1992).

Supporting Families

Family-centered and child-centered practice includes working with families to support their choices in communication development, and to support them in modifying these choices over time. In structured family-centered programs, many children are provided with hearing aids, either in support of oral language acquisition, or in support of environmental sound awareness or bimodal communication, or as part of an assessment for cochlear implant candidacy. Audiologists can support families in making amplification-related decisions by providing clear information about the nature of a child's unaided hearing, realistic expectations about aided hearing abilities, trial fittings, best practices, and an overall family-centered approach that builds trust between the professional and the caregiver. Early hearing aid fittings are typically simple, with the focus on orientation to the device, getting started with device use, and monitoring the newly fitted infant or child for their responses to aided sound. These topics will be covered in more detail later in this chapter.

Key messages to families in the early days include reasonable expectations for clinical processes and behavioral outcomes based on the link between auditory input and the development of oral speech and language. Clinicians communicate the typical frequency and type of appointments to expect, as well as the types of procedures that will be completed. Although this will vary with the age of the child and the nature of their hearing loss and other health conditions, good generic descriptions, pictures, and videos are available from evidence-based, parent-focused web resources (www.babyhearing.org) or from books that share stories from other families of children who have hearing loss (e.g., Waldman and Roush, 2009). Parent-to-parent organizations can provide valuable support to families, regardless of their choice of oral and/or manual communication modes (DesGeorges, 2013).

Assessing Hearing Aid Candidacy

When families choose to pursue hearing aids for their infants or children, they may do so to support oral language development or to support sound awareness, or they may wish to explore whether or not hearing aid use will be helpful to their child. In cases of uncertain candidacy, either because the hearing loss is mild, profound, or unilateral, the fitting may be done on a trial basis to determine candidacy for continued use. In all cases, the idea behind hearing aid fitting is to provide the child with more access to the

auditory world than what is available without hearing aids. To understand this, we should consider what may help about hearing aids as well as any barriers to and potential negative side effects of hearing aid use.

Barriers to consistent hearing aid use can include the financial costs to families. These include the direct and indirect costs of attending appointments, including time off work, child minding for siblings, parking, travel, and appointment fees, as well as costs associated with device use including device purchase, earmolds, batteries, accessories, and insurance. Assistance with financial costs is often within the scope of the pediatric audiologist, by supporting families in applications and with temporary devices while funding access is in process.

Negative side effects that have been considered in the literature are rare and diverse, and prevention strategies are available (AAA, 2013). Certain ear impression and earmold materials may sensitize the hearing aid user to contact dermatitis, but we can avoid the use of these materials (AAA, 2013). We can protect the child's pinna from harm if struck during hearing aid use by ordering earmolds made of soft materials. We can protect the child's hearing from overamplification by fine tuning and verifying to ensure that the child's hearing aids do not exceed prescribed levels of sound from a validated prescription (more on this later in this chapter). Further protection from overamplification is available by including signal processing technologies that reduce the levels of loud sounds, such as output compression limiting and wide dynamic range compression. Finally, we can minimize the impacts of additional noise and/or distortion by choosing hearing aids with low noise and low distortion, performing careful listening checks, and using venting whenever possible and indicated.

Candidacy-related conversations with families will typically focus more on whether positive effects of hearing aid use are expected. Families will need to know the type and nature of the child's hearing loss, that hearing may not be the same across frequencies, and that the child's audibility of speech will be affected in a frequency-specific way according to their hearing thresholds. An understanding of these basic concepts will help to support understanding of how hearing aids should help: By amplifying more in some frequencies, hearing aids overcome some of the effects of the specific shape and degree of hearing loss for the child. Realistic expectations for auditory development should be built for the child-specific case, taking into account current evidence on outcomes, the child's degree of hearing loss, the effects of support, daily use, and room acoustics, and the importance of engaging in appropriate interventions. Answering parents' questions as they are asked and providing written materials to take home are two of many strategies for supporting families as they receive this new information.

At early stages, audiologists can help parents understand (un)aided speech acoustics through the use of visual depictions of speech and from listening to filtered speech demonstrations. One example of this is the familiar sounds audiogram, which can be used to relate audiometric data to the audibility of specific phonemes and environmental sounds, keeping the audiometric and phonemic information on a common dB HL scale. Another example is the SPLogram, which was developed to assist explanations of audibility of speech, by demonstration with and without a hearing aid. SPLograms display the audiometric and spectral information on a common dB SPL scale. Whether the clinician chooses to use an HL or SPL approach during informational counseling, it is wise to remember that the change between HL and SPL is confusing at first, so care should be taken when moving between display formats. Displays are available in most hearing aid fitting and verification software, and in more detail within the Situational Hearing Aid Response Profile (SHARP; Boys Town National Research Hospital, 2013). A sample unaided SPLogram (Figure 40.3) provides an example of unaided speech versus a child's hearing thresholds and upper limits of comfort (ULC), with normal hearing sensitivity plotted for comparison. The region between thresholds and ULC is the *auditory area.* This example uses the same case shown in Figure 40.2 and assumes average ear canal resonances for a 5-month-old infant. The audiogram and field levels of speech have been converted to ear canal SPL using established procedures (Bagatto et al., 2005). The unaided long-term average speech spectrum (LTASS) is the middle spectrum, with the two other lines indicating the peaks (top line) and valleys (bottom line) of the amplitude distribution around the LTASS. A level of 60 dB SPL was used to represent average conversational speech (Olsen, 1998). Comparing these levels

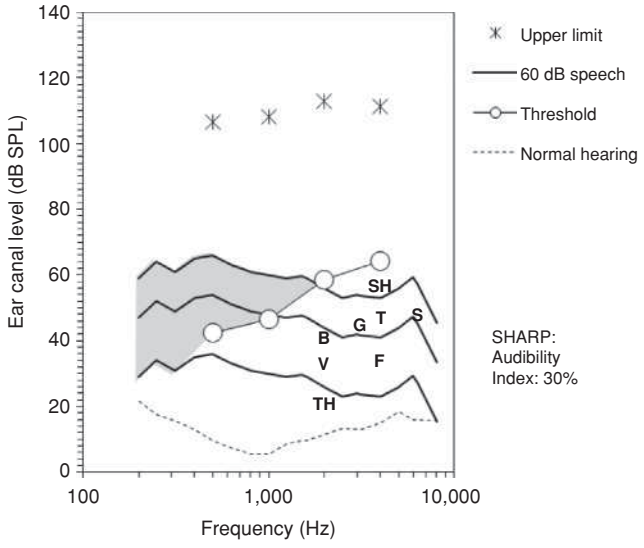


FIGURE 40.3 SPLogram for unaided conversation-level speech for a 5-month-old infant with the audiogram shown in Figure 40.2. The *shaded region* indicates speech energy that is audible. *Overlaid letters* indicate the spectral peaks of each phoneme.

to the normal thresholds, we see that all of the unaided speech amplitude distribution is above threshold. In contrast, only the shaded region is above threshold for this child. The SHARP version 7 (Boys Town National Research Hospital, 2013) was used to estimate the audibility index for this SPLogram resulting in an estimate of 30%. This value should be interpreted according to the speech cues available for the audible bandwidth, rather than as an estimate of the percentage of speech that might be understood correctly (Gustafson and Pittman, 2010). In this case, the 30% of audible speech contains only low-frequency energy, which will allow detection of most broadband environmental sounds and recognition of some vowels, nasals, liquids, glides, and certain mid-frequency consonants. However, high-frequency environmental sounds as well as fricatives, upper vowel formants, and high-frequency bursts or plosives may be inaudible. Explaining these distinctions to families is important, because a child with this degree and configuration of hearing loss will most certainly be aware of sounds in the environment but is at risk for poor oral language development without appropriate amplification. If the family chooses hearing aid intervention as a support for oral language development, bandwidth of audibility is an important factor to consider in candidacy. Limited bandwidth from children's hearing aids restricts access to some phonemes (see review by Stelmachowicz et al., 2004) and may impair word learning (Pittman, 2008).

Children who have similar hearing loss in both ears are likely candidates for binaural hearing aid fitting. Selection decisions can be more complex when the hearing profile is asymmetrical. Children are generally considered candidates for a trial with amplification even when hearing loss is mild or confined to only the high frequencies. Children with unilateral cochlear implants are generally considered candidates for amplification in the nonimplanted ear unless contradictions are present. Further guidance and evidence review on specific profiles is available from the AAA (AAA, 2013) and from evidence-based practice guidelines (e.g., Cincinnati Children's Hospital Medical Center, 2009). When candidacy for amplification is uncertain, offering a trial with loaner devices with monitoring of outcomes is an important tool that can assist families in determining whether or not hearing aid use is desired.

SELECTING AND PRESCRIBING HEARING AIDS

Physical Characteristics

The AAA provides good general guidelines for the selection of physical characteristics of hearing aids (AAA, 2013). They emphasize that most children's needs will be met with binaural, flexible fittings that offer direct audio input, usually in the form of behind-the-ear (BTE) casings attached to soft earmolds or soft-cased receiver tips. Soft casings are used to protect the external ear from injury. Earmold venting is used, when the ear's size permits it, to allow airflow into the ear canal and possibly to provide access to timing cues for localization, depending on the configurations of hearing thresholds in both ears (Johnstone et al., 2010). Other physical configurations should be considered as warranted by the child's hearing level, manual dexterity, age, and other factors. For example, signals may be routed via a remote microphone to an open-fitted receiver instrument coupled to the better ear of a child with unilateral deafness, rather than providing a power hearing aid fitting to the ear with profound hearing loss. Another example is fitting a bone-conducted hearing aid for use while candidacy for either bone-anchored or middle-ear implants is not yet an option because of skull growth.

For air-conducted devices, choice of an appropriate earhook can affect both frequency response and physical retention of the device on the infant or child's small ear. A full-sized unfiltered earhook on a BTE hearing aid adds resonant peaks to the electroacoustic response of the hearing aid's receiver. Simulated and real earhook resonances have not been shown to decrease speech intelligibility or quality ratings for average-level speech in adult hearing impaired listeners (Cox and Gilmore, 1986; van Buuren et al., 1996). However, there may be other reasons to remove earhook resonances through the use of filtered ("damped") earhooks. First, earhook resonances may increase the risk of acoustic feedback (Agnew, 1996) and/or loudness discomfort (van Buuren et al., 1996) for the hearing aid user, because they can add roughly 10 dB to the output of the device at approximately 1,000 Hz. Second, the peak in the hearing aid response often governs the maximum output setting of the hearing aid. If an audiologist adjusts the hearing aid to meet the target output levels, the off-peak frequencies will be unnecessarily lower than the target maximum output level. The response from a damped or filtered earhook will be smoother, allowing a broader band of audible sound to be made available. In many cases, filtering the earhook is desirable. Earhook filters may have less effect with some newer earhook designs: Some recently introduced shorter earhooks are about half the length of a traditional hook. This reduces the magnitude of the earhook resonance, resulting in less need for damping or filtering. If in doubt, test the frequency response of the hearing aid both with and without a damped hook and compare the two. Small earhook designs for pediatric uses are particularly important for infant hearing aid fitting. Pediatric earhooks are typically designed to be smaller and more tightly curved than those intended for adult pinnae. They "hug" the top of the pinna and aid in retention of the hearing aid. When combined with other retention strategies, such as two-sided tape or hearing aid retention cord systems, they can have a significant impact on hearing aids staying on rather than flopping off. Together with tamper-resistant battery doors to prevent the infant or toddler from ingesting toxic batteries, a pediatric earhook is a common physical feature of hearing aids for very young children.

A well-fitted, comfortable soft earmold or a soft custom casing for a receiver-in-the-ear is also a key requirement for most children's hearing aid fittings. Either of these requires that an accurate ear impression is taken and that earmold fit is closely monitored over time, particularly during ages 0 to 2, when rapid growth of the external ear is expected. This continues at a slower rate to about age 5 or 6. Ear impression techniques require good distraction of the child (with videos, toys, or games) while the clinician ensures the ears are clear, places a tight otoblock, and injects impression material into the ear canal. Remember that the child will not be able to hear you while the ear impression material is in his or her ears. The ear canal should be examined following impression taking to ensure that all material has been removed and that no injury or skin reactions are observed and the results should be documented. Pediatric ear impression supplies are available, including low-capacity syringes that hold less ear impression material (for smaller ears) and come in bright colors. Thick-walled tubing in an earmold can reduce the risk of feedback. However, in very small earmolds, tubing may be a narrower gauge and/or extend only halfway through the mold to prevent high-frequency rolloff caused by tube crimping at the end of the earmold (AAA, 2013). Colorful earmolds with sparkles, swirls, and pictures of favorite characters are all options that young children like to choose. Teens and tweens may continue to choose bright colors or may wish to change styles and colors to minimize contrast with skin and hair tones. Matte tubing or receiver-in-the-ear options may facilitate continued acceptance of BTE or mini-BTE styles and therefore maintain options for high-gain fitting and/or remote microphone coupling via DAI or other routings.

Signal Processing

Modern hearing instrument technology includes signal processing to control the level of sound, to split it into multiple channels with channel-specific processing, to use one or more microphones to aim a beam of sensitivity to a particular direction, to attenuate noisy signals, and to use two hearing aids as a team if they are linked wirelessly. Hearing aid features allow effective feedback control, delivery of telephone signals to both ears simultaneously, and incorporation of small external microphones to enhance SNRs. Fitting software has become similarly complex, with many software-guided fitting algorithms that attempt to aid the clinician in choosing options and fine tuning the strength of some features. In this chapter, general guiding principles will lend insight into pediatric-specific issues within this landscape.

Audibility Technologies

Hearing aids improve the audibility of speech and other sounds by providing amplification of the incoming sound energy and by shaping the amplification according to the listener's hearing loss. Shaping is typically performed according to a prescription, discussed below. We can pair this shaped amplification with one of three other ear-level audibility strategies: Amplitude compression, frequency lowering, and beamforming microphone technology. In addition, we can pair ear-level technologies with remote microphones to enhance listening across distances and in the presence of competing signals. Each of these options can be considered from a pediatric perspective.

Amplitude Compression

Historically, hearing aids provided one level of gain for any level of input, until the hearing aid reaches its saturation point. This type of linear gain system is now uncommon in modern hearing aids. More commonly, the hearing aid monitors the level of the incoming sounds using a frequency-domain analysis and makes automatic adjustments to the gain of the hearing aid. This type of processing is called *amplitude compression* and is recommended for use in most pediatric fittings to improve audibility of low-level sounds (AAA, 2013; McCreery et al., 2012a). Specific products that offer amplitude compression may differ in the magnitude and the speed of the change, as well as the frequency range affected, because the frequency-domain analysis allocates unique compression parameters to each channel within the hearing aid. Alternatives to amplitude compression also exist, with systems monitoring the levels of the incoming signal in the time domain rather than in the frequency domain. With either type of system, a frequency-shaped automatic volume control is achieved: When sounds get softer, the hearing aid turns the gain up and vice versa. Specific products may combine amplitude compression with either input compression limiting (to prevent microphone distortion), input expansion (to lower the gain applied to the microphone noise floor), and/or output compression limiting (to prevent receiver saturation). Combinations of these nonlinear technologies are common in today's products and are often used in children's hearing aids.

Frequency Lowering

Some hearing aid users may have limited access to the high-frequency portions of the incoming signal, even with well-fitted conventional hearing aids. If this could limit the benefit derived from amplification, these hearing aid users may be considered candidates for frequency-lowering systems. These devices apply signal processing to move high-frequency energy from the input into a lower-frequency range within the aided signal. Fitting procedures are often aimed at providing enough lowering effect to move the aided lowered signal to a usable region of hearing. There are several ways that this may be accomplished. Early commercial devices divided the incoming frequency range into two frequency ranges and transposed the higher-frequency inputs

into the lower-frequency range. This used different frequency range definitions depending on whether the input signal was judged as vowel like or consonant like by the device's processor. More recently introduced devices include other strategies, such as frequency compression, spectral warping, and others (Alexander, 2013). All of these are encompassed by the umbrella term "frequency lowering," but they may provide different types and strengths of frequency lowering when compared with one another. Some confine their effects to only the very high frequencies, some are adaptive processors, and some create nonlinearities, mixing of lowered with unlowered energy, and/or have significant filtering effects on the signal. To varying degrees, these side effects of frequency lowering have been viewed with skepticism, resulting in more cautious acceptance of frequency lowering at this time than is present for older technologies such as amplitude compression.

Research studies are emerging that present profiles of candidacy and outcomes with various frequency-lowering schemes (Auriemmo et al., 2009; Glista et al., 2009; McCreery et al., 2012c; Parsa et al., 2013; Wolfe et al., 2010) and/or that propose or illustrate the efficacy or rationale for fitting methods. These methods underscore the importance of avoiding excessive strengths of frequency compression processors (Glista and Scollie, 2009; Kuk, 2013; McCreery et al., 2012; Scollie and Glista, 2011). Benefits for children with hearing losses that are severe to profound in the high frequencies are likely attainable with well-fitted frequency-lowering hearing aids, particularly following a period of acclimatization (Glista et al., 2012; Wolfe et al., 2011). Children who can achieve good audibility of high-frequency phonemes such as /s/ without frequency-lowering signal processing may not be candidates for this category of processor. Determining candidacy for this technology requires the audiologist to strike a balance between two of the fundamental requirements for signal processing: Avoiding unnecessary distortion and providing sufficient frequency shaping to meet prescriptive requirements (AAA, 2013). If high-frequency prescriptive requirements cannot be met, frequency lowering may be indicated. Monitoring of outcomes from frequency-lowering fitting is recommended practice (AAA, 2013).

Directional Microphones

Modern directional microphones use beamforming technology, which combines signals from more than one microphone to create a beam of sensitivity that may be steered to a specific direction. Sounds outside of the beam area are attenuated. Beamforming technology is commonly used in a wide range of microphone applications. In hearing aids, this takes the form of two or three microphone ports along the top edge of a BTE hearing aid with signal processing to combine the multiple inputs. Wireless microphone hearing aid accessories may also use multiple microphones to create a beam. Signal processing is used to steer the beam, such as in front of the listener for conversations in noise, or to adapt the beam to the location that provides the best SNR. Some beams can be pointed behind the listener, to serve the user in situations like driving a car with conversation partners seated one behind the other. Beamforming microphone arrays on hearing aids are known to provide SNR enhancement when used appropriately, which usually requires the listener to point their nose to the target talker. Children are able to derive substantial benefit from the beam when it is correctly oriented, but less is known about how children can appropriately use these technologies in real-world environments (Crukley and Scollie, 2013; McCreery et al., 2012b; Ricketts et al., 2007; Ricketts and Picou, 2013). Children orient their heads to the target talker less than half of the time (Ching et al., 2009; Ricketts and Galster, 2008), and directional decrements in performance are possible when the beam is aimed away from the target talker, such as when listening to someone seated to the side or rear (AAA, 2013; Ching et al., 2009; Ricketts et al., 2007; Ricketts and Picou, 2013). The age, motor abilities, and cognitive abilities of the children may factor into their ability to use a directional system appropriately. It should also be cautioned that ear-level beamforming systems are designed for use when the listener is in close proximity to the target signal. Ear-level beamformers are not a substitute for the long-distance SNR advantages available from systems with remote microphones, such as FM systems. These complex issues lead to specific pediatric recommendations: Consider technology use on a case-by-case basis, consider avoiding full-time directional use, consider adaptive directional systems if their properties are well understood, and consider omnidirectional fittings with remote microphone support to improve SNRs in specific environments (AAA, 2013; Foley et al., 2009).

Remote Microphone Systems

Remote wireless microphone technologies, such as FM systems, are an important consideration in the pediatric population. The purpose of a remote wireless microphone is to pick up a signal of interest and send it wirelessly to a receiver that is connected to a loudspeaker (soundfield configuration) or connected to the hearing aid (personal configuration). This sends a clear, high-level signal that is less contaminated by reverberation and/or background noise than the signal received at the hearing aid microphone. By sending this cleaner signal to the hearing aid, we can overcome many of the deleterious effects of distance, reverberation, and noise that often occur in everyday communication environments. In educational environments, it is possible to configure these systems to support clear access to a teacher's voice, yet also allow for multiple talkers and interfacing with equipment such as computers and room projection systems (AAA, 2008). In home use, caregivers can use these systems to support clear access to a caregiver's voice, and use the

remote microphone in the car, on playgrounds, in shopping centers, or other situations where distance, noise, and reverberation can inhibit effective communication (AAA, 2008). Historically, remote microphone systems typically used FM signal transmission and were referred to as "FM systems." More recently, alternatives to FM signal transmission have come to market, and these use various types of digital signal encoding and transmission. Some types use Bluetooth transmission or other encoding schemes for transmission only, whereas others combine this with digital noise reduction and level-dependent processing in an attempt to deliver a cleaner signal. Some are designed as adult-focused hearing aid accessories and may not be designed for the more rigorous school environment in terms of breakdown or options for interfacing other talkers. More details on the remote microphone systems are discussed in Chapter 37.

Pediatric protocols for selecting, programming, verifying, and validating remote microphone systems are available and specify required equipment and decision considerations for children who do and do not use hearing aids (AAA, 2008). For children who use hearing aids coupled to personal remote microphone systems, protocols focus on ensuring that the remote microphone transmitter/receiver is successfully sending the signal from microphone to hearing aid, often using an SPLogram and comparing to the hearing aid fitting as a reference signal. Fitting protocols also include ensuring that systems in adjacent classrooms do not interfere with one another and allocating signal processing to the microphone to enhance audibility of the target talker.

Prescription and Verification of Audibility

Prescription and verification of the gain, output, and amplitude compression of a child's hearing aid are intended to assist the pediatric audiologist in ensuring that the fitted hearing aids provide appropriate amplification across children. Prescription involves the use of a computational formula via software to compute target levels of gain or output across input levels, signal types, and frequencies that are tailored to the individual child's ear acoustics and hearing. Verification involves the use of electroacoustic test equipment to evaluate whether or not the hearing aid closely approximates the target levels. The audiologist performs fine tuning as needed to optimize the match to targets or to make other adjustments as required to meet the listening needs of the child. Because modern hearing aids use amplitude compression, verification and fine tuning are typically completed for more than input level. The use of calibrated speech during verification is increasingly preferred for its robust nature and high face validity.

An example of a basic verification and fine tuning for conversation-level speech is shown in Figure 40.4. Prescriptive targets for the case shown in Figures 40.2 and 40.3 were developed using the Desired Sensation Level (DSL) v5 prescriptive method (Scollie et al., 2005). Figure 40.4 shows verification curves that meet the target levels. The maximum output curve shows hearing aid output for a 90 dB SPL puretone sweep: This curve is at a lower SPL level than the predicted ULC for this child. This is an acceptable fitting and a typical outcome for fittings that use amplitude compression. The aided speech curves for speech at an input level of 60 dB SPL show that the peaks, LTASS, and valleys of speech have been amplified to suprathreshold levels, resulting in audibility of the peaks of the various phonemes shown and an increase of the SHARP audibility index from the unaided level of 30% to an aided level of 97%. The audible bandwidth covers the entire range of the measurement, for the peaks and LTASS of speech, with some possible inaudibility in the very low frequencies. This fitting would be expected to support access to the important cues of conversation-level speech and should support speech sound awareness in a quiet environment.

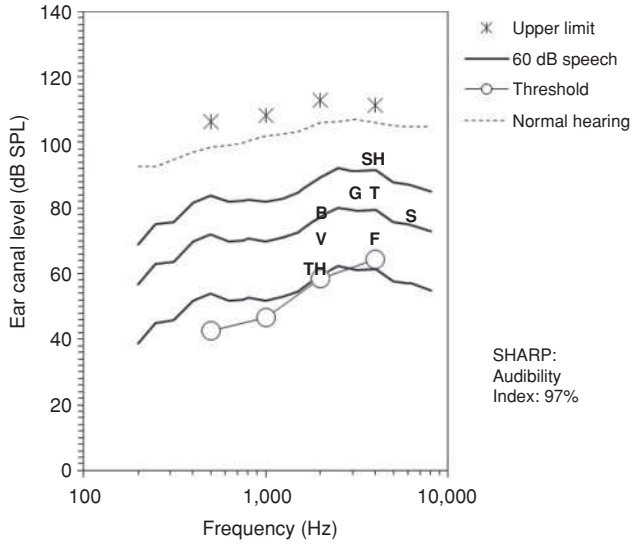


FIGURE 40.4 SPLogram for aided conversation-level speech for the same 5-month-old infant assessed in Figures 40.2 and 40.3. Format generally follows that of Figure 40.3.

This simple example attempts to illustrate the purpose and outcome of prescription and verification: A well-fitted hearing aid that provides access to a comfortable, beneficial amplified speech signal. Clinical use of this strategy requires a more detailed understanding at a deeper level, including the rationale and differences between varying target prescriptions, the specific methods used for multilevel verification, and the use of verified fittings within the context of multimemory hearing aids.

PRESCRIPTIVE FORMULAE

Historically, many prescriptive formulae have been proposed, all with the goal to harness the correlation that exists

between hearing thresholds and the level of hearing aid gain or output that might be helpful. The ratio of prescribed gain to hearing thresholds has ranged between about 30% to 66%, with some variation across prescriptive methods, and variables in addition to hearing thresholds being used within some methods. In contemporary practice, two prescriptive methods offer pediatric-specific solutions that include infant-friendly features. The sections below will review these methods.

The National Acoustic Laboratories Formula

The National Acoustic Laboratories (NAL) family of formulae have been under development for more than 30 years and are the most common prescriptive formulae used in adult hearing aid research. The original NAL formula was developed and modified by Denis Byrne and colleagues, resulting in the NAL-R prescription which aimed to amplify all bands of speech to the most comfortable level (Byrne, 1986a, 1986b; Byrne and Dillon, 1986; Byrne and Murray, 1986). Byrne et al. (1990) proposed a modification of the NAL-R formula called "NAL-RP" for use when auditory thresholds are greater than 95 dB HL at 2,000 Hz. The NAL-RP formula adds extra low-frequency gain and reduces high-frequency gain. Prescriptions for hearing aid output limits have also been proposed (Dillon and Storey, 1998). Both NAL-R and NAL-RP are linear gain prescriptions and therefore do not prescribe targets for amplitude compression.

NAL prescriptions for amplitude compression hearing aids have been developed in two subsequent versions by Harvey Dillon, Teresa Ching, Gitte Keidser, and colleagues. Both methods provide multilevel targets that are appropriate for use with amplitude compression hearing aids. A hallmark of the NAL-NL methods is a method of reducing gain and output when limits of "effective audibility" are predicted to have been reached. Effective audibility includes stimulation levels that can be used well by adults for the purposes of speech recognition (Ching et al., 1998, 2001). For example, a listener with normal hearing will have increased speech recognition scores as the level of speech increased from threshold to suprathreshold levels, until they achieve the maximum score of the test. This typically occurs when the speech signal is fully audible, usually when the peaks of speech are about 30 dB above threshold. For a listener with sensory/neural hearing loss, maximum performance may not reach the maximum possible score of the test, and this may occur at sensation levels less than about 30 dB because of the listener's reduced auditory area. This limits effective audibility. The NAL-NL methods combined experimentally derived limits with a model of loudness. An iterative algorithm was used to find hearing aid frequency responses that would allocate loudness of aided speech to those frequency regions that demonstrated greater effectiveness in a sample of adults. In NAL-NL1, this target derivation resulted in missing targets at frequencies with limited effectiveness, thus limiting the prescribed bandwidth of the fitting. In NAL-NL2, the effectiveness limit was revised to prescribe less gain rather than a missing target, allowing the prescription to compute a target across frequencies (Keidser et al., 2011). Although this makes it more difficult to see where effectiveness limits have been reached, it makes clinical use of the prescription more feasible, particularly with hearing thresholds in the severe to profound range. In the NAL-NL2 procedure, children are provided with more hearing aid gain than adults, and normative data are included that estimate and incorporate pediatric ear canal acoustics.

The Desired Sensation Level Formula

The DSL family of formulae have also been under development for many years and are the most common prescriptive formulae used in pediatric hearing aid research and in clinical practice (Jones and Launer, 2011; McCreery et al., 2013). The original DSL formulae up to and including version 3.1 were developed by Richard Seewald and colleagues. These were developed for use with linear hearing aids and included a prescription for hearing aid output limiting (Seewald et al., 1985; Seewald, 1991). Similar to the NAL-NL formulae, Seewald sought to prescribe a comfortable level of speech that was associated with maximum speech sound recognition performance in children. These early versions of DSL incorporated children's ear canal acoustics and child-friendly verification procedures and tended to prescribe more gain than the same-era NAL prescriptions.

DSL prescriptions for use with amplitude compression include the DSL[i/o] and DSLm[i/o] algorithms. These algorithms underlie versions 4 and 5 of the DSL method in software-based tools. DSL[i/o] computes targets for gain and output across frequencies and can be adjusted for use with linear or amplitude compression hearing aids (Cornelisse et al., 1995). The DSL[i/o] algorithm was updated in 2005 to include multichannel amplitude compression targets, infant-focused features, corrections for conductive and binaural fittings, and targets for adult listeners (Bagatto et al., 2005; Scollie et al., 2005). DSL version 5 continues to prescribe pediatric target listening levels that are similar to those used in the linear gain version, which aim for a broad band of audible conversation-level speech placed within the midpoint of the child's auditory area. Targets for adults receive a similar frequency shape but at a lower level of output and gain. If amplitude compression is selected, compressive target input/output functions are fitted through the conversation-level target. Infant features include normative data and incorporation of ear canal acoustics across ages, and embedded corrections from nHL to eHL if hearing assessment was performed with frequency-specific ABR (Figure 40.1).

Studies of Prescriptive Formulae

Studies evaluating outcomes with NAL, DSL, and other prescriptions have been completed for the linear gain era formulae either in isolation or when comparing the effects of linear and nonlinear prescriptions (e.g., Snik and Stollman, 1995; Snik et al., 1995; Ching et al., 1997; Jenstad et al., 1999; Scollie et al., 2000). One series of studies compared DSL[i/o] and NAL-NL1 and incorporated double blinding and a period of acclimatization to each prescription (Ching et al., 2010a, 2010b; Scollie et al., 2010a, 2010b). These studies revealed that individual children may have preference or performance advantages with one prescription or another, with children who had increased loudness sensitivity being more satisfied with the lower gain provided by NAL-NL1. Situational preferences were also common, with most children in the study preferring to use the higher gain from DSL[i/o] when listening to sounds from behind or soft sounds and some children preferring the lower NAL-NL1 gain when listening in loud or noisy environments. More recent studies have evaluated outcomes from children either with DSLm[i/o] (e.g., Bagatto et al., 2011; Crukley and Scollie, 2012, 2013; Sininger et al., 2010) or including comparisons between DSLm[i/o] and NAL-NL2 (Ching et al., 2013). On average, outcomes are generally appropriate with either prescription, provided that the hearing aids use amplitude compression and are routinely verified and fine-tuned by a pediatric audiologist who incorporates the child's ear canal acoustics. Exceptions may occur for children with severe to profound hearing losses, who perform significantly better with DSLm[i/o] than with NAL-NL1 (Quar et al., 2013). Because the two prescriptions differ most markedly when losses are severe or poorer in any frequency region, it may be the case that children with either flat, severe/profound, or steeply sloping losses are those who may have differing outcomes depending on the prescription used. The use of a validated prescription with routine verification and fine tuning of amplitude compression hearing aids appears to provide more consistent outcomes across large caseloads than fitting by other methods and is recommended practice for that reason (AAA, 2013; McCreery et al., 2013). In part, this consistency arises because pediatric prescription/verification methods are designed to take into account the pediatric principles discussed below.

BEST PRACTICES IN LINKING ASSESSMENT AND VERIFICATION

During hearing assessment, an individual's ear canal adds a unique acoustic signature to levels of sound reaching the child's eardrum, middle ear, and cochlea. Calibration in the dB HL scale assumes an average adult ear; therefore, the ear of an infant or young child may differ substantially from those assumed in calibration standards. As the child grows, this changes. This issue presents a problem for consistency and accuracy of hearing prescription, both at any one time and over time. There are two proposed solutions to this problem.

One solution is to convert the child's thresholds from HL to real-ear SPL or coupler SPL, which may be compared directly to hearing aid verification curves on an SPLogram as shown in Figure 40.4. Procedures for these conversions are well established and require either measurement or age-appropriate prediction of the child's ear canal acoustics (Figure 40.5, Table 40.1). The generic procedure is to obtain an HL audiogram, measure or predict the ear canal resonance that is appropriate for the specific procedure used during audiometry, and sum these together with the reference equivalent threshold sound pressure level (RETSPL;

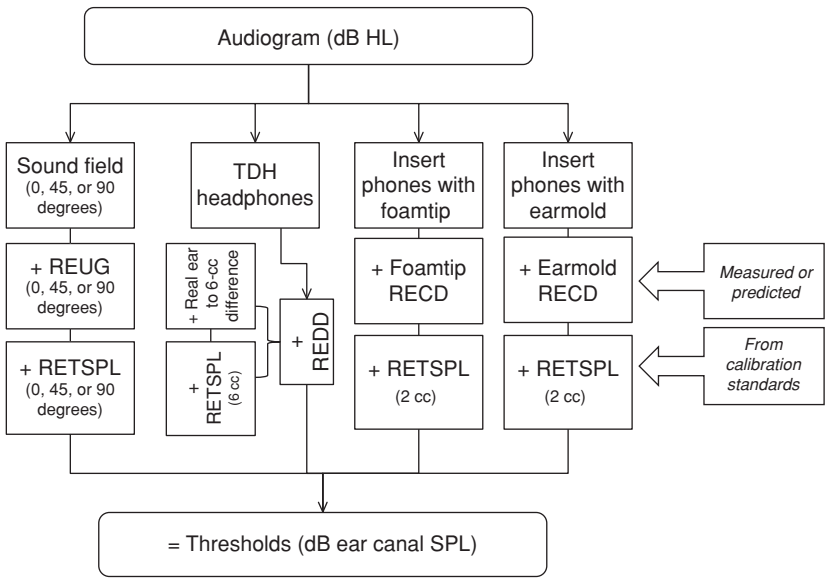


FIGURE 40.5 Computation path for converting HL assessment data to ear canal sound pressure level (SPL) across audiometric transducers. Refer to Table 40.1 for definitions of transform components.

TABLE 40.1

Summary of Transforms Used in Hearing Aid Prescription and Verification, with Acronyms and Definitions for Each

Variable	Acronym	Description
Reference equivalent threshold sound pressure level	RETSPL	The level per frequency (dB SPL) at 0 dB HL for audiometric test signals delivered to either a coupler or a field via a calibrated audiometric transducer. The coupler type and specific levels vary with audiometric transducer, and for sound field calibration also varies with head azimuth and whether or not thresholds are tested binaurally or monaurally
Real ear unaided gain	REUG	The difference (dB) between a signal in free field versus the level measured in the ear canal from the same loudspeaker. Varies with head azimuth
Real ear to dial difference	REDD	The difference (dB) between a signal in the ear canal and the audiometric dial level used to generate the signal. Typically measured with TDH headphones
Real ear to coupler difference	RECD	The difference (dB) between a signal routed to the ear and the same signal routed to the coupler. Typically measured with an insert transducer. Results vary with the specific transducer used, the coupling type (foamtip or earmold) and coupler used (HA1, HA2)
Microphone location effects	MLE	The difference (dB) between the free field levels of a signal, and the level at the hearing aid microphone. Varies with style of hearing aid (BTE, ITE, ITC, CIC)
Transform for estimating real ear output	TEREO	The sum of the RECD and MLE. This is added to 2 cc hearing aid output to predict output levels in the ear canal. Some software systems may compress the MLE portion of the TEREO because MLEs are present within the input to the hearing aid. This can also be done by filtering MLEs into the input signal during coupler-based verification

ANSI, 2010). The result is a prediction of ear canal SPL at threshold (Gustafson et al., 2013). If the HL audiogram was derived from frequency-specific ABR, a correction to estimated HL should precede the conversion to SPL. These procedures are used within the DSL method, before prediction of ULC and target calculations, so that the child's ear canal acoustics are incorporated (Bagatto et al., 2005). If the child's ear grows, the ear canal size increases and the magnitude of the ear canal resonance will change accordingly. This is intended to ensure consistent serial prescriptions, particularly if the ear canal acoustics are remeasured as the ear and earmold change over time.

Normative trends have been defined for children's real ear unaided gain (REUG; Kruger, 1987) and for both definitions of the real ear to coupler difference (RECD; Bagatto et al., 2005), but not for the real ear to 6-cc transform or real ear to dial difference (REDD; Cole and Sinclair, 1998). Within DSL, the clinician can enter measured thresholds and either RECDs or REUGs. If no measured transforms are available, age-predicted values are used. The most commonly measured transform is the RECD, which defines the difference between levels in the child's ear canal and the levels in the standard 2-cc coupler used for audiometer calibration or hearing aid measurements. Care must be taken to ensure that the correct coupling/coupler definition of the RECD is used (Gustafson et al., 2013). Appropriately developed software tools will automate this. Average adult values are applied for the REDD. Measured values may be used but clinical options for measuring this transform are rare.

Another solution is to convert the child's thresholds from raw dB HL values to equivalent adult hearing levels (Ching and Dillon, 2003). In this procedure, the child's own ear canal resonance is compared to the corresponding average adult ear canal resonance. The difference between the two is a correction. The correction is used to adjust the measured audiogram to levels that would have been measured in an average adult ear canal. These equivalent adult hearing levels can be used for computing prescriptive targets. This may ensure consistent serial prescriptions, especially when used together with repeated ear canal measurements as the child matures. This method is used by NAL-NL2 for insert phone and soundfield audiograms. As with DSL v5, age-dependent corrections for TDH headphones are not available in NAL-NL2, and the details in Table 40.1 are included in transducer-dependent corrections.

For either of the two methods just discussed, assessment of hearing thresholds with insert phones allows incorporation of ear canal acoustics and measurement of ear-specific hearing. Insert phones are the preferred audiometric transducer for this reason. One important facilitator of insert phone audiometry in older infants and young children is the child's own earmold. Fabricated to fit the ear, the soft earmold is comfortable and retains well in the ear during

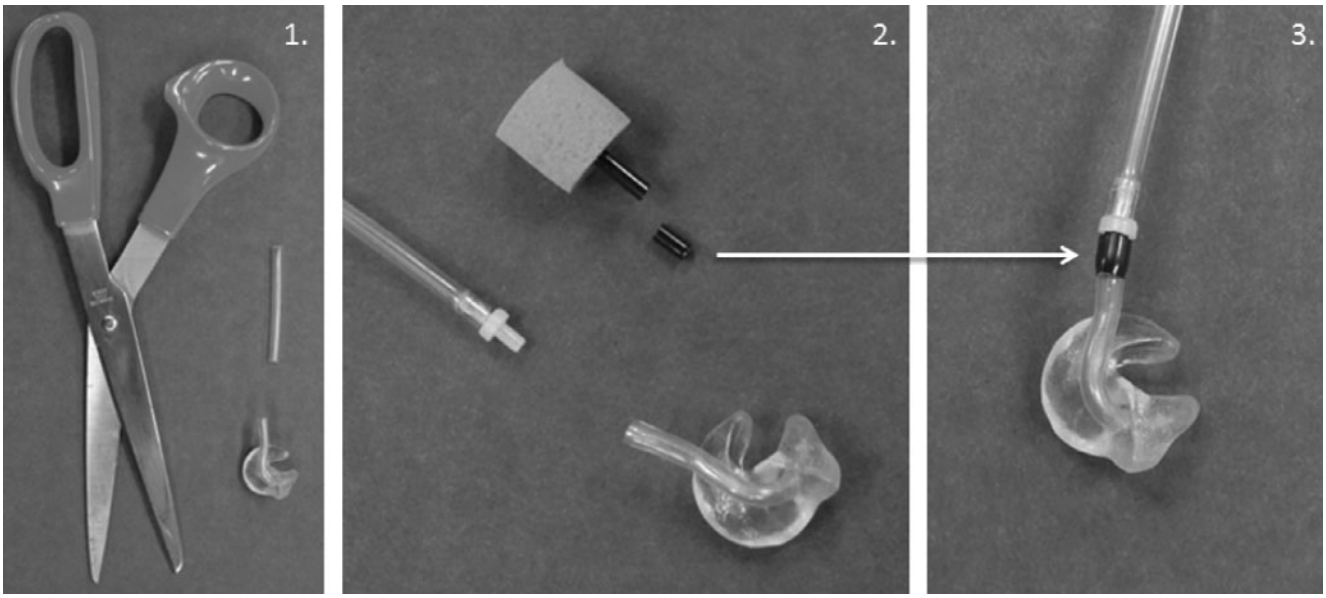


FIGURE 40.6 Illustration of a method for coupling children's custom earmolds to insert earphones for use during audiometry and RECD measurement. **Panel 1:** Trim the earmold tubing to length for daily use. **Panel 2:** Trim about 5 mm of tubing from a standard foamtip. **Panel 3:** Couple the black tubing to the end of the insert phone connector and insert it fully into the earmold tubing. The black tubing ensures a tight seal even if the earmold tubing has expanded to the size of the BTE earhook.

audiometry. The earmold may be attached to insert phones using the three-step procedure shown in Figure 40.6. Once the earmold tubing is trimmed to the correct length, a small portion of black tubing may be trimmed from an insert phone foamtip. This is placed on the end of the insert phone connector, allowing it to be firmly coupled to the earmold, even when the earmold tubing end has been stretched to a larger bore by the BTE earhook. This strategy prevents the insert phone from "popping out" of the child's ear during head turns, because the earmold fits the child's ear more snugly than a standard-sized foamtip. The insert phone plus earmold combination can then be used for both audiometry and for RECD measurement, allowing accurate incorporation of individual ear canal acoustics within assessment, prescription, and verification.

One important facilitator of incorporating ear canal acoustics is the measurement and correct definition of the RECD (Table 40.1). The RECD is measured by delivering a test signal to the ear using an insert transducer, measuring the SPL across frequencies, and then repeating this procedure in a standard coupler. The difference between the ear levels and the coupler levels is the RECD. Within software systems, clinicians are asked to define the RECD by coupling type (either a foamtip or an earmold), coupler type (HA1 or HA2), and whether the RECD was measured or should be predicted from normative data. These parameters need to be defined because the normative data for foamtip and earmold RECDs are different, especially in the high frequencies where the longer earmold tubing rolls off the high frequencies (Bagatto et al., 2005). The type of coupler also affects the RECD, because the HA2 coupler includes an earmold simulator. Measuring an RECD accurately involves the ability to correctly place a probe tube within the ear of a young child and troubleshoot it for accuracy. Placement techniques vary by age and child state. For a young, sleeping 4-month-old infant, an approximate insertion depth of 11 mm from the opening of the external auditory meatus may be a reasonable estimate (Bagatto et al., 2006). An active toddler will be seated up and will require entertainment or distraction (a video, bubbles, a toy, a mirror, a ball of tape) to occupy their interest while probe placement and RECD measurement are completed. Insertion depth guidelines from the intertragal notch for children include (1) at least 5 mm deeper than the end of their earmold; or (2) placed using otoscopy to the start of the third bend of the ear canal; or (3) placed using the 6,000-Hz notch method (Dillon, 2012). Combinations of these strategies are common: Clinicians may premark the probe relative to the earmold, then use otoscopy or notch methods to confirm correct placement in the child's ear. The clinician then measures the RECD and evaluates the result, checking for common problems: (1) Child vocalizing during measurement requiring remeasurement while the child is silent; (2) unintentional slit leak because of lack of seal by the eartip/mold causing negative RECD values in the low frequencies; and (3) shallow probe placement causing notches in the high frequencies. Correct tube placement ensures accurate estimates of ear canal pressure to about 4,000 Hz. Above this frequency, standing waves cause large changes in SPL with only minor changes in probe location. Although alternatives to measurement of ear canal SPL have

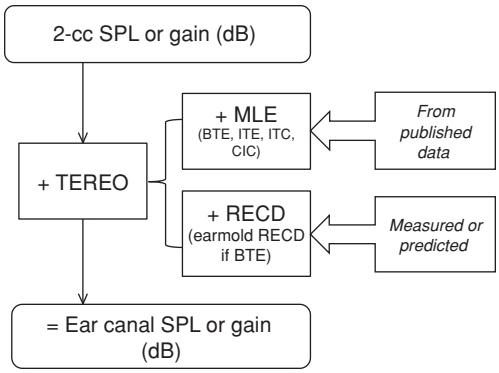


FIGURE 40.7 Computation path for converting coupler output or gain to ear canal output or gain. Refer to Table 40.1 for definitions of transform components.

promise for addressing this issue (McCreery et al., 2009), clinical tools for their implementation do not yet exist.

Verification of the child's hearing aid also incorporates individually measured or predicted ear canal characteristics. Clinically, we can choose to verify and fine-tune hearing aids using measures made on a coupler or on an ear. Coupler measures of fit to targets can incorporate the child's ear canal characteristics and the hearing aid's microphone location effects (Table 40.1) to create a reasonably accurate prediction of hearing aid output in the ear. This "coupler approach" to verification is also sometimes called "simulated real-ear verification" and uses the procedures shown in Figure 40.7 (Bagatto et al., 2005). The coupler approach is feasible for use with infants and young children because they are not required to sit up and be still in front of a loudspeaker for repeated real-ear measurements. However, the coupler approach does not account for venting effects. Real-ear evaluation is therefore preferred for older children who can comply, particularly if venting plays a major role in the fitting. Also, the fitting should be checked on-ear for feedback following the fitting on the coupler, because the full seal on the coupler does not mimic the true feedback path when worn. If significant changes are made to the hearing aid's frequency response while managing feedback, remeasurement on the coupler may be warranted to ensure accurate clinical documentation of the audibility provided from the fitting.

Real-ear evaluation can take several forms. Traditional adult-focused protocols have commonly used the real ear insertion gain (REIG) format (ANSI, 2007; Table 40.1). However, there are several problems with using the REIG as the primary measure in hearing aid fittings. Most importantly, REIG measurements are not directly comparable to auditory threshold or loudness discomfort level (LDL). Therefore, it is difficult to use REIG hearing aid responses to determine if the aided response is either inaudible or exceeding the ULC. For infants and young children, comparison between aided responses and thresholds/ULC acts as a surrogate for wearer feedback and is therefore an essential component of interpreting the adequacy of a hearing aid fitting (Figure 40.2). A more technical problem with the REIG arises because of how it is conventionally calculated. In many prescriptive, fitting, and verification systems, the target insertion gain curve is calculated using an average adult REUR, whereas the measured insertion gain curve is calculated using the client's measured real ear unaided response (REUR). If the client is lucky enough to have a "typical" REUR, this will not pose a problem. However, infants and young children are rarely in this situation, which can add substantial error to the process. Although this problem was addressed in the development of DSL v5, insertion gain tends not to be the preferred option, because clinicians prefer the more meaningful format of the SPLogram. The SPLogram is based on either direct measurement or prediction of the real ear–aided response (REAR; ANSI, 2007). Measures of the REAR have several practical challenges that should be addressed to ensure accuracy. First, patient and observer vocalizations will be included in the measurement: The clinician must take steps to capture the measure while the child and caregivers are not speaking. Second, low-level measures may be contaminated by room noise, and measures at any level may include room reflections, rendering real-ear measures less smooth in appearance than corresponding coupler measures. One solution to these problems is to locate the verification equipment within a sound booth. Third, measures in REAR format require correct probe microphone placement for accurate high-frequency measurement, particularly in comparison to the REIG procedure. Practice and use of clear markers on the probe tube to indicate target insertion depth are helpful strategies for ensuring accurate probe microphone placement.

Tailoring the Hearing Aid Options

Once the hearing assessment, ear canal acoustics assessment, prescription, fine tuning, and verification for output limiting and average speech audibility have been completed, an initial hearing aid fitting of one ear is nearly complete. Checking that both ears have loudness balance with one another is also important. Also, today's hearing aids have many options, so the next steps will include reviewing the setup of audibility for multiple input levels of speech, the setup of user controls and alerts, and the setup of multiple programs. Verification across multiple input levels of speech is recommended to ensure that the amplitude compression (and possibly other features) allows access to soft and/or distant speech and avoids excessive outputs for loud speech. Soft conversational speech is usually 50 to 55 dB SPL in overall level (Olsen, 1998), whereas loud speech is usually in the range of 75 to 80 dB SPL, with shouted speech exceeding this level. Evaluating the fit to targets across two or three levels spanning the range of 55 to 75 dB SPL is common practice. Enhanced multilevel verification is available from the SHARP software system.

User controls such as volume controls, on-off switches, battery compartment locks, and remote controls require planning and consideration of the wide range of uses and users that span infancy through the teenage years. Whereas a teen's hearing aid may provide full access to these, as would an adult's, an infant's hearing aid typically provides locked or restricted-range volume controls and locked battery compartments to prevent inadvertent adjustments and prevent battery ingestion. These decisions are typically software adjustable and can be revisited to promote the child's ownership and responsibility for their own hearing aids as their developmental status permits. Similarly, program alerts and low battery alerts may be irrelevant for a young infant's hearing aid (unless needed for use by parents during listening checks) but may be essential for older children and teens.

Multiple program profiles in hearing aids typically offer a program for listening while in a quiet place alongside programs intended for use in noise and on the phone. Programs for use with remote microphone systems are also common. When programming, it is useful to link these programs while fine tuning of the main program is taking place, so that changes to the frequency-gain response carry over to the others. Providing alternative programs for use in noise for children may improve loudness comfort (Crukley and Scollie, 2013) and improve learning through listening (Pittman, 2011), while maintaining speech recognition in noise and possibly improving it if paired with a directional or remote microphone (AAA, 2013; McCreery et al., 2012b). Audiologists can configure and reconfigure the program structure over time, adding in new features as the child's lifestyle and outcomes warrant change. For example, we can support telephone use in young children by providing hearing aids that automatically switch into a phone program when exposed to the telefield of a landline phone.

Amplification for Children with Conductive Hearing Losses

Some children have permanent or transient outer and/or middle-ear conditions that may require assistance through amplification. Transient conductive hearing losses are often because of otitis media and may or may not warrant the use of either hearing aids or remote microphone systems depending on the expected duration of the conductive loss and the child's needs. Children who have permanent conductive hearing losses, however, are considered candidates for hearing aid use in many cases (AAA, 2013). Some may be able to use air-conducted hearing aids, which can be fitted using corrections for conductive hearing loss within modern prescriptive methods (see Johnson, 2013 for comparison and review). Others may be candidates for one of several device types that provide sound via bone conduction. These include bone conduction devices, which send vibrations through the skin from a smooth hearing device that has a vibration plate. The transmission path of these devices is similar to that of a bone conduction oscillator used in audiometry. Modern bone conduction hearing aids are designed to be worn with a soft headband that can be adjusted to a comfortable tension. Surgically mediated devices that anchor either to the skull or to the middle-ear system are also available. Use of these devices in children typically requires waiting until skull growth is nearly complete, at approximately age 5. One recent study reviewed benefits of such devices in children with unilateral or bilateral permanent conductive hearing losses (deWolf et al., 2011). Procedures for prescribing, fitting, and verification of bone-anchored devices are under development and may offer systematic approaches particularly for the adjustment of the frequency response to maximize audible bandwidth (Hodgetts et al., 2011). More information on bone-anchored hearing aid technology can be found in Chapter 38.

Hearing Aid Orientation

Although a well-fitted hearing aid is an excellent first step in a child's habilitation program, it represents the point of entry into the long-term habilitation process. Hearing aids do not replace ongoing care and therapy. Nor do they maintain themselves, and thus we need to ensure that some person in the child's life is trained to do this important job. Early infant hearing aid fitting appointments are usually focused on completing a hearing aid fitting and emphasize supporting caregivers in being the enablers of routine hearing aid use by their child. Coaching sessions are necessary to train caregivers on insertion and removal of earmolds, device cleaning, battery maintenance and safety, hearing aid retention devices and strategies, and begin focusing caregiver observations on infant behaviors that may indicate responses to aided sound. Supplies and training on daily hearing aid checks are provided to help caregivers prevent or detect the regular malfunctions that may occur because of battery failure, moisture or wax blockage in the earmold, and electroacoustic malfunction. One recent survey indicates that roughly half of parents reported inadequate coaching on how to perform daily checks of children's hearing aids (Muñoz et al., 2013), so repeated coaching sessions may be necessary until caregivers feel fully competent at this important activity. Let's face it: There's a lot to learn in the early days and we don't remember it all from only one session. Daily use logs during the first weeks of hearing aid use may help in identifying problems and establishing a pattern of daily use. Typical use times in children vary with a child's age and degree of hearing loss, but on average is about 8 hours per day for the 0 to 2 age range once regular use is established (Walker et al., 2013). Supportive information about preventing hearing aid damage and provision of cost-effective and long-lasting warranties against damage are essential. Providing written information for caregivers to take home and offering a drop-in service when problems

arise may ease the burden of having to remember all of the information received the first time it is given.

Validation and Monitoring of Hearing Aid Outcome

Validation of hearing aid fittings typically follows an initial trial period, evaluates whether the hearing aid(s) change the child's ability to detect sound and understand sound, and may consider the child's perception of loudness, comfort, or ease of listening. Perceptual tests, questionnaires, or checklists are all commonly used to elicit this type of feedback, either directly from the child and/or from caregivers or others in the child's life. Collaborative team meetings with other professionals are another source of information on outcome, particularly reports on progress and performance in therapy sessions from speech-language pathologists and auditory-verbal therapists or on performance in the educational setting from teachers, educational audiologists, or their colleagues. The section below will focus on measures that the audiologist can make to monitor outcome following hearing aid fitting.

The Aided Audiogram

One common outcome measure is the *aided audiogram,* which assesses the lowest level of sound that can be detected by the child. If this is compared to the child's unaided threshold, the result is called *functional gain.* The aided audiogram and functional gain are not used for verification of hearing aid frequency responses, but are recommended as a potential outcome measure for children aged 0 to 3 years (AAA, 2013; Scollie et al., 2012). This can confirm that the child is hearing aided sound and that an improvement in sound detection has been achieved. The major limitation of aided threshold testing is that it does not assess suprathreshold use of aided sound.

SUPRATHRESHOLD PERCEPTUAL TESTING

Although speech intelligibility and loudness perception are commonly used as objective outcome measures in the adult population, their use with the pediatric population may not be possible or may require modification. For example, a cartoon-based LDL rating scale may be used to measure aided loudness in children (Crukley and Scollie, 2013) and any age-appropriate word recognition materials may be used in an aided format. For example, child-appropriate materials exist for measuring perception of sentences in noise (AAA, 2013) and word-final fricatives for assessing bandwidth of audibility (Glista and Scollie, 2012). Choice of materials requires selection of tests that are appropriate for the developmental status of the child, contain stimuli that probe the hearing aid signal processing of interest, and can be feasibly administered in the equipment available. A list of age-appropriate speech tests is available (AAA, 2013). Some clinics choose a set of materials that will be used consistently over time, which can lead to the development of local trends of expected outcomes. Another common practice is to use live voice to assess the child's ability to recognize and discriminate the Ling 6 sounds (/m/, /u/, /a/, /i/, /sh/, /s/) by the clinician—this procedure is used most often with young children, by parents as part of a daily check and by SLPs or AVTs prior to sessions to check device function. This surprisingly simple test has a number of internet videos illustrating how to perform and interpret it (e.g., Alberta Health Services, 2012).

Questionnaires, Checklists, and Standardized Tests

Structured, evidence protocols exist for using subjective reports from caregivers for outcome measurement in children who use hearing aids. Some use nonstandardized checklists, such as the longstanding battery of functional assessment tools developed at the Marion Downs Center (http://www.mariondowns.com/assessment-tools) and by Karen Anderson (https://successforkidswithhearingloss.com/tests). The UWO PedAMP protocol uses age-appropriate parent-report questionnaires to monitor the auditory development and performance of the child in real-world situations. It pairs these measures with assessments of the quality of the hearing aid fitting, allowing programs and clinicians to track hearing aid adequacy alongside outcomes. This battery was developed using a combination of evidence review and clinician review and includes typical performance ranges for the specific questionnaires (Bagatto et al., 2011). Other comprehensive batteries of standardized and laboratory measures have been developed for research purposes (http://www.uiowa.edu/~ochl/index.html, http://www.hearingcrc.org/research/projects/r462). These examples are from large-scale longitudinal studies that should be monitored for their contribution to the literature in the upcoming years.

Regardless of the specific protocol used for validation and monitoring of outcome, it should continue the pattern of family-centered care established in the choice of whether to use hearing aids described earlier in this chapter. The measurement of outcomes is not an end unto itself: The results should inform both the audiologist and the family and facilitate good dialogue on how things are going and what the next steps should be. For example, a hearing aid review session could include a review of parent-report questionnaire, a review of hearing thresholds and ear acoustics, minor adjustments to hearing aid frequency response, and a discussion of goals for the next visit. It is possible that the outcomes of the fitting and the parent-report questionnaire may be shared with parents and other professionals at a team meeting to facilitate planning and goal setting. At this appointment, the audiologist

and family may decide to revise the hearing aid options to include more or different features, perhaps because they have set the goal to introduce the child to telephone use. They may decide to increase the target number of hours of hearing aid use per day, perhaps because the child has stopped napping and is awake more hours per day. Perhaps ear impressions are taken for the next set of earmolds. These ongoing revisions are normal in the pediatric population, because change is continuous. The interested reader is referred to Chapter 44 of this book for information on ongoing intervention, education, and therapy for children with hearing loss.

CHAPTER SUMMARY AND CLINICAL CHALLENGES

Pediatric hearing aid fitting procedures combine family-centered care with evidence-based procedures to provide consistent, comfortable access to the auditory world through the use of modern hearing aid technology. The cornerstone procedures for ensuring accuracy and consistency across children and across ages are the use of high-quality assessment procedures, the use of validated prescriptions together with speech-based verification and individualized ear canal acoustics, and follow-up with monitoring of outcomes and ongoing review. Signal processing options are available for both younger and older children to ensure audibility of speech signals and to manage listening requirements in noisy places. Coupling of hearing aids to remote microphone systems in educational settings is required to overcome the effects of distance and room reverberation.

Challenges in clinical practice abound in every area of pediatric amplification. Because our technologies and fitting procedures improve regularly, the candidacy ranges for these interventions change as well. One interesting example for discussion includes whether or not to fit an older child with a traditional earmold, versus an in the ear/canal product, versus a slim-tube, versus a receiver-in-the-canal mini-BTE. If the child has a moderate hearing loss, all of these products will be considered in the child's fitting range but each offers its own cosmetic details, low-frequency response characteristics, high-frequency response characteristics, and options for coupling to accessory devices. This is a complex decision for any professional to consider, let alone to explain to families. Which styles would you recommend and why? Which option is most cost-effective? Which parameters are the most important to the family?

Another important clinical challenge comes in serving families of children who have multiple medical conditions. Comprising about one-third of our clinical caseload (Bagatto et al., 2011), these children are more likely to experience delayed onset of hearing assessment and intervention because of multiple medical interventions that may take both priority and time. In family-centered practice, audiologists will likely join teams of professionals for collaborative planning of care with one another and with the family. These meetings require that audiologists work as consultants and collaborative case managers. We are required to explain hearing, hearing loss, the child's needs for auditory access, hearing technology, and current progress to our team members in ways that they can understand. If we are new to this situation and accustomed to "talking in jargon" with our hearing professional colleagues, we may need to develop some new skills in interprofessional communication. These and many other issues identified in this chapter are worthy of probing more deeply than can be explored here. Remembering that the family is our context for service delivery is an important touchstone in navigating the challenges of service delivery in our world of rapid changes, both in the child and in technology.

FOOD FOR THOUGHT

1. What is one strategy suggested in this chapter that can be used in cases of children who have borderline audiometric candidacy for hearing aid use, but for whom oral language development is a goal?
2. What are the listening situations in which either directional microphones or remote microphone systems are expected to provide benefit?
3. What aspects of hearing aid programming are expected to change between the first hearing aid fitting in infancy versus follow up hearing aid fittings during the school age years?

REFERENCES

Agnew J. (1996) Acoustic feedback and other audible artifacts in hearing aids. *Trends Amplif.* 1, 45–82.

Alberta Health Services. (2012) The Ling Six Sound Test. Resource. Accessed online at: http://www.youtube.com/watch?v=fFnFvWu5mJo)

Alexander JM. (2013) Individual variability in recognition of frequency-lowered speech. *Seminars in Hearing.* 34, 86–109.

American Academy of Audiology. (2008) Clinical practice guidelines: remote microphone hearing assistance technologies for children. Accessed online at: http://www.audiology.org/resources/documentlibrary/documents/hatguideline.pdf

American Academy of Audiology. (2013) Clinical practice guidelines: pediatric amplification. Accessed online at: http://www.audiology.org/resources/documentlibrary/Documents/PediatricAmplificationGuidelines.pdf.

American National Standards Institute. (2007) *Methods of Measurement of Real-Ear American National Standards Institute. ANSI S3.46-1997.* New York: Acoustical Society of America.

American National Standards Institute. (2010) *Specification for Audiometers. ANSI S3.6-2010.* New York: Acoustical Society of America.

Auriemmo J, Kuk F, Lau C, Marshall S, Thiele N, Pikora M, et al. (2009) Effect of linear frequency transposition on speech recognition and production of school-age children. *J Am Acad Audiol.* 20, 289–305.

Bagatto M, Moodie S, Scollie S, Seewald R, Moodie S, Pumford J, et al. (2005) Clinical protocols for hearing instrument fitting in the Desired Sensation Level method. *Trends Amplif.* 9, 199–226.

Bagatto MP, Moodie ST, Malandrino AC, Richert FM, Clench DA, Scollie SD. (2011) The University of Western Ontario Pediatric Audiological Monitoring Protocol. *Trends Amplif.* 15, 57–76.

Bagatto MP, Seewald RC, Scollie SD, Tharpe AM. (2006) Evaluation of a probe-tube insertion technique for measuring the real-ear-to-coupler difference (RECD) in young infants. *J Am Acad Audiol.* 17, 573–581.

Boys Town National Research Hospital. (2013) The Situational Hearing Aid Response Profile version 7. Accessed online at: http://www.audres.org/rc/sharp/.

Byrne D. (1986a) Effects of bandwidth and stimulus type on most comfortable loudness levels of hearing-impaired listeners. *J Acoust Soc Am.* 80 (2), 484–493.

Byrne D. (1986b) Effects of frequency response characteristics on speech discrimination and perceived intelligibility and pleasantness of speech for hearing-impaired listeners. *J Acoust Soc Am.* 80 (2), 494–504.

Byrne D, Dillon H. (1986) The National Acoustic Laboratories' (NAL) new procedure for selecting the gain and frequency response of a hearing aid. *Ear Hear.* 7, 257–265.

Byrne D, Murray N. (1986) Predictability of the required frequency response characteristic of a hearing aid from the pure-tone audiogram. *Ear Hear.* 7, 63–70.

Byrne D, Parkinson A, Newall P. (1990) Hearing aid gain and frequency response requirements for the severely/profoundly hearing impaired. *Ear Hear.* 11, 40–49.

Ching TY, Dillon H, Hou S, Zhang V, Day J, Crowe K, et al. (2013) A randomized controlled comparison of NAL and DSL prescriptions for young children: hearing-aid characteristics and performance outcomes at three years of age. *Int J Audiol.* 52 (Suppl 2), S17–S28.[Epub ahead of print].

Ching TY, Dillon H, Katsch R, Byrne D. (2001) Maximizing effective audibility in hearing aid fitting. *Ear Hear.* 22, 212–224.

Ching TY, Newall P, Wigney D. (1997) Comparison of severely and profoundly hearing-impaired children's amplification preferences with the NAL-RP and the DSL 3.0 prescriptions. *Scand Audiol.* 26 (4), 219–222.

Ching TYC, Dillon H. (2003) Prescribing amplification for children: adult-equivalent hearing loss, real-ear aided gain, and NAL-NL1. *Trends Amplif.* 7, 1–9.

Ching TYC, Dillon H, Byrne D. (1998) Speech recognition of hearing-impaired listeners: predictions from audibility and the limited role of high-frequency amplification. *J Acoust Soc Am.* 103, 1128–1140.

Ching TYC, O'Brien A, Dillon H, Chalupper J, Hartley L, Hartley D, et al. (2009) Directional effects on infants and young children in real life: implications for amplification. *J Speech Lang Hear Res.* 52, 1241–1254.

Ching TYC, Scollie SD, Dillon H, Seewald RC. (2010a) A cross-over, double-blind comparison of the NAL-NL1 and the DSL v4.1 prescriptions for children with mild to moderately severe hearing loss. *Int J Audiol.* 49, S4–S15.

Ching TYC, Scollie SD, Dillon H, Seewald RC, Britton L, Steinberg J, et al. (2010b) Evaluation of the NAL-NL1 and the DSL v.4.1 prescriptions for children: paired-comparison judgments and functional performance ratings. *Int J Audiol.* 49, S35–S48.

Cincinnati Children's Hospital Medical Center. (2009) *Best Evidence Statement (BESt). Audiologic Management for Children with Permanent Unilateral Sensorineural Hearing Loss.* Cincinnati, OH: Cincinnati Children's Hospital Medical Center.

Clemis JD, Ballad WJ, Killion MC. (1986) Clinical use of an insert earphone. *Ann Otol Rhinol Laryngol.* 95, 520–524.

Cole WA, Sinclair ST. (1998) The Audioscan RM500 Speechmap/DSL fitting system. *Trends Amplif.* 3, 125–139.

Cornelisse LE, Seewald RC, Jamieson DG. (1995) The input/output formula: a theoretical approach to the fitting of personal amplification devices. *J Acoust Soc Am.* 97, 1854–1864.

Cox RM, Gilmore C. (1986) Damping the hearing aid frequency response: effects on speech clarity and preferred listening level. *J Speech Lang Hear Res.* 29, 357–365.

Crukley J, Scollie SD. (2012) Children's speech recognition and loudness perception with the Desired Sensation Level v5 Quiet and noise prescriptions. *Am J Audiol.* 21, 149–162.

Crukley J, Scollie SD. (2013) The effects of digital signal processing features on children's speech recognition and loudness perception. *Am J Audiol.* 23, 99–115.

DesGeorges J. (2013) Family support & cultural competence. In: Schmeltz L, ed. *The NCHAM eBook: A Resource Guide for Early Hearing Detection & Intervention.* Accessed online at: http://www.infanthearing.org/ehdi-ebook/index.html.

deWolf MJF, Hol MKS, Mylanus EAM, Snik AFM, Cremers CWRJ. (2011) Benefit and quality of life after bone-anchored hearing aid fitting in children with unilateral or bilateral hearing impairment. *Arch Otolaryngol Head Neck Surg.* 37 (2), 130–138.

Dillon H. (2012) *Hearing Aids.* 2nd ed. New York: Thieme.

Dillon H, Storey L. (1998) The National Acoustic Laboratories' procedure for selecting the saturation sound pressure level of hearing aids: theoretical derivation. *Ear Hear.* 19, 255–266.

Foley R, Cameron C, Hostler M. (2009) Guidelines for fitting hearing aids to young infants. National Health Service Newborn Hearing Screening Programme. Available online at: http://hearing.screening.nhs.uk/getdata.php?id=19254.

Glista D, Scollie SD. (2009) Modified verification approaches for frequency lowering devices. *Audiol Online.* Accessed online at: http://www.audiologyonline.com/articles/article_detail.asp?article_id=2301.

Glista D, Scollie SD. (2012) Development and evaluation of an English language measure of detection of word-final plurality markers: the University of Western Ontario Plurals Test. *Am J Audiol.* 21, 76–81.

Glista D, Scollie SD, Bagatto M, Seewald RC, Parsa V, Johnson A. (2009) Evaluation of nonlinear frequency compression: clinical outcomes. *Int J Audiol.* 48 (9), 632–644.

Glista D, Scollie SD, Sulkers J. (2012) Perceptual acclimatization post nonlinear frequency compression hearing aid fitting in older children. *J Speech Lang Hear Res.* 55, 1765–1787.

Gravel JS. (2002) Potential pitfalls in the audiological assessment of infants and young children. In: Seewald RC, Gravel JS, eds. *A Sound Foundation through Early Amplification.* Stafa, Switzerland: Phonak AG; pp 85–102.

Gustafson S, Pittman A, Fanning R. (2013) Effects of tubing length and coupling method on hearing threshold and real-ear to coupler difference measures. *Am J Audiol.* 22, 190–199.

Gustafson SJ, Pittman AL. (2010) Sentence perception in listening conditions having similar speech intelligibility indices. *Int J Audiol.* 50, 34–40.

Hodgetts WE, Hagler P, Hakansson BE, Soli SD. (2011) Technology-limited and patient-derived versus audibility-derived fittings in bone-anchored hearing aid users: a validation study. *Ear Hear.* 32, 31–39.

Jenstad LM, Seewald RC, Cornelisse LE, Shantz J. (1999) Comparison of linear gain and wide dynamic range compression hearing aid circuits: aided speech perception measures. *Ear Hear.* 20, 117–126.

Johnson EE. (2013) Prescriptive amplification recommendations for hearing losses with a conductive component and their impact on the required maximum power output: an update with accompanying clinical explanation. *J Am Acad Audiol.* 24, 452–460.

Johnstone PM, Nábělek AK, Robertson VS. (2010) Sound localization acuity in children with unilateral hearing loss who wear a hearing aid in the impaired ear. *J Am Acad Audiol.* 21, 522–534.

Joint Committee on Infant Hearing. (2007) Year 2007 position statement: principles and guidelines for early hearing detection and intervention programs. *Pediatrics.* 120, 898–921.

Joint Committee on Infant Hearing. (2013) Supplement to the JCIH 2007 Position Statement: principles and guidelines for early intervention after confirmation that a child is deaf or hard of hearing. *Pediatrics.* 131 (4): e1324–e1349.

Jones C, Launer S. (2011) Pediatric fitting in 2010: the Sound Foundations Cuper Project. In: Seewald RC, Bamford J, eds. *A Sound Foundation through Early Amplification: Proceedings of the Fifth International Conference.* Stäfa, Switzerland: Phonak; pp 187–192.

Keidser G, Dillon H, Flax M, Ching T, Brewer S. (2011) The NAL-NL2 prescription procedure. *Audiol Res.* 1 (e24), 88–90.

Kruger B. (1987) An update on the external ear resonance in infants and young children. *Ear Hear.* 8, 333–336.

Kuk F. (2013) Considerations in verifying frequency lowering. *Hear Rev.* Accessed online at: http://www.hearingreview.com/continuing-education/21339-considerations-in-verifying-frequency-lowering.

McCreery RW, Bentler RA, Roush PA. (2013) Characteristics of hearing aid fittings in infants and young children. *Ear Hear.* 34, 701–710.

McCreery RW, Brennan MA, Hoover B, Kopun J, Stelmachowicz PG. (2012) Maximizing audibility and speech recognition with nonlinear frequency compression by estimating audible bandwidth. *Ear Hear.* 20, 1–4.

McCreery RW, Pittman AL, Lewis JD, Neely ST, Stelmachowicz PG. (2009) Use of forward pressure level to minimize acoustic standing waves during probe-microphone hearing aid verification. *J Acoust Soc Am.* 126, 15–24.

McCreery RW, Venediktov RA, Coleman JJ, Leech HM. (2012a) An evidence-based systematic review of amplitude compression in hearing aids for school-age children with hearing loss. *Am J Audiol.* 21, 269–294.

McCreery RW, Venediktov RA, Coleman JJ, Leech HM. (2012b) An evidence-based systematic review of directional microphones and digital noise reduction hearing aids in school-age children with hearing loss. *Am J Audiol.* 21, 295–312.

McCreery RW, Venediktov RA, Coleman JJ, Leech HM. (2012c) An evidence-based systematic review of frequency lowering in hearing aids for school-age children with hearing loss. *Am J Audiol.* 21, 313–328.

Muñoz K, Blaiser K, Barwick K. (2013) Parent hearing aid experiences in the United States. *J Am Acad Audiol.* 24, 5–16.

Olsen WO. (1998) Average speech levels and spectra in various speaking/listening conditions: a summary of the Pearson, Bennett, & Fidell (1977) report. *Am J Audiol.* 7, 21–25.

Parsa V, Scollie SD, Glista D, Seelisch A. (2013) Nonlinear frequency compression: effects on sound quality ratings of speech and music. *Trends Amplif.* 17, 54–68.

Pittman AL. (2008) Short-term word learning rate in children with normal hearing and children with hearing loss in limited and extended high-frequency bandwidths. *J Speech Lang Hear Res.* 51, 785–797.

Pittman AL. (2011) Age related benefits of digital noise reduction for short-term word learning in children with hearing loss. *J Speech Lang Hear Res.* 54, 1448–1463.

Pittman AL, Stelmachowicz PG. (2003) Hearing loss in children and adults: audiometric configuration, asymmetry, and progression. *Ear Hear.* 24 (3), 198–205.

Quar TK, Ching TYC, Newall P, Sharma M. (2013) Evaluation of real-world preferences and performance of hearing aids fitted according to the NAL-NL1 and DSL v5 procedures in children with moderately severe to profound hearing loss. *Int J Audiol.* 52, 322–332.

Ricketts TA, Galster JA. (2008) Head angle and elevation in classroom environments: implications for amplification. *J Speech Lang Hear Res.* 51 (2), 516–525.

Ricketts TA, Galster JA, Tharpe AM. (2007) Directional benefit in simulated classroom environments. *Am J Audiol.* 16 (2), 130–144.

Ricketts TA, Picou EM. (2013) Speech recognition for bilaterally asymmetric and symmetric hearing aid microphone modes in simulated classroom environments. *Ear Hear.* 34 (5), 601–609.

Sabo DL, Paradise JL, Kurs-Lasky M, Smith CG. (2003) Hearing levels in infants and young children in relation to testing technique, age group, and the presence or absence of middle-ear effusion. *Ear Hear.* 24 (1), 38–47.

Scollie S, Glista D. (2011) Digital signal processing for access to high frequency sounds: implications for children who use hearing aids. *ENT Audiol News.* 20 (5), 83–87.

Scollie S, Glista D, Tenhaaf Le Quelenec J, Dunn A, Malandrino A, et al. (2012) Ling 6 stimuli and normative data for detection of Ling-6 sounds in hearing level. *Am J Audiol.* 21 (2), 232–241.

Scollie SD, Ching TYC, Seewald R, Dillon H, Britton L, Steinberg J. (2010a) Evaluation of the NAL-NL1 and DSL v4.1 prescriptions for children: preference in real world use. *Int J Audiol.* 49, S49–S63.

Scollie SD, Ching TYC, Seewald RC, Dillon H, Britton L, Steinberg J, et al. (2010b) Speech perception and loudness ratings of children who used hearing aids fitted with the DSL v.4.1 and the NAL-NL1 prescriptions. *Int J Audiol.* 49, S26–S34.

Scollie SD, Seewald RC, Cornelisse L, Moodie S, Bagatto M, Laurnagara D, et al. (2005) The Desired Sensation Level multistage input/output algorithm. *Trends Amplif.* 9, 159–197.

Scollie SD, Seewald RC, Moodie KS, Dekok K. (2000) Preferred listening levels of children who use hearing aids: comparison to prescriptive targets. *J Am Acad Audiol.* 11, 230–238.

Seewald RC. (1991) Hearing aid output limiting considerations for children. In: Feigin J, Stelmachowicz PG, eds. *Pediatric Amplification: Proceedings of the 1991 National Conference.* Omaha, NE: Boys Town National Research Hospital Press, pp 19–35.

Seewald RC, Ross M, Spiro MK. (1985) Selecting amplification characteristics for young hearing-impaired children. *Ear Hear.* 6 (1), 48–53.

Sininger YS, Grimes A, Christensen E. (2010) Auditory development in early amplified children: factors influencing auditory-based communication outcomes in children with hearing loss. *Ear Hear.* 31 (2), 166–185.

Snik AF, Stollman MH. (1995) Measured and calculated insertion gains in young children. *Br J Audiol.* 29 (1), 7–11.

Snik AF, van den Borne S, Brokx JP, Hoekstra C. (1995) Hearing-aid fitting in profoundly hearing-impaired children. Comparison of prescription rules. *Scand Audiol.* 24 (4), 225–230.

Stelmachowicz PG, Pittman AL, Hoover B, Lewis D, Moeller MP. (2004) The importance of high-frequency audibility in the speech and language development of children with hearing loss. *Arch Otolaryngol Head Neck Surg.* 130 (5), 556–562.

Tharpe AM. (2008) Unilateral and mild bilateral hearing loss in children: past and current perspectives. *Trends Amplif.* 12, 7–15.

van Buuren RA, Festen JM, Houtgast T. (1996) Peaks in the frequency response of hearing aids: evaluation of the effects on speech intelligibility and sound quality. *J Acoust Soc Am.* 39, 239–250.

Waldman D, Roush J. (2009) *Your Child's Hearing Loss: A Guide for Parents.* 2nd ed. San Diego, CA: Plural.

Walker EA, Spratford M, Moeller MP, Oleson J, Ou H, Roush P, et al. (2013) Predictors of hearing aid use time in children with mild-to-severe hearing loss. *Lang Speech Hear Serv Sch.* 44, 73–88.

Wolfe J, John A, Schafer E, Nyffeler M, Boretzki M, Caraway T. (2010) Evaluation of non-linear frequency compression for school-age children with moderate to moderately severe hearing loss. *J Am Acad Audiol.* 21 (10), 618–628.

Wolfe J, John A, Schafer E, Nyffeler M, Boretzki M, Caraway T, et al. (2011) Long-term effects of non-linear frequency compression for children with moderate hearing loss. *Int J Audiol.* 50 (6), 396–404.

Wright DC, Frank T. (1992) Attenuation values for a supra-aural earphone for children and insert earphone for children and adults. *Ear Hear.* 13 (6), 454–459.

CHAPTER 41

Hearing Aid Fitting for Adults: Selection, Fitting, Verification, and Validation

Michael Valente and Maureen Valente

INTRODUCTION

Audiologists have a responsibility to achieve hearing aid fittings that provide the best possible "benefit" and "satisfaction." Surprisingly, these goals can often contradict each other. For example, patients commonly request hearing aids providing the best performance in noise (i.e., benefit), but this is immediately followed by the need for the hearing aids to be "invisible" (i.e., satisfaction). At this point, a conflict can present itself because the technology capable of providing the greatest benefit in noise (i.e., directional microphone and/or personal listening devices) may be in conflict with the desire for "invisibility" (i.e., completely-in-the-canal (CIC) or a Lyric® hearing aid or similar hearing aid that is deeply seated in the ear canal). The audiologist must either educate the patient on the technologies available that provide the best benefit in noise and "convert" the patient to this technology, or simply surrender to the patient's demand for cosmetic appeal knowing the fitting may not provide the best benefit in noise. Ultimately, this patient may be fit with bilateral behind-the-ear (BTE) hearing aids with directional microphones at one clinic, whereas the same patient could have been fit with bilateral smaller hearing aids at another clinic. One fit may satisfy the goal of hearing better in noise, whereas the other fit may satisfy the cosmetic concerns of the patient.

To address the concern of "cosmetics," BTE open-fit hearing aids with a directional microphone using either a thin tube or a wire connected to a receiver placed in the ear canal are now readily available as are hearing aids deeply seated in the ear canal. Kuk and Keenan (2005) and Valente and Mispagel (2008) reported on the performance of open-fit BTEs using directional microphones. Kuk and Keenan (2005) evaluated 8 participants with one manufacturer's open-fit BTE, whereas Valente and Mispagel (2008) evaluated 26 participants with another manufacturer's open-fit BTE. Kuk and Keenan (2005) examined the Hearing in Noise Test (HINT) reception threshold for sentences (RTS in decibels). The HINT sentences were presented at 0° azimuth whereas HINT noise was presented at 75 dB SPL with loudspeakers at 90°, 180°, and 270°. Valente and Mispagel (2008) measured RTS with HINT sentences presented at 0° and uncorrelated restaurant noise presented at 65 dBA from eight loudspeakers set 45° apart. Results from both studies reported no significant differences between unaided and aided omnidirectional conditions, but a directional benefit of slightly less than 2 dB relative to omnidirectional and unaided performance. These results suggest that when communicating in a noisy listening environment, patients require at least a directional microphone with an open-fit hearing aid if aided performance is to be better than unaided performance. Assuming a 10% per decibel improvement in sentence intelligibility of HINT sentences, the directional microphone improved performance in noise, re: Unaided and omnidirectional performance, by approximately 20%.

The above-mentioned examples illustrate one of the issues confronting audiologists when selecting hearing aids for their patients. To help the reader achieve the "best fit" for his/her adult patients, this chapter will provide a comprehensive overview of clinical procedures audiologists can implement when selecting, fitting, verifying, and validating hearing aid fittings for adults. The procedures will be presented chronologically in the manner that hearing aids are typically selected, fit, verified, and validated by us.

This process often requires four visits at our clinics. The *first* visit includes a comprehensive case history and audiologic evaluation, counseling, and informally assessing patient's motivation toward amplification. The *second* visit may include loudness judgments for puretones measured in dB SPL near the tympanic membrane, extensive counseling on fitting options, taking impressions of the ear canal(s), and obtaining an unaided outcome measure (e.g., Client Oriented Scale of Improvement (COSI), Dillon et al., 1997; or Washington University questionnaire (Wash U)) (Figure 41.1). If time allows, the first and second visits can be collapsed into a single visit. The *third* visit includes verifying real-ear performance of the hearing aids and obtaining aided loudness judgments for speech. This visit also includes counseling on the use and care of the hearing aids

WASHINGTON UNIVERSITY QUESTIONNAIRE

Patient _____ **Date** _____ **Aid** _____ **Ear(s)** _____ **Age** _____

[X] Unaided [O] Own Aid(s) [+] New Aid(s)

	Always	Often	Sometimes	Rarely	Never	N/A
Difficulty at Home						
Communicate with spouse	[X]	[ ]	[O]	[ ]	[+]	[ ]
Family members/friends	[ ]	[X]	[O]	[ ]	[+]	[ ]
Children	[X]	[ ]	[ ]	[O]	[ ]	[ ]
TV, audio equipment	[X]	[O]	[ ]	[+]	[ ]	[ ]
Telephone	[X]	[ ]	[O]	[+]	[ ]	[ ]
Difficulty at work						
Telephone	[X]	[O]	[ ]	[+]	[ ]	[ ]
One-on-one in noisy situations	[X]	[O]	[ ]	[+]	[ ]	[ ]
Small meetings	[ ]	[X]	[ ]	[O]	[ ]	[ ]
Large meetings with speaker greater than 12 feet	[X]	[ ]	[O]	[ ]	[+]	[ ]
Difficulty in social situations						
Family gatherings	[ ]	[X]	[O]	[+]	[ ]	[ ]
Noisy restaurant	[X]	[O]	[ ]	[+]	[ ]	[ ]
House of worship	[ ]	[ ]	[ ]	[ ]	[ ]	[X]
Theater	[X]	[ ]	[O]	[+]	[ ]	[ ]
Party	[X]	[O]	[ ]	[ ]	[+]	[ ]

Comments/observations: _____

FIGURE 41.1 Example of the Washington University questionnaire.

and the method of coupling the hearing aids to the ear(s). In addition, the patient is contacted within two working days to determine his/her initial experiences to the fitting. If problems are present, the patient is immediately scheduled to return to the clinic so the issue(s) can be resolved. The *final* visit, scheduled approximately 30 days after the initial fitting, includes obtaining the "aided" measure using the COSI or Wash U questionnaire. Recently, we added EARtrak® as another outcome measure of hearing aid usage to obtain greater information about the satisfaction of our patients in varying listening situations (11 situations), hearing aid features (12 features), and service providers (7 situations) (Figure 41.2). This computer-based outcome measure is accessed by our patients via our website (http://hearing.wustl.edu) and has proven to be a very valuable tool. This visit may also include fine-tuning the hearing aids in response to the patient's assessment of performance during the 4-week trial period. Finally, the patient is provided a Zephry® Dry & Store to help remove moisture from the hearing aids and reduce the number of patient visits related to the problems caused by excessive moisture.

PREFITTING DATA (FIRST VISIT)

Comprehensive Audiometry and Assessing Motivation

During this visit, a comprehensive case history is taken and puretone and speech audiometry are assessed in the conventional manner. Immittance tests are administered, including tympanometry, acoustic reflex thresholds with contralateral and ipsilateral stimulation, and reflex decay if the results from puretone and/or speech audiometry warrant ruling out conductive, mixed, or retrocochlear pathology. If the results from these measures suggest that amplification is appropriate, then candidacy for amplification is suggested to the patient. The patient is counseled to schedule an appointment with his/her physician to obtain

Your responses for Question 12 - Satisfaction with Listening Situations					
With one person	**In small groups**	**In large groups**	**Outdoors**	**At a concert or movie**	**At church or at a lecture**
Very satisfied	Satisfied	Neutral	Satisfied	Satisfied	Satisfied
Watching TV	**In a car**	**At work**	**On the phone**	**At a restaurant**	
Very satisfied	Neutral	Not relevant	Very satisfied	Satisfied	

Your responses for Question 13 - Satisfaction with Hearing Aid Features					
Overall fit/ comfort	**Ease of adjusting volume**	**Visibility**	**Cleaning frequency**	**Ongoing expense**	**Battery life**
Satisfied	Satisfied	Satisfied	Satisfied	Neutral	Satisfied
Reliability	**Clarity**	**Sound of own voice**	**Localisation**	**Loud sounds**	**Whistling**
Satisfied	Satisfied	Satisfied	Satisfied	Neutral	Very satisfied

Your responses for Question 14 - Satisfaction with Service Provider			
Professionalism of clinician	**Friendliness of staff**	**Patience of clinician**	**Explanations given**
Very satisfied	Very satisfied	Very satisfied	Very satisfied
Amount of time spent	**Cleanliness and appearance of office**	**Service after purchase**	
Very satisfied	Very satisfied	Very satisfied	

FIGURE 41.2 Example of a patient response to the EARtrak® web-based outcome measure to obtain greater information about the satisfaction of this patient with 11 listening situations, 12 hearing aid features, and 7 questions related to the services provided by his/her audiologist.

medical clearance for amplification. If the audiologist is questioned about the need for a medical examination, it is best to inform them that it is in the patient's best interest, but the patient has the right to sign a medical waiver. This signed form is placed in the patient's electronic medical record (EMR) and later attached to the fee ticket as required by law in the state where we reside. It is crucial during this initial visit that audiologists informally assess the patient's motivation toward amplification. We believe that dispensing hearing aids to unmotivated patients can do significantly more harm than good. If the experience with amplification is unsatisfactory, these feelings of dissatisfaction will probably be transferred to friends and/or family members who may also be considering amplification. Therefore, it would not be surprising if these other "potential" patients no longer pursue amplification because of the negative expression of their friend or family member. Dispensing hearing aids to an unmotivated patient is strongly discouraged, because this practice may lead to far more than simply a "return for credit." When counseling unmotivated patients, we suggest this may not be the best time for them to consider amplification. Rather, we urge such patients to make an appointment when they feel more motivated and the hearing loss is negatively impacting quality of life (QOL).

HEARING AID EVALUATION (SECOND VISIT)

Following the audiologic evaluation, a hearing aid evaluation (HAE) is scheduled. The HAE is the appointment dedicated to discussing all aspects of hearing aid use, hearing aid technology, and hearing aid styles. For the HAE, the patient is encouraged to bring family members. During this visit, the audiologist will

1. Counsel the patient, once again, on the results from the audiologic evaluation because several weeks may have passed since the initial audiologic examination. Experience has shown that patients may not accurately recall the counseling that was completed at the initial audiologic evaluation and a review is often very helpful.
2. Counsel the patient on anatomy and physiology of the ear using figures from a wide variety of resources. This review allows the patient to have a greater appreciation of how the hearing loss is related to anatomical changes in the ear.
3. Calculate the patient's Speech Intelligibility Index (SII) using free software available at www.sii.to. This is a very helpful tool to provide a greater understanding of the impact of the patient's hearing loss on the audibility of speech. Calculation of the SII for unaided and aided performance is also available with most real-ear analyzers.
4. Counsel the patient, when appropriate, on the advantages of bilateral amplification for improved sound quality, hearing in background noise, and localization. This is described in greater detail in a section that follows.
5. Counsel the patient on the realistic expectations of amplification. Aided performance in noise should be better than unaided performance in noise, but aided performance in noise will probably not be as good as aided performance in quiet. It is emphasized that even listeners with normal hearing perform poorer in noise than quiet. This is described in greater detail in a section that follows.

6. Demonstrate different hearing aid styles. This may include custom in-the-ear (ITE), in-the-canal (ITC), CIC, conventional BTE instruments, BTE using a thin tube to direct the amplified sound to the ear canal, or BTE using a wire connected to the receiver placed in the ear canal. The patient may also be shown examples of custom earmolds, or manufacturer domes and custom earmolds that can be used to deliver the amplified sound to the ear canal.
7. Counsel the patient on different features of hearing aids such as volume control, program button, remote control, and directional microphones. Also, discuss wireless communication to cellular and landline telephones, computers, televisions, hearing-assistive technology (HAT), and a wide variety of other auditory inputs.
8. Counsel the patient on the importance of the telecoil (t-coil) for communication on the telephone and HAT. Emphasize that with current hearing aids it is possible for the hearing aid to automatically switch to the t-coil when a telephone is placed near the ear. Also, with some current hearing aids, the signal from the telephone placed over one ear can be heard, automatically, in both hearing aids.
9. Counsel the patient on the relationship between the "levels" of hearing aid technology and the associated charges. This is described in greater detail in a section that follows, but these differences in "levels" include
   a. The number of available "bands" which is related to the flexibility of the audiologist to "match" a validated prescriptive target using real-ear measures (REM).
   b. The number of "channels" which is the effectiveness of the signal processing in the hearing aid(s) to process environmental noise and feedback as well as the effectiveness of the directional microphones.
   c. The number of automatic features.
   d. Warranty for repair (1 to 3 years).
   e. Warranty for loss and damage (1 to 3 years).
10. Administer a questionnaire to assess what the patient may expect his/her hearing aid(s) to provide. For example, a patient may want the hearing aids to improve the ability to recognize (a) female and child talkers, (b) speech while watching television, (c) speech in noisy listening situations, and (d) communication on the telephone. This information will assist the patient to establish realistic goals. This also is described in greater detail in a section that follows.
11. Measure the patient's loudness discomfort levels (LDLs) to determine when signals are "loud, but OK." These LDLs will serve as the target when measuring the output of hearing aids to a loud input during the hearing aid fitting (HAF). This is described in greater detail in the next section.
12. Take impressions of one or both ear canals to order custom earmolds for BTEs or custom hearing aids. On the other hand, some hearing aids may be coupled to the ear using noncustom "domes." Again, this is described in greater detail in a section that follows.

Real-Ear Measures of Loudness Discomfort

One of the most important facets of successful hearing aid fittings is to ensure that the amplified signal does not exceed the level where the patient reports the amplified sound to be "loud, but OK." Munro and Patel (1998) reported that if the measured real-ear saturation response with a 90-dB SPL puretone sweep ($RESR_{90}$) is below the individually measured LDL, then participants did not report "real-life" listening to be uncomfortably loud. This contrasted with participants whose $RESR_{90}$ exceeded the individually measured LDL. Jenstad et al. (2003) reported that the primary complaint of patients newly fit with hearing aids, who returned to the clinic for fine-tuning, was the amplified sound was too loud.

As part of the HAE, measures can be made to determine the intensity level (in dB SPL measured near the tympanic membrane) where sound is "loud, but OK." To accomplish this, a probe-tube, attached to a probe microphone, is placed in the ear canal approximately 4 to 6 mm from the tympanic membrane to directly measure the dB SPL near the tympanic membrane. To assure that the end of the probe-tube is within 4 to 6 mm from the tympanic membrane of an adult male patient, the probe-tube is marked 30 mm from the tip (26 mm for an adult female). This mark is placed at the intratragal notch of the outer ear and taped into position.

Using this method, calibrated TDH-series or ER-3A insert earphones are connected to the earphone output of a calibrated audiometer (ANSI-2004) and placed in the ear canal along with the probe-tube from the real-ear analyzer (Figure 41.3). At this point, a continuous puretone at 500 to 4,000 Hz in octave and mid-octave intervals is presented to the patient. The patient is asked to judge the loudness of the signal with choices of "very soft," "soft," "comfortable, but

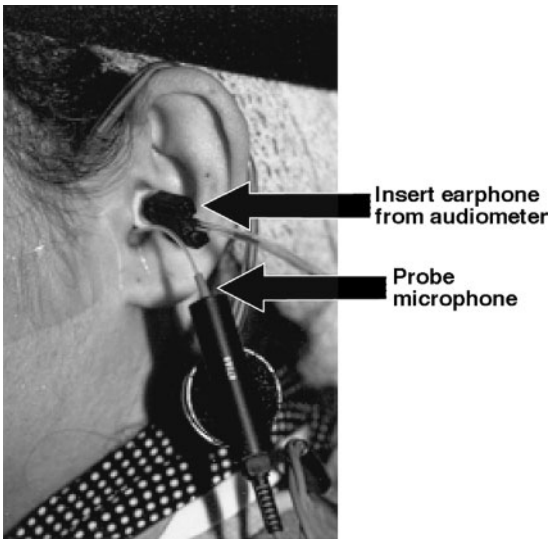


FIGURE 41.3 Placement of a probe-tube, probe microphone, and insert earphone for measuring individual LDLs.

Washington University School of Medicine
Loudness Measures

Patient Name:_____ **DOB:**_____
Date:_____ **Audiologist:**_____

Right Ear

	HL	SPL	REDD	ANSI-1989
500 Hz	85	96	11	12.0
1 kHz	85	95	10	9.0
2 kHz	90	105	15	15.0
3 kHz	95	106	11	15.5
4 kHz	95	105	10	13.0

FIGURE 41.4 Example of a report form used to document a patient's measured LDL in dB HL and dB SPL and REDD at 500, 1,000, 2,000, and 4,000 Hz. The data to the right are the average REDD. This form is placed in the patient's chart for future reference when measuring the $RESR_{90}$.

slightly soft," "comfortable," "comfortable, but slightly loud," "loud, but OK," and "uncomfortably loud."

Using an ascending procedure, the audiometer attenuator is increased in 5 dB steps to determine the intensity level (in dB HL and SPL) where the patient consistently reports that the puretone signals are "*loud, but OK.*" This level is read in dB SPL from a video monitor of the real-ear analyzer and is recorded as the patient's LDL_{spl} at each test frequency and these values are placed in the patient's chart (Figure 41.3). In Figure 41.4, the LDL in dB HL is placed in the first column (85 to 95 dB HL for this patient) and the measured LDL_{spl} is placed in the next column (96 to 106 dB SPL for this patient). The difference between these two measures is the real ear to dial difference (REDD) and these values are placed in the third column (10 to 15 dB for this patient). The last column to the right reports the average REDD. As part of the hearing aid fit at a subsequent visit, the audiologist uses the real-ear analyzer and measures the output (in dB SPL) from the hearing aid at the tympanic membrane in response to a puretone sweep presented at 90 dB SPL (i.e., $RESR_{90}$; Figure 41.5). On completing the $RESR_{90}$,

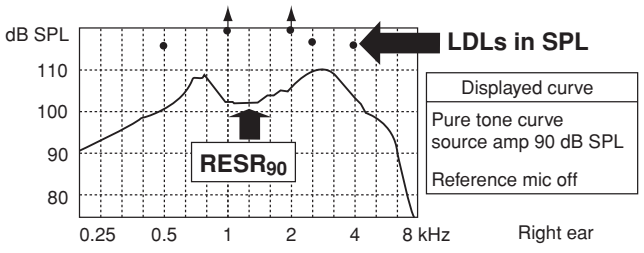


FIGURE 41.5 Example of the measured $RESR_{90}$ (*solid curve*) below the individually measured LDL in dB SPL (*dots*). The ↑ *symbol* at 1,000 and 2,000 Hz reports that the measured LDL in dB SPL, in this case, was greater than 120 dB SPL (*upper line on the ordinate*).

the audiologist can determine the relationship between the measured $RESR_{90}$ and the previously measured LDL_{spl}. The audiologist must verify that the $RESR_{90}$ is below the measured LDL_{spl}; if so, this suggests the patient will not judge loud environmental sounds to be uncomfortably loud. As can be seen in Figure 41.5, this goal has been achieved for all test frequencies.

Patient Counseling on Realistic Goals

It is important that patients obtain information that will portray a realistic "picture" of the benefits they can expect from amplification; this may vary somewhat across patients. With these goals in mind, the patient is counseled on some or all of the following points:

a. Performance with hearing aids in "quiet" will be significantly better than performance without the hearing aids. At this point, the patient is reminded that the final judgment of "*significant benefit*" does not rely on the outcome of the numerous measures described in this chapter. Rather, the final decision concerning benefit lies exclusively with the patient.
b. Performance with the hearing aids in "noise" must be significantly better than the performance without the hearing aids (at least for patients with mild-to-moderate hearing loss). Achieving this goal is very important because most patients seek amplification because they experience difficulty recognizing speech in noise. Currently, it is our experience that fitting hearing aids incorporating directional microphones significantly increases the likelihood of achieving this goal (Kochkin, 2000; Valente et al., 1995).
c. Patients should not expect performance in "noise" to be as satisfactory as performance in "quiet." We explain that even listeners with normal hearing do not recognize speech as well in a "noisy restaurant" as they do in a "quiet living room." Therefore, the patient is reminded there is no reason to expect his/her hearing aids to perform as well in "noise" as in "quiet."
d. Input signals of a "soft" input level (i.e., 40 to 50 dB SPL) should be perceived as "soft, but audible."
e. Input signals of an "average" input level (i.e., 60 to 70 dB SPL) should be perceived as "comfortable."
f. Input signals of a "loud" input level (i.e., 80 dB SPL or greater) should be perceived as "loud, but not uncomfortable."
g. Earmolds or custom shells must be comfortable.
h. The patient's voice should not sound as if he/she is speaking at the bottom of a barrel (i.e., occlusion effect).
i. The patient should not experience acoustic or suboscillatory feedback.
j. It is common for patients purchasing "new" hearing aids to expect to hear as well as their normal-hearing friends in a noisy restaurant. To address this expectation, we ask

the patient to mentally "score" the percent of the conversation understood from his/her "normal"-hearing friend(s) when communicating in noisy situations. Next, the patient then asks his/her normal-hearing friend(s) what percent of the patient's conversation did the friend understand. It has been our experience that very often the difference between these two "scores" is often not as great as the patient previously predicted they might be. The patient then has a greater appreciation that even friends with "normal"-hearing experience significant difficulty in noisy environments. After completing this simple exercise, the patient typically has a better "feel" for the benefit achieved with the aids and his/her expectations are more realistic. Moreover, audiologists should counsel that even greater benefit in noise can be achieved with HAT (Lewis et al., 2004) and auditory rehabilitation.

k. If these expectations are not fulfilled, the patient must return to the office so that the hearing aids can either be readjusted or replaced with another form of technology. If these problems cannot be addressed in a satisfactory manner, then the hearing aids must be returned for credit. We counsel patients that it is not our intent for these hearing aids to be another set of hearing aids lying in a dresser drawer.

Bilateral versus Monaural Amplification

Next, and when appropriate, we counsel on the advantages of bilateral amplification. At our clinic, we strongly promote bilateral fittings to eliminate the *head shadow effect* and to yield a more "natural" amplified sound. During the 30-day trial period, patients are counseled to use the hearing aids equally as a monaural and bilateral fit. We want the patient to assess whether or not the addition of the second hearing aid proves beneficial. Using this approach, approximately 85% of patients in our practice have decided that the second hearing aid provides significant benefit and to keep the second hearing aid. The remaining 15% feel that performance was equal with one hearing aid in either ear in comparison to two. These relatively few patients decide to return one hearing aid for credit. Using a different strategy, we found that if the initial monaural fitting was found to be successful, only 15% of our patients returned to convert their monaural fitting to bilateral. For these reasons, when the audiometric results and case history profile are appropriate, we typically fit bilaterally at the time of the initial fitting.

Hearing Aid Style, Technology, and Signal Processing

We counsel the patient on

a. Style: Conventional BTE, BTE with a receiver in the ear canal (RIC), BTE with a thin tube, ITE, ITC, CIC, and a "Lyric®-type" device described earlier.
b. Microphone technology: Omnidirectional, fixed directional, adaptive directional, automatic adaptive directional, and automatic multichannel adaptive directional microphone. An adaptive directional microphone is designed to automatically apply the most effective polar design depending on the azimuth and input level of the noise. A cardioid design will automatically be in place when the noise is directly behind and a bidirectional polar design will be in place if the noise moves from the back directly to the side. An automatic adaptive directional microphone is similar with the exception that the microphones will automatically change from directional to omnidirectional when the environment switches from noisy to quiet. The patient is not required to press a switch to change the hearing aid from omnidirectional to directional and vice versa. When in the directional mode as described above, the microphone is fully adaptive. Thus, an automatic adaptive microphone can theoretically provide the greatest benefit if patient convenience is a consideration.
c. Various benefits of digital signal processing (DSP): Feedback management, expansion to reduce/eliminate circuit noise for those patients with normal low-frequency hearing, noise reduction (NR) for improved comfort in noisy listening situations, and multiband processing for greater precision in shaping the frequency-gain/output response.
d. Telephone communication: Programmable t-coil, wireless Bluetooth communication, automatic t-coil, and bilateral t-coil to a telephone signal placed at one ear.
e. HAT: Our facility has a fully stocked HAT display in two counseling rooms. In these rooms, patients can see and experience the benefits provided by such devices. In these rooms, there are infrared television listening devices, amplified telephones, wireless Bluetooth communication systems, FM devices, alarm clocks, and other devices. Also, both counseling rooms are induction looped so patients may experience the benefits of looping using the t-coil(s).
f. When applicable, we cover special applications:
   1. Wireless CROS/BICROS.
   2. Auditory osseointegrated system (AOIS), SoundBite, transcranial CROS, and TransEar for patients with unilateral hearing loss (UHL).

For additional information on the various types of hearing aid technologies and signal processing, the reader is referred to Chapter 38.

Counseling Goals

The overall goals of this extensive counseling are threefold. First, the audiologist must determine a hearing aid fitting that is the most appropriate for the patient in terms of addressing (a) the patient's goals (i.e., "hear better in noise," "improved communication on the telephone"), (b) style

(BTE, ITE, ITC, CIC, deeply seated style such as the Lyric®), and (c) technology (number of channels and bands, feedback management, adaptive directional microphone, expansion, cost, and other features). Second, the authors believe that a well-informed patient will more likely result in greater user satisfaction than an uninformed or underinformed patient. Third, the patient needs to feel that he/she is as much a part of the decision in selecting the "appropriate" hearing aids as is the audiologist. The final decision of which hearing aids to pursue is based on the interaction of such issues as

a. The magnitude, configuration, and symmetry of the hearing loss.
b. Listener lifestyle and the demands on communication.
c. The patient's need or demand for the "best and/or most convenient" signal processing technology to reduce the deleterious effects of background noise. We cannot remember a single patient reporting he/she hears better in "noise" than in "quiet." Therefore, our primary goal is to counsel the patient on the available technology that has been proven via research to improve the recognition of speech in noise.
d. Importance of communication on the telephone and the sensitivity of the telecoil.

Unaided Outcome Measure

It is imperative that a comprehensive hearing aid fitting process must include some outcome measure assessing the patient's impressions of hearing aid benefit (i.e., the degree of perceived improvement in the aided vs. unaided listening conditions). Numerous self-assessment questionnaires are available to consider. These include the Communication Profile of Hearing Impairment (Erdman and Demorest, 1990), Hearing Performance Inventory (Giolas et al., 1979), Hearing Handicap Inventory for the Elderly (Newman et al., 1990), Abbreviated Profile of Hearing Aid Benefit (APHAB) (Cox and Alexander, 1995), Glasgow Hearing Aid Benefit Profile (Gatehouse, 1999), Satisfaction with Amplification in Daily Life (Cox and Alexander, 2001), and COSI (Dillon et al., 1997). Currently, APHAB and COSI are available on the latest HIMSA module as well as several manufacturers' NOAH modules.

To satisfy the above-mentioned goal and as mentioned earlier, we ask the patient to initially complete the "unaided" portion of either the COSI or the Washington University questionnaires. With the COSI, the patient generates as many as five specific expectations (goals) he/she wants to achieve with the hearing aids. For example, "hearing better in noise," "hearing better in church," "hearing better on the telephone," or "hearing better while communicating around the dinner table." By being "forced" to focus on his/her goals, the counseling can focus on the hearing aid technology that will most likely allow the patient to achieve his/her goals. In addition, the responses allow the clinician to gain further insight into the patient's expectations and, in turn, allow the clinician to determine if these expectations are realistic and attainable. After the patient has had the opportunity to wear the hearing aids for 4 weeks, he/she is asked to check the box on the COSI form that asks, "to what degree have the hearing aids changed your expectations?" The choices are "worse," "no difference," "slightly better," "better," or "much better." In addition, the patient is asked to check the correct box on the COSI form completing the statement "if, by using the hearing aids, you can hear satisfactorily: "Hardly ever (10%)," "occasionally (25%)," "half the time (50%)," "most of the time (75%)," or "almost always (95%)." The COSI has proven to be a very useful clinical tool, because it is easy to use and the goals are set by the patient rather than by the clinician.

With the Washington University questionnaire (Figure 41.1), the patient is asked to mark (placing a check in a column) the magnitude of difficulty ("always," "often," "sometimes," "rarely," "never," or "not applicable") under the unaided condition ("X" in Figure 41.1). The patient is furthermore asked to rate his/her current aids (if experienced user; "O" in Figure 41.1) for 14 listening situations divided into 3 areas (difficulty at home, difficulty at work, and difficulty in social situations). After the patient has had the opportunity to wear the hearing aid for at least 4 weeks, he/she is asked to check the column corresponding to his/her perception of the performance of the new aids relative to the unaided condition and, if applicable, to his/her current hearing aids ("+" in Figure 41.1). In this manner, the audiologist can quickly determine if the patient perceives the aided performance as being better than either the unaided condition or with his/her current hearing aids. This is because the check mark would be placed in a column further to the right than was marked for the unaided and/or current aids. The Washington University questionnaire has proven to be an efficient and useful clinical tool, because of its ease of use for both the patient and the audiologist.

Earmold Impressions

Finally, and at the end of this second visit, impressions are made for the purpose of ordering earmolds for BTE fittings or custom products. Figure 41.6 illustrates an important point concerning the benefits of ordering a Libby 3- or 4-mm horn for a BTE fitting. In this case, the first author was fitting a patient who arrived at the clinic from another facility with a BTE coupled to the earmold with No. 13 tubing. Initial real-ear insertion gain (REIG) measures were performed to verify if the measured REIG reasonably matched the prescribed NAL-RP (solid curve) target. The initial REIG (lower curve) with the No. 13 tubing revealed that the measured REIG was significantly below gain of the prescribed formula. Rather than attach the hearing aid to the programmer to increase the gain to match the target, it was decided to remove the No. 13 tubing and drill the bore

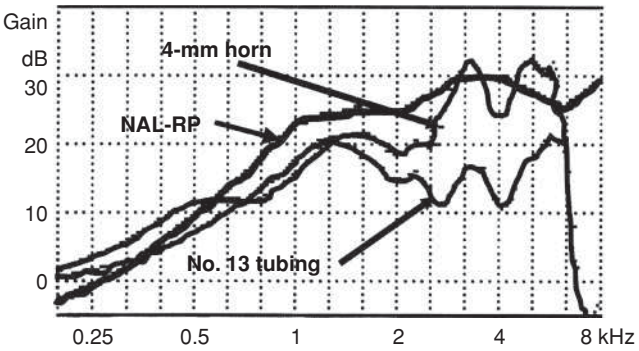


FIGURE 41.6 Difference in measured REIG between No. 13 tubing and 4-mm Libby horn. The *wider curve* represents the NAL-NL1 prescriptive target for a 65-dB SPL input level.

to make it wider to accept the wider outside diameter of the 4-mm horn. A repeat REIG with the 4-mm horn (upper curve in Figure 41.6) clearly indicates that the REIG with the 4-mm horn arrives much closer to the prescribed REIG than was possible with the No. 13 tubing. More importantly, the amplifier in the hearing aid was not programmed to achieve the greater required gain (i.e., the clinician was able to maintain greater headroom). This leaves the amplifier available for future increases in gain, should the patient's hearing loss decrease. By not programming the amplifier to provide greater amplification, there was probably less distortion at the output of the hearing aid and the amplified sound was crisper. It is our strong belief that almost all patients should be fit with hearing aids coupled with a 3- or 4-mm horn unless the hearing loss is of a rising configuration. In our clinics, virtually all of our patients are fit with a 3- or 4-mm horn! Also, we order earmolds and custom products with a select-a-vent (SAV) to provide greater flexibility for controlling the low-frequency response to reduce or eliminate the occlusion effect and maintain some control over feedback.

One significant advance in earmold technology has been the manner in which impressions are being processed after the impression has been made. In the past, the impression was made and placed in a box with the order form and forwarded to the hearing aid manufacturer or earmold laboratory. At this point, a cast of the impression was made to create the end product. This process is still completed, but with one major change. Advances in computer and software technology in the past several years allow most manufacturers to scan the received impression and store it in their computers. The scanned impression is then modeled and decisions are made by the software for deriving the final product. Initially, scanning technology was only available for the leading hearing aid manufacturers and earmold laboratories for the purpose of modeling custom products. A few years ago, one manufacturer allowed audiologists to scan the impression within the clinic and download the scanned image to the manufacturer via the Internet. Now, several laboratories provide similar technology. One of the obvious advantages of this method is that it is no longer necessary to remake impressions when a problem is present with the initial impression. The remake can be manufactured from the scanned image. A more recent advance from at least two manufacturers is the ability for audiologists to directly scan the ear and ear canal (i.e., no impression is made) and send the scanned image immediately over the Internet to earmold laboratories and hearing aid manufacturers. Using this process, the concerns and issues of placing otodams and impression material into the ear canal will soon be a process of the past.

Coupler Measures (between the Second and Third Visits) with ANSI-2003 (Revised in 2009)

When the hearing aids arrive from the manufacturer, they are placed on an HA-1 or HA-2 coupler and their performance is compared to the ANSI-2003 (Figure 41.7) measures supplied by the manufacturer. Recently, ANSI-2003 was revised to ANSI-2009, but we experience, at the time of preparing this chapter, no hearing aid analyzer has the software to implement its use and no hearing aid manufacturer is delivering hearing aids measured using ANSI-2009. The performance of the hearing aid must adhere to the specifications provided by the manufacturer. If not, the hearing aid is sent back to the manufacturer to be replaced by another unit. See Chapter 39 for a detailed discussion of the ANSI-2003 standard and any impending changes.

In addition to measuring the performance of the hearing aids re: ANSI-2003, the authors may also evaluate the "smoothness" of the frequency-gain response to input levels of 50 to 90 dB SPL, in 10-dB steps (ANSI-1992). Figure 41.8 illustrates the frequency-gain response of one hearing aid measured for input levels of 50 dB (upper curve) to 90 dB SPL (lower curve) in 10-dB steps. Notice how the "smoothness" of the frequency-gain response for the 90 dB SPL input is the same as the "smoothness" of the frequency-gain response for the 50 dB SPL input. On the other hand, if the hearing aid yielded significant intermodulation distortion, one would expect the frequency-gain response obtained with the 80 to 90 dB input (lower curves) to be "irregular" or "jagged" when compared to the frequency-gain responses at 50 to 70 dB SPL. Revit (1994) reported that the appearance of a "jagged" frequency-gain response at higher input levels is an indication of the presence of intermodulation distortion, which in turn can result in reduced recognition of speech or poor sound quality. We feel that clinicians should return any hearing aid to the manufacturer for which a "jagged" frequency-gain response is obtained and a copy of the coupler printout verifying the presence of the jagged frequency-gain response should be sent along with the repair form.

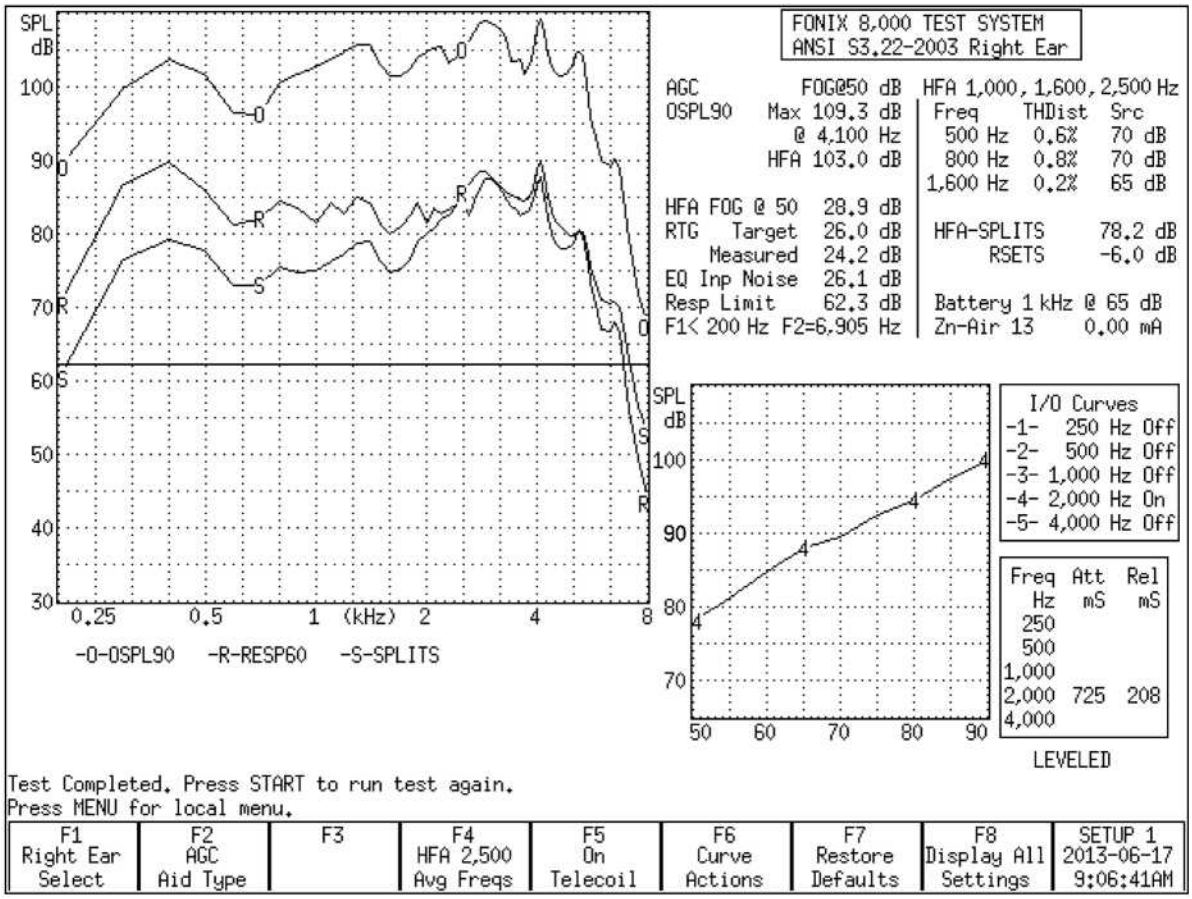


FIGURE 41.7 Example of ANSI S3.22-2003 coupler measure for a hearing aid with nonlinear signal processing and a telecoil.

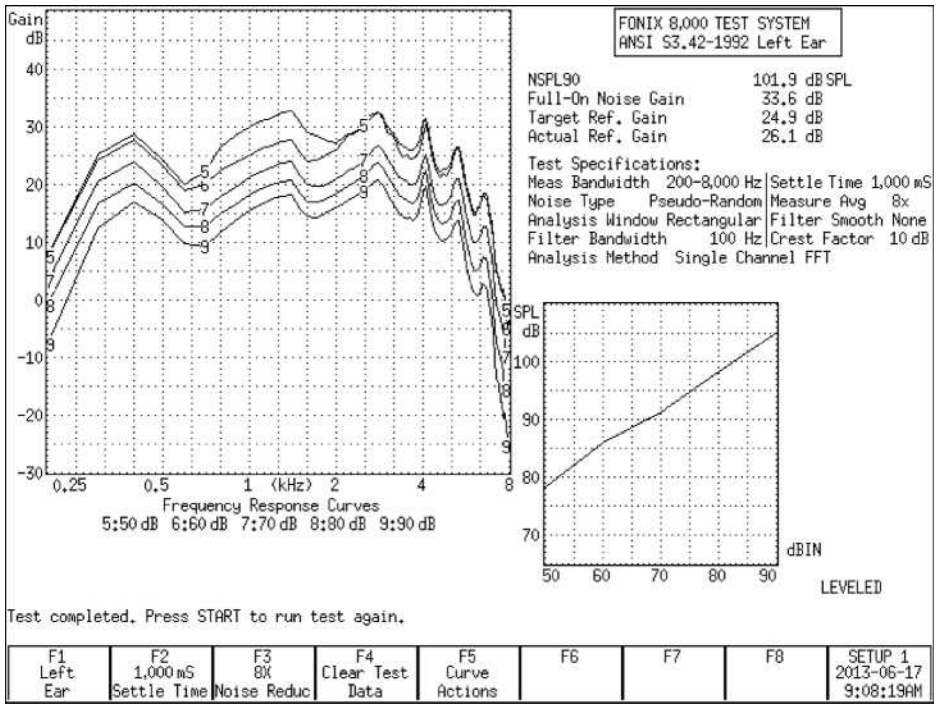


FIGURE 41.8 Example of ANSI S3.42-1992 coupler measure for input levels of 50 to 90 dB SPL for the same hearing aid.

Special Tests

Virtually all current hearing aids are delivered incorporating multichannel, DSP. As indicated earlier, there are many significant advantages provided by DSP when compared to previous analog signal processing. One potential disadvantage of DSP in increasing the number of channels of signal processing is group delay. "Processing time" or group delay is defined as the finite time delay created as an input signal passes through a hearing aid from the microphone to the receiver. The group delay in digital hearing aids is considerably longer in comparison to analog hearing aids because of the complex conversion of the input sound signal into discrete quantities for signal processing. Whereas the time required for a single-channel analog hearing aid to process input signals is a few tenths of a millisecond, the time needed for DSP can vary widely depending on the DSP algorithm and number of processing channels. In general, the greater the number of processing channels, the longer the processing time or group delay. One major concern of current open-fit hearing aids is the possibility of an unprocessed signal passing through a vent or leak around the earmold or shell and being heard earlier than the processed signal that is delayed because of the delay created by the hearing aid.

Previous research has demonstrated that long group delay can negatively affect speech production and perception for normal-hearing and hearing impaired patients (Stone and Moore, 2005). Specifically, concerns of auditory confusion and degradation of speech production and perception of participants' own voices (Stone and Moore, 2005) as a result of delay have been investigated.

Auditory confusion can occur when there is a delay between the hearing aid user observing the movement of the talker's lips and hearing the sound of the talker's voice. Summerfield (1992) reported that sound can lag the visual image by up to 80 ms before confusion will occur. Therefore, he recommended that processing for hearing aid users with severe-to-profound hearing loss be as short as possible. A group delay as long as 40 ms was felt to be acceptable.

Stone and Moore (2002) reported on the effect of group delay on normal-hearing participants' own speech production and own voice perception using a simulation of hearing loss. They reported that delays greater than 20 ms can lead to the perception of an "echo" in the participant's own voice, whereas delays of less than 10 ms might lead to a perception of a subtle change in the timbre of the sound. In a follow-up study, Stone and Moore (2005) utilized hearing impaired participants to measure the effect of group delay (13 to 40 ms) on perception of the participant's own voice and speech production. It was concluded that participant disturbance to the sound of his/her voice increased with increasing group delay. Additionally, participants with low-frequency (500, 1,000, and 2,000 Hz) hearing loss greater than 50 dB HL were significantly less disturbed than those participants with milder low-frequency hearing loss. Specifically, the results showed that delays of greater than 15 ms can be unacceptable to listeners with low-frequency hearing loss at approximately 35 dB HL, but patients with more moderate-to-severe hearing loss in the low frequencies may be able to tolerate longer delays.

Stone and Moore (2002) also analyzed objective and subjective measures of the effects of hearing aid delay on speech production and perception in two different environments with the goal of defining an upper limit to permissible processing delay. They concluded that normal-hearing participants reported that the disturbing effects on perception became significant when delays exceeded 15 ms in an office environment and 20 ms in a test booth. Objective measures of speech production did not show any significant negative effects of delay until the delay reached 30 ms. As a result of these findings, Stone and Moore (2002) proposed that DSP hearing aids should be able to incorporate delays as long as 15 ms with few negative side effects. Additionally, the amount of tolerable processing delay decreased by about 5 ms in reverberant environments as compared to a near-anechoic environment.

Electroacoustic Procedures for Assessing DSP Features

Although not included as part of the ANSI-2003 standard, several tests are now available to verify a number of DSP features. These include measures of group delay, use of modulated "speech-like" signals to assess the compression characteristics of the hearing aid, and the use of a bias signal to assess the effectiveness of the NR algorithm.

Group Delay

Although this may vary between manufacturers, using the Frye Electronics™ hearing aid and real-ear analyzers, the group delay test uses a simple broadband impulse signal and a 20-ms time window. The measured group delay is calculated from the sampling rate (25.6 kHz), internal analyzer delay (approximately 0.5 ms), and the characteristics from the hearing aid. Figure 41.9 illustrates the group delay (curve to the left) for the left (1.5 ms) hearing aid. Ideally, for reasons described above, the audiologist would hope to record a value of ≤15 ms. Also, this simple test can inform the audiologist whether the hearing aid is analog or digital. If the group delay is ≤1 ms after completing the test, then the hearing aid is probably a single-channel analog device. On the other hand, if the group delay is ≥1 ms, then the hearing aid is probably a multichannel DSP device.

Modulated Speech Signal

The NR feature in current DSP hearing aids often reacts to a continuous speech-shaped signal as if it were noise and

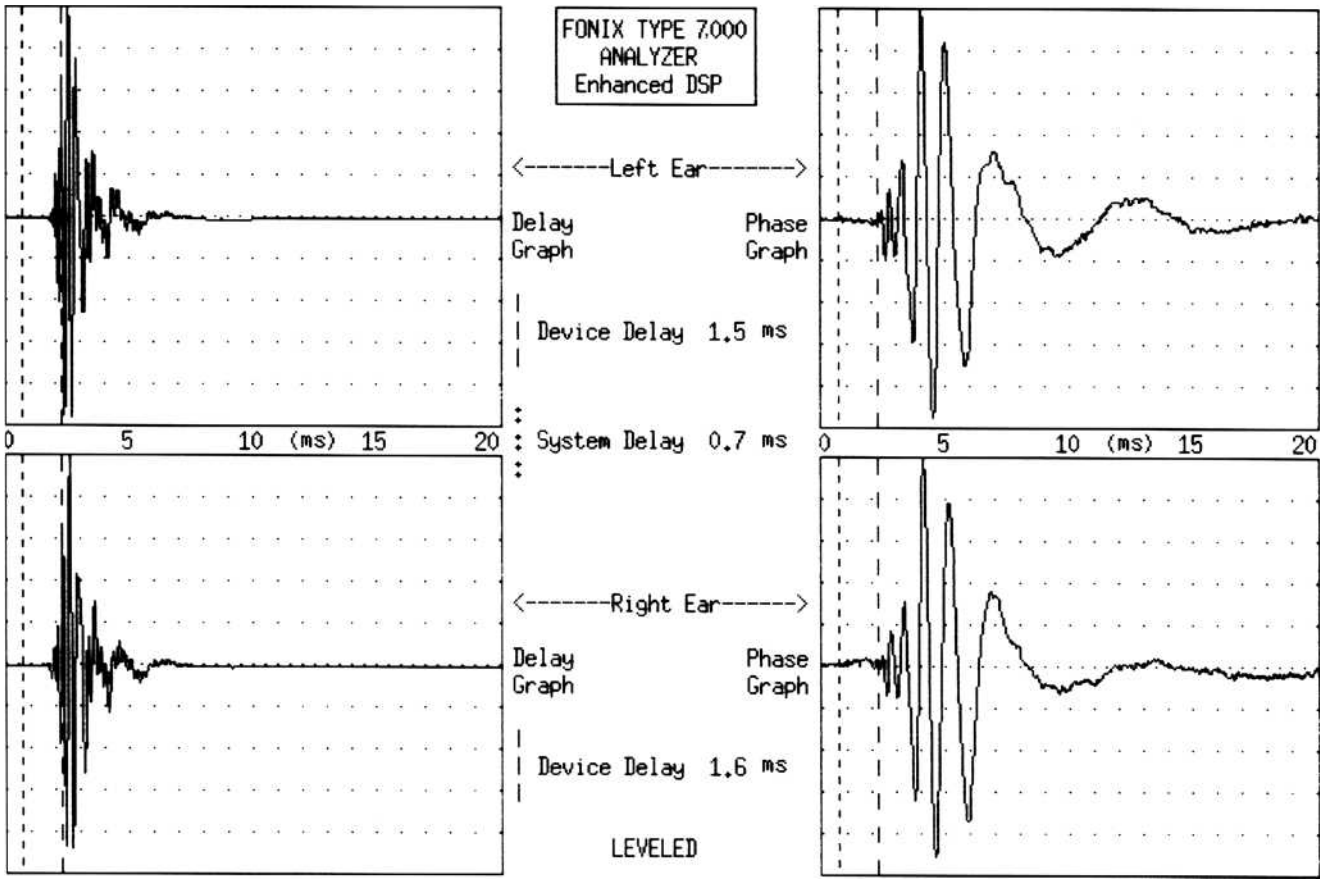


FIGURE 41.9 Example of measurement of group delay (1.5 ms) for left and right hearing aids.

reduces gain/output. This can pose problems when trying to verify hearing aid gain/output characteristics. To resolve this problem, manufacturers of hearing aid and real-ear analyzers have developed *modulated* or "live speech" signals so the DSP processes the signal as speech and does not reduce gain/output. With the Frye Electronics™ units, a continuous composite speech signal can also be presented as a randomly amplitude-modulated speech signal in bursts that are 300 ms wide. To verify that the noise suppression (compression) feature of a DSP aid is operating effectively, a simple test is to examine the performance using the continuous composite speech ANSI signal. The audiologist then takes a second measure with the modulated ANSI signal (Figure 41.10). The difference between the two measures (typically the measure for the continuous signal will be lower than the measure for the modulated signal) reflects the amount of overall noise suppression provided by the hearing aid to a broadband noise. If the two curves are superimposed, it is a good indication that the noise suppression feature has been disabled or is not functioning properly, or the hearing aid does not contain noise suppression. It is important to remember that significant differences may be present in the noise suppression feature between hearing aid manufacturers. It is also possible that manipulation of the input level may be required to activate the noise suppression or the audiologist may need to wait a few seconds before the noise suppression becomes active.

In several hearing aid and real-ear analyzers, audiologists also have the option of using several "speech-like" signals. With the Frye Electronics™ units, the audiologist has the option of using an ANSI (ANSI S3.42-1992) or the International Collegium of Rehabilitative Audiology (ICRA) speech signal. The spectra of these two input signals in Frye Electronics™ equipment are quite different (Figure 41.11). The ANSI signal decreases at a rate of 6 dB/octave above 900 Hz, whereas the ICRA signal is flat to about 500 Hz and then decreases more sharply at a rate of 10 dB/octave above 500 Hz. Below 900 Hz, ICRA is 3 dB more intense than ANSI. At frequencies above 900 Hz, ICRA rolls off more steeply than ANSI such that at 2,000 Hz, ICRA is 8 dB less intense than ANSI and by 8,000 Hz ICRA is 16 dB less intense than ANSI. This means it is possible to measure significantly different gain and output values depending on the signal processing of the hearing aid. If the hearing aid has a low compression kneepoint in the high frequencies, it is likely that the measured gain/output using the ICRA signal will be considerably greater than with the use of the ANSI spectra (middle and upper curves in Figure 41.12). The lower curve in Figure 41.12 is the response of the hearing aid to a continuous composite speech signal. This curve

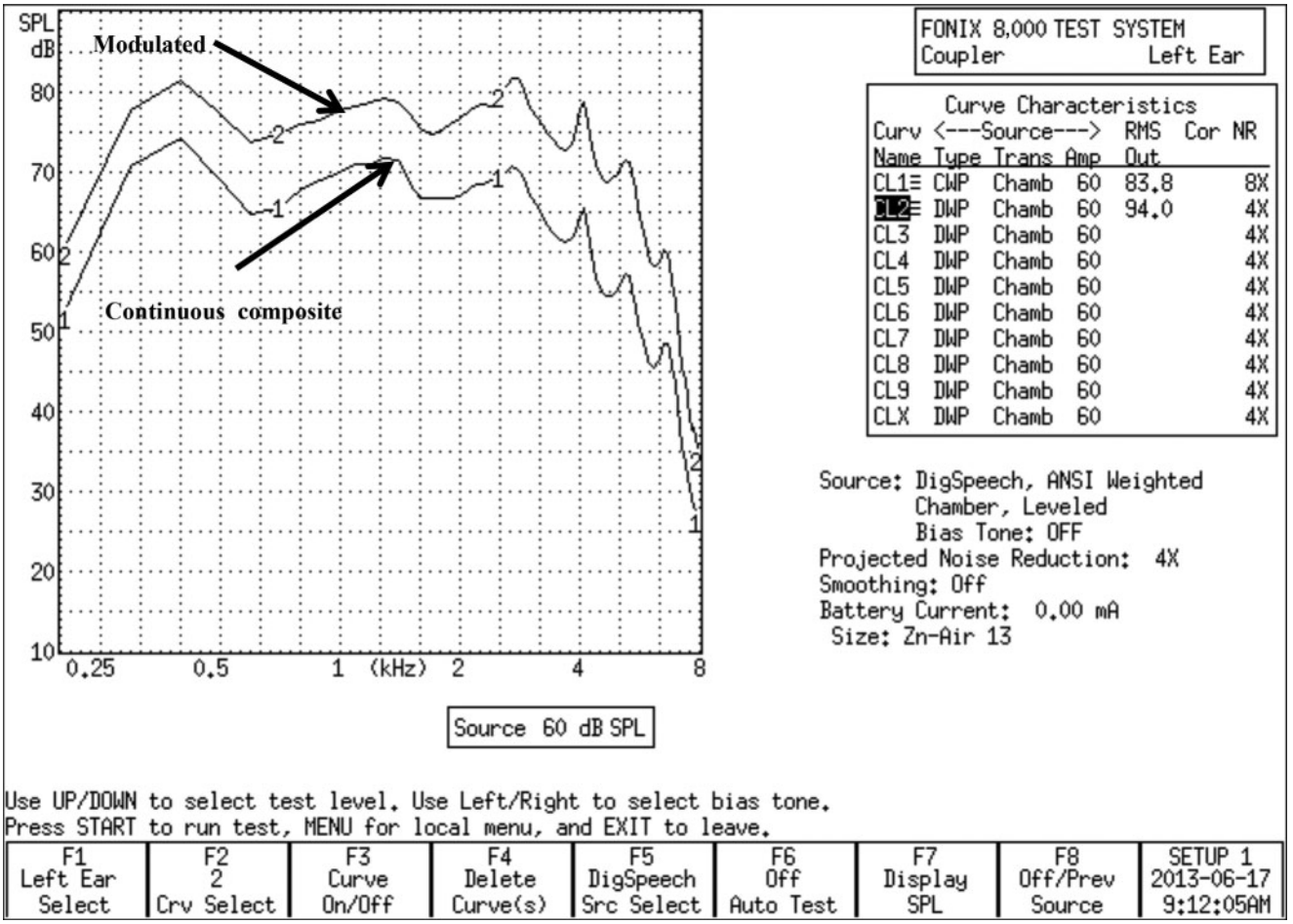


FIGURE 41.10 Example of the differences in measured REAR using a continuous ANSI composite signal (*lower curve*) and a modulated ANSI signal (*upper curve*) illustrating the compression of a DSP hearing aid.

shows the least amount of gain, because the hearing aid is processing this signal as noise.

Bias Signal

To help audiologists verify the effectiveness of NR filters of a DSP hearing aid, Frye Electronics™ introduced a "bias signal" test. With this test, a randomly modulated speech spectrum signal (the audiologist can control the overall level of the speech signal) and a randomly modulated puretone signal are presented simultaneously (the audiologist can control the frequency and intensity of the bias signal). With

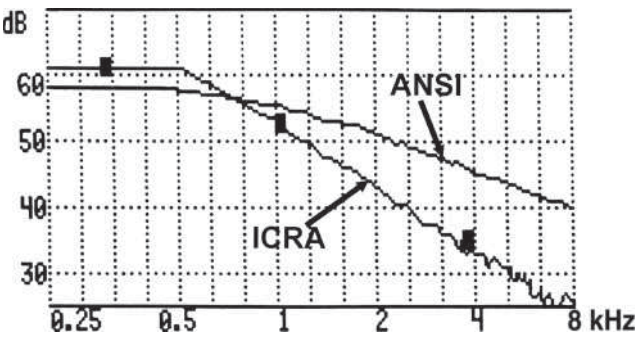


FIGURE 41.11 Difference in spectrum between the ANSI and ICRA speech signals on the Frye 7000.

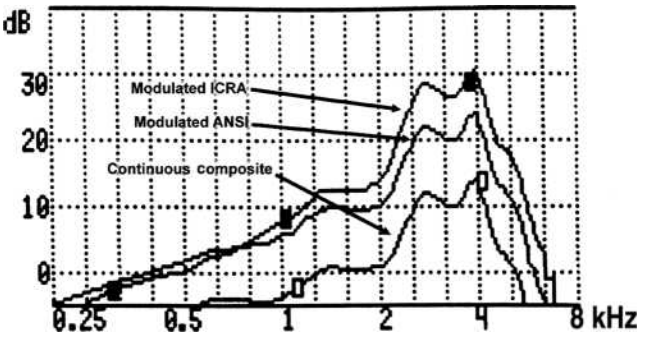


FIGURE 41.12 Difference in measured REIG for a DSP hearing aid using continuous ANSI composite (*lower curve*), modulated ANSI (*middle curve*), and ICRA (*upper curve*).

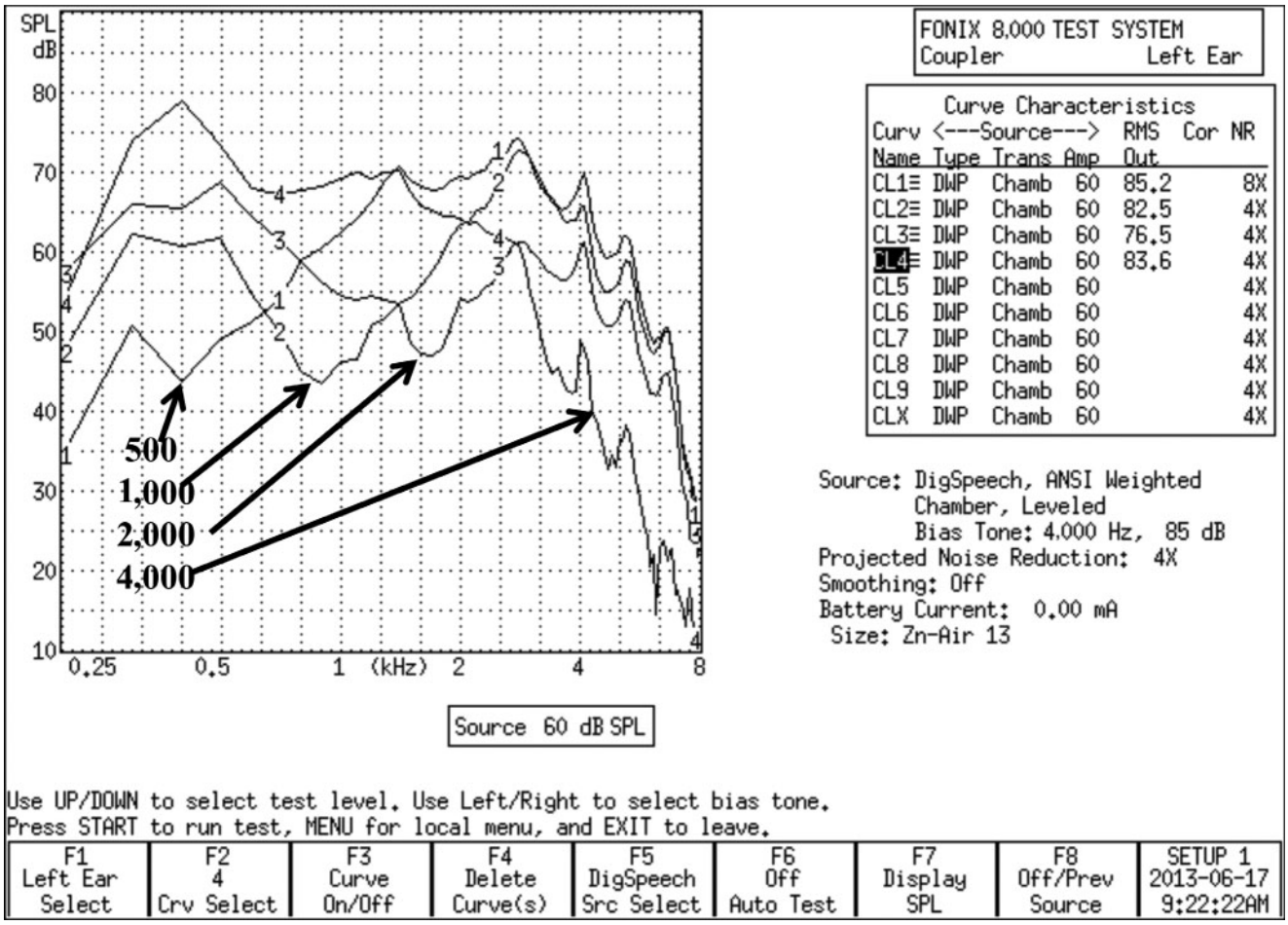


FIGURE 41.13 Illustration of the effectiveness of the NR feature of a DSP hearing aid using a 500-, 1,000-, 2,000-, and 4,000-Hz bias signal.

this test, and if the NR filter is functioning properly, the audiologist will see a reduction in gain/output in the frequency region surrounding the bias puretone signal. The remaining portion of the frequency response above and below the bias signal will maintain full amplification. For example, in Figure 41.13, a 500-Hz bias signal is presented with modulated ANSI speech. As can be seen, only the frequency region around 500 Hz reveals attenuation whereas the frequency region above approximately 500 Hz shows full amplification. Also, reported in Figure 41.13 is the same reduction of the output at 1,000, 2,000, and 4,000 Hz using the 1,000-, 2,000-, and 4,000-Hz bias signal. The more channels the DSP aid has, the narrower the frequency region in which the reduction of gain/output occurs with this test. On the other hand, in a hearing aid with fewer channels, the broader bandwidth will reduce the gain/output in a broader frequency region. One must remember that when there is a reduction of gain/output in response to a noise, there is also a reduction of gain/output in the same region of the speech signal (i.e., no change in the signal-to-noise ratio within that channel). It can be assumed that a hearing aid with a greater number of channels may provide greater speech intelligibility/comfort in noise. This is because a narrower slice of the speech signal is being reduced in response to the NR feature of the hearing aid.

HEARING AID FITTING (THIRD VISIT)

Real-Ear Measures

The most reliable and efficient method for verifying the performance provided by amplification is REM. Research reports that the 95% confidence interval (CI) for repeatability of REM is approximately 3 dB (Valente et al., 1991). By comparison, the 95% CI for functional gain measures (i.e., unaided sound-field puretone or spondee thresholds minus aided sound-field puretone or spondee thresholds) is approximately 15 dB (Hawkins et al., 1987). If the audiologist were to incorporate a modification to a hearing aid (i.e., change in the vent size or tubing diameter, change in the damper in the earhook, or reprogramming of the aid) the difference between the gain measured after the change would have to be at least 3 dB different than the first REM

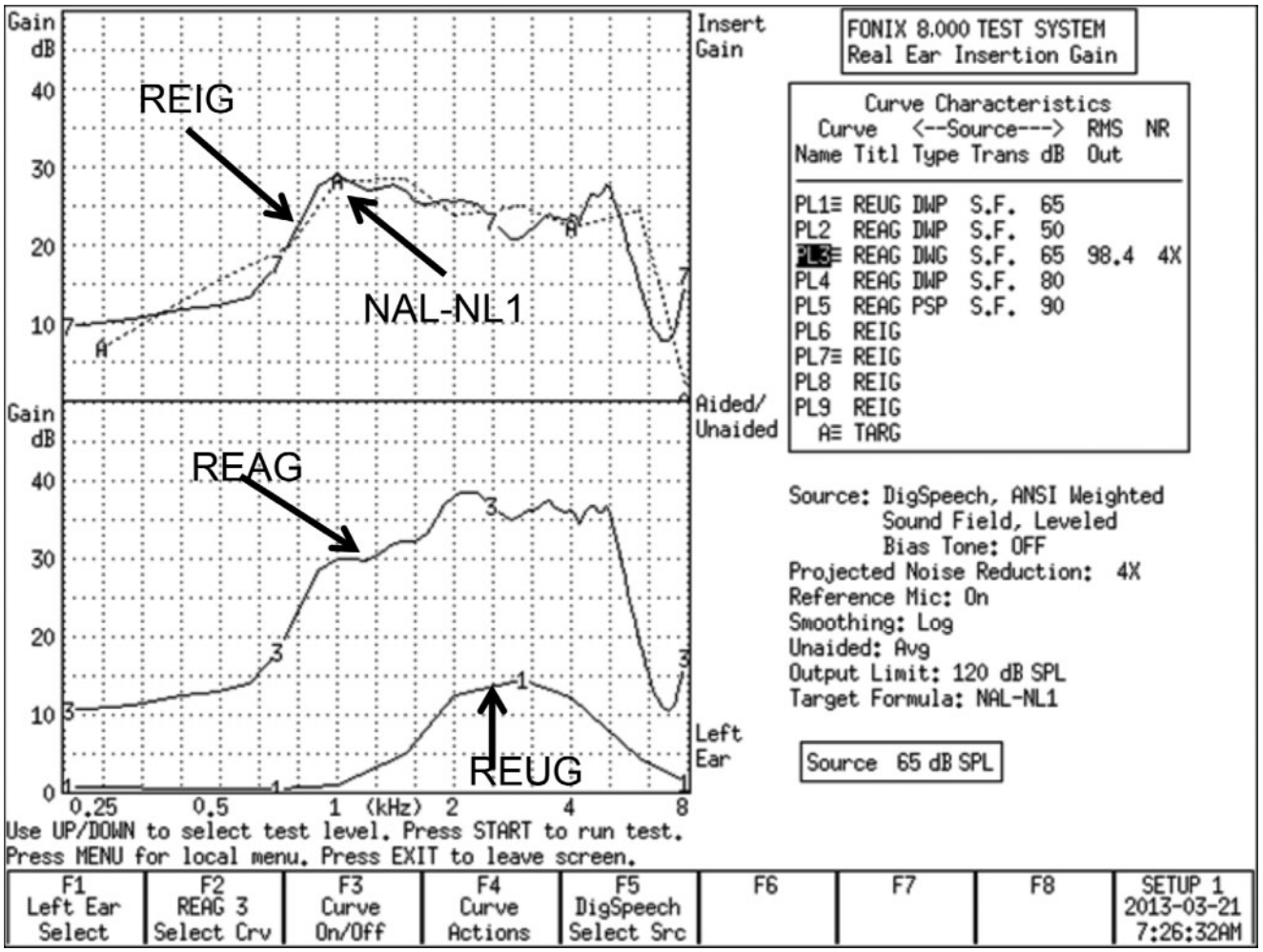


FIGURE 41.14 Example of measured REUG (*lower curve*), REAG (*middle curve*), and REIG (*upper curve*) for a linear hearing aid to the NAL-NL1 ("*A*") prescriptive target for an input level of 65 dB SPL.

for the difference to be considered statistically significant. Because of the greater variability inherent in functional gain measures, the difference between the second and first measures would have to be greater than 15 dB for the results to be statistically significant. One can readily see that REMs are considerably more reliable than functional gain measures. Consequently, it is highly recommended that REMs be used consistently instead of functional gain measures.

Typically, at least three REMs are obtained clinically. The first involves the measurement of the response of the ear canal without the hearing aid in place. This is referred to as the real-ear unaided gain (REUG in gain) or real-ear unaided response (REUR in dB SPL) and is an accurate and reliable measure of the resonance of the ear canal (lower curve in Figure 41.14). To make this measure, the patient is seated in a chair directly facing a loudspeaker (i.e., 0° azimuth) that is placed at ear level at a distance of 12 to 18 inches. The probe-tube from the probe microphone is marked 30 mm from the tip for adult males and 26 mm for adult females. This mark is placed at the intratragal notch and taped in place. A short burst of a speech-weighted composite noise is presented at a level of 65 dB SPL and stored as the unaided response (lower curve in Figure 41.14). As stated above, this measure represents the ear canal resonance. In the normal adult ear, the REUG has a peak amplitude of approximately 18 dB at 2,800 Hz.

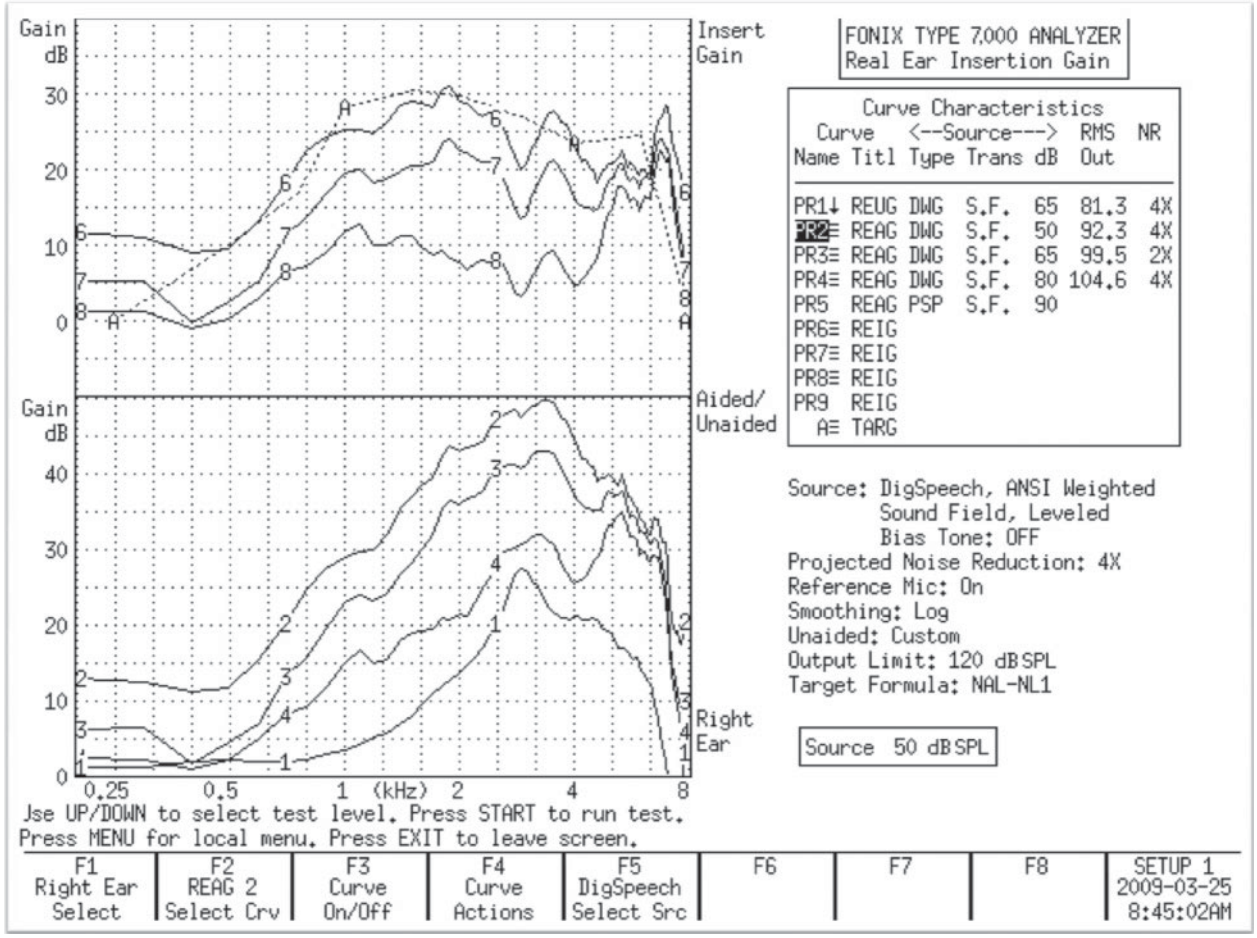
Next, the hearing aid shell, or earmold coupled to a BTE, is placed in the ear canal and the volume control is typically adjusted to the patient's most comfortable level (MCL). The resulting measure is the real-ear aided gain (REAG in gain) or real-ear aided response (REAR in dB SPL) provided by the hearing aid (middle curve in Figure 41.14). The difference between the REAG and REUG is the REIG (upper curve in Figure 41.14), or the gain provided by the hearing aid. The REIG is compared with the prescribed target ("A" in Figure 41.14) to determine if the measured frequency-gain response is appropriate for the patient's hearing loss.

To obtain the target REIG, the patient's audiometric data are entered into a real-ear analyzer to generate a "target" REIG ("A" in Figure 41.14) to which the measured REIG is compared. For the hearing loss illustrated in Figure 41.14, most audiologists would agree that the measured REIG closely matches the prescribed NAL-NL2 (Dillon et al., 2011) target.

Verification using a single input level is appropriate if the hearing aid has linear signal processing, because gain remains constant and measuring gain at several input levels would provide little additional information. On the other hand, if the hearing aid has nonlinear signal processing, then verifying gain should be made using several input levels. Figure 41.15A to C illustrates the verification of a hearing aid with nonlinear signal processing where it is easy to see that the measured $REIG_{50}$ matches NAL-NL1 for a 50-dB SPL input signal (Figure 41.15A), 65-dB SPL input signal (Figure 41.15B), and 80-dB SPL input signal (Figure 41.15C). It can be seen in Figure 41.15A to C that REIG is greatest for the 50-dB SPL input level and the REIG decreases, as the input level increases from 65 to 80 dB SPL.

Measuring the REAR (dB SPL) to the Dynamic Range (dB SPL)

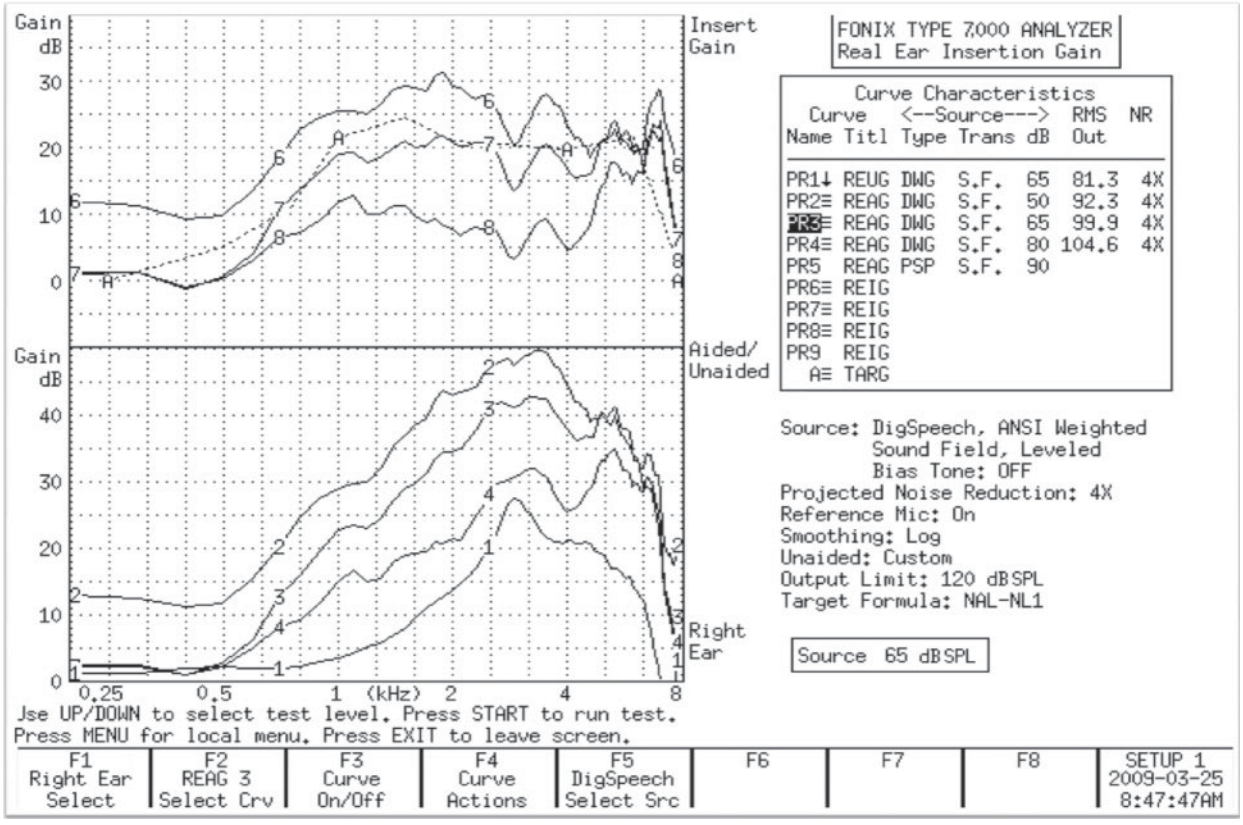
In a recent survey, Mueller and Picou (2010) reported that of the approximately 70% of audiologists who routinely verify the performance of hearing aids using REM, 78% measure the REAR to a prescriptive target and approximately 22% use the REIG. Since the previous edition of this chapter, there has been a significant increase in the number of audiologists selecting to verify where the amplified output (i.e., REAR in dB SPL) resides within the patient's individually measured dynamic range (DR). Using this method, the "prescriptive target" is the individual DR and the purpose is to verify where the amplified output, in response to a wide range of input levels, falls within the DR.



A

FIGURE 41.15 Example of REUG (*curve 1*), $REAG_{50}$ (*curve 2*), $REAG_{65}$ (*curve 3*), $REAG_{80}$ (*curve 4*) in the *lower segment* of **A**–**C**. Also reported is the $REIG_{50}$ (*curve 6*), $REIG_{65}$ (*curve 7*), and $REIG_{80}$ (*curve 8*) for a nonlinear hearing aid to the NAL-NL1 ("*A*") prescriptive target for an input level of 50 (**A**), 65 (**B**), and 80 dB SPL (**C**). (*continued*)

B



C

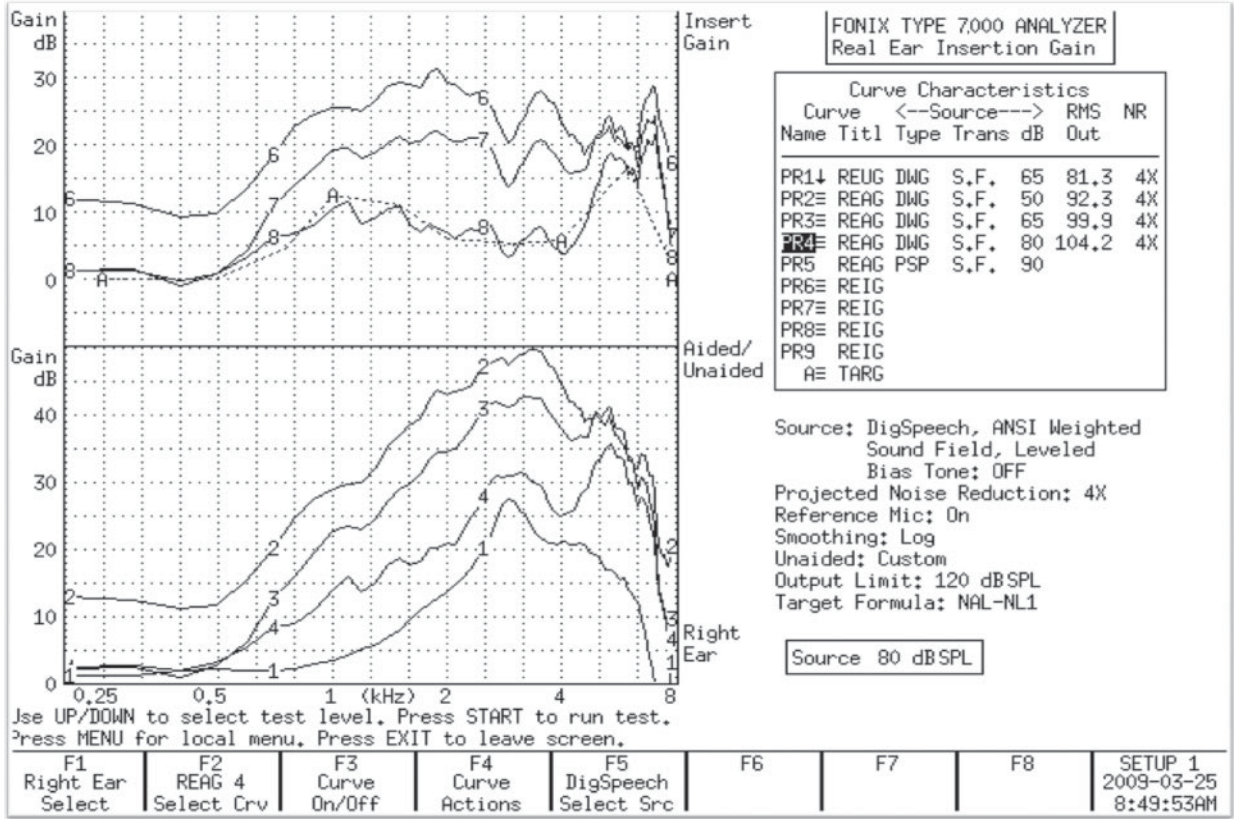


FIGURE 41.15 (*Continued*)

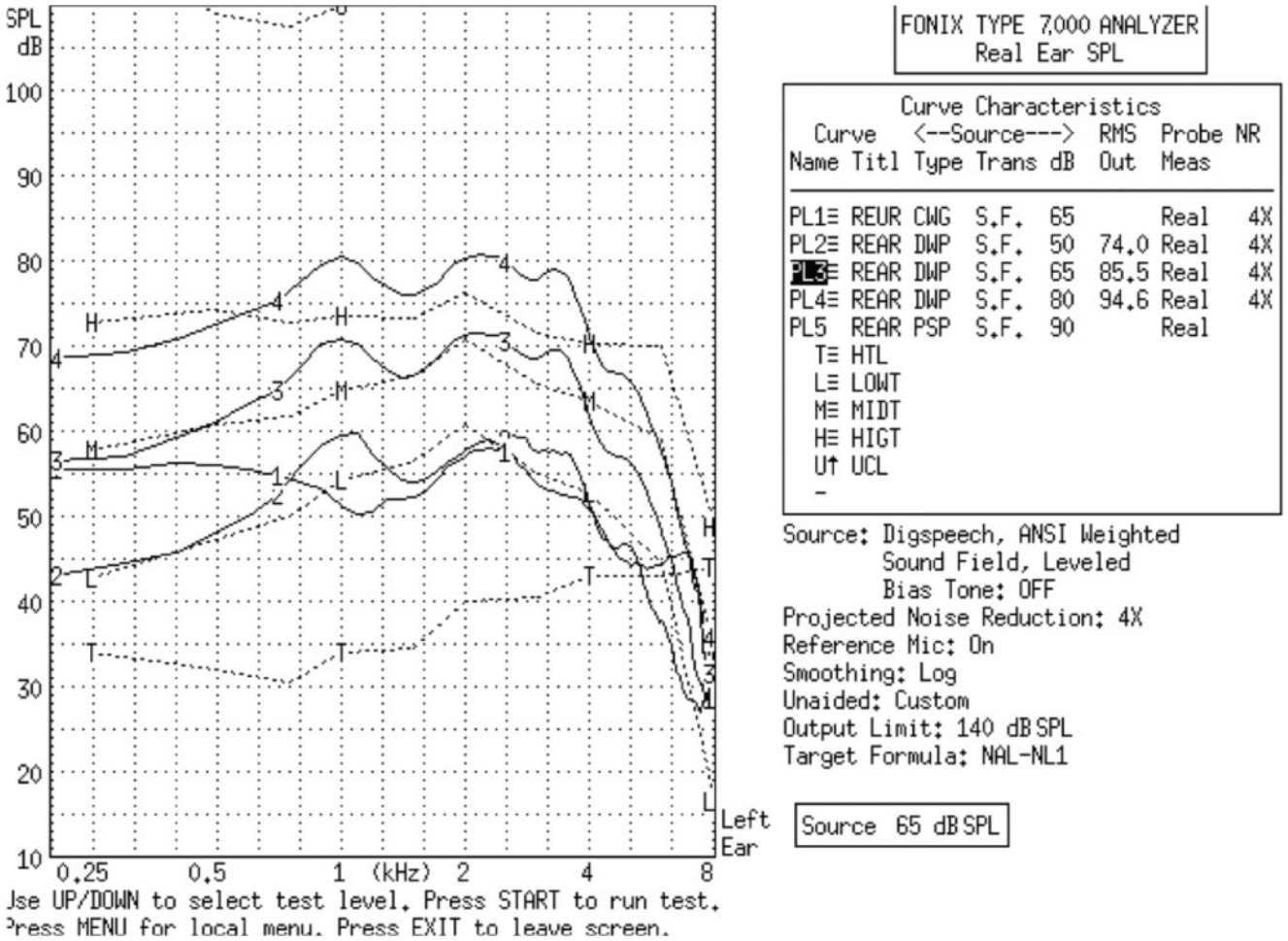


FIGURE 41.16 Example of REAR measures for a nonlinear hearing aid. In this case, the "*T*" represents the predicted threshold in dB SPL and "*U*" in the *upper part* of the figure represents the predicted LDL in dB SPL. The area between the "*T*" and "*U*" is the predicted residual dynamic range for the listener. The "*L,*" "*M,*" and "*H*" represent the NAL-NL1 REAR targets for soft, average, and loud input levels, respectively. The *2*, *3,* and *4* curves represent the measured REAR for input levels of 50, 65, and 80 dB SPL.

Figure 41.16 illustrates how this is accomplished. In this figure, "T" represents the predicted threshold in dB SPL, "L" is the NAL-NL1 target for an input of 50 dB SPL, "M" is the NAL-NL1 target for an input of 65 dB SPL, "H" is the NAL-NL1 target for 80 dB SPL, and "U" (the most upper curve) represents the LDL. The space between "T" and "U" is the predicted residual DR for the patient. Here, the reader can see the REAR for an input of 50 dB is very close to the "L" target for most of the frequency range. The REAR for the 65-dB input ("3") is at or near the "M" target. The REAR for the 80-dB input ("4") is at or near the "H."

Figure 41.17 illustrates similar information using the MedRx Live Speech Mapping unit. In this figure, the lower "O" line represents threshold and the upper "U" line represents LDL. The lower curve is the REAR to an input of 56 dB SPL using live speech (e.g., spouse talking to the patient using a microphone in combination with the software) and the measured REAR is above the "O" target indicating soft speech is audible. The middle curve is the REAR in response to an input of 67 dB SPL using the same stimuli and method, whereas the upper curve is the REAR in response to an input of 89 dB SPL. Again, it can be verified that the REAR to the input range of 33 dB (56 to 89 dB) falls within the DR.

Finally, Figure 41.18 illustrates similar information using the Audioscan Verifit system. In this example, the "X" and "O" in the left and right boxes represent threshold for the right and left ears, respectively; the "." represents the LDL and the "+" represents the REAR target for an input of 65 dB SPL.

Although significant differences exist between these pieces of equipment, their commonality is in allowing the audiologist to measure how the output of the hearing aid lies within the DR range (measured in dB SPL near the tympanic membrane) of the patient. These units allow audiologists to verify that the measured output to an input level of 50 dB SPL is above threshold, the measured output to an input level of 65 dB SPL falls approximately midway between threshold

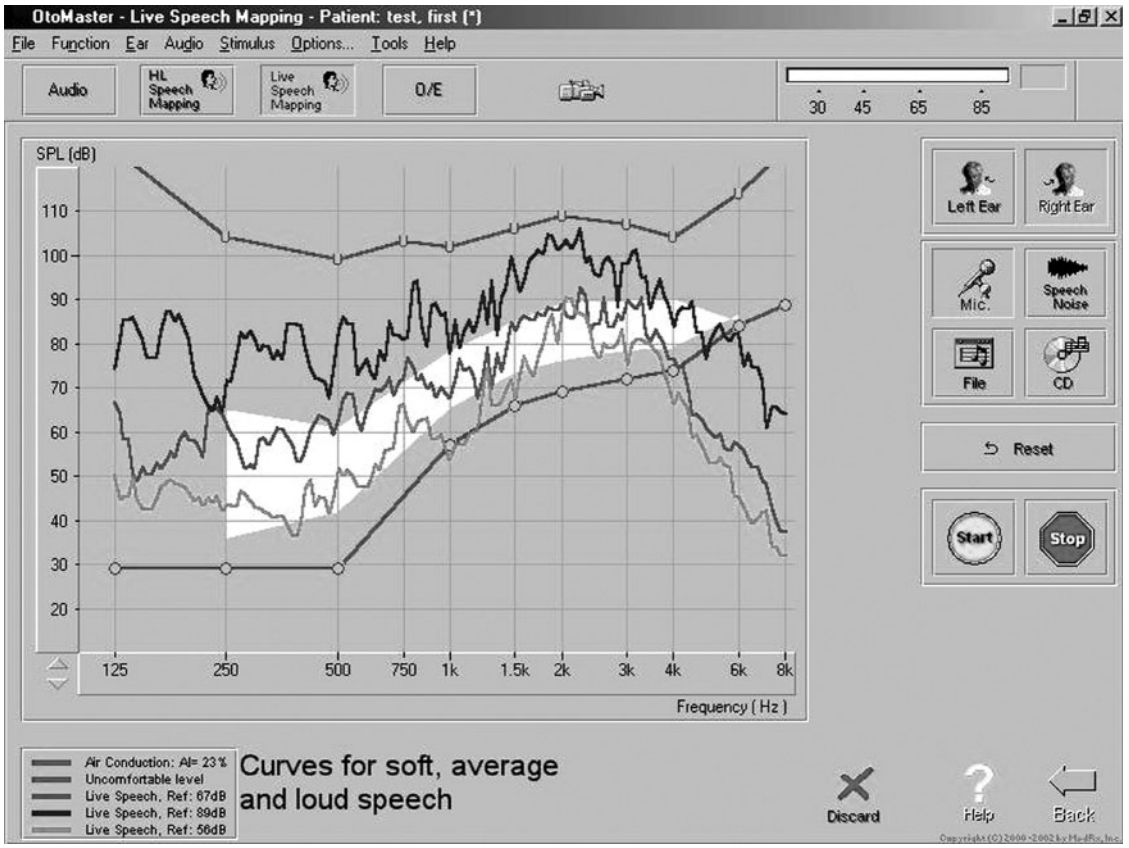


FIGURE 41.17 Example of REAR measures for a nonlinear hearing aid using the MedRx. [Courtesy of MedRx.]

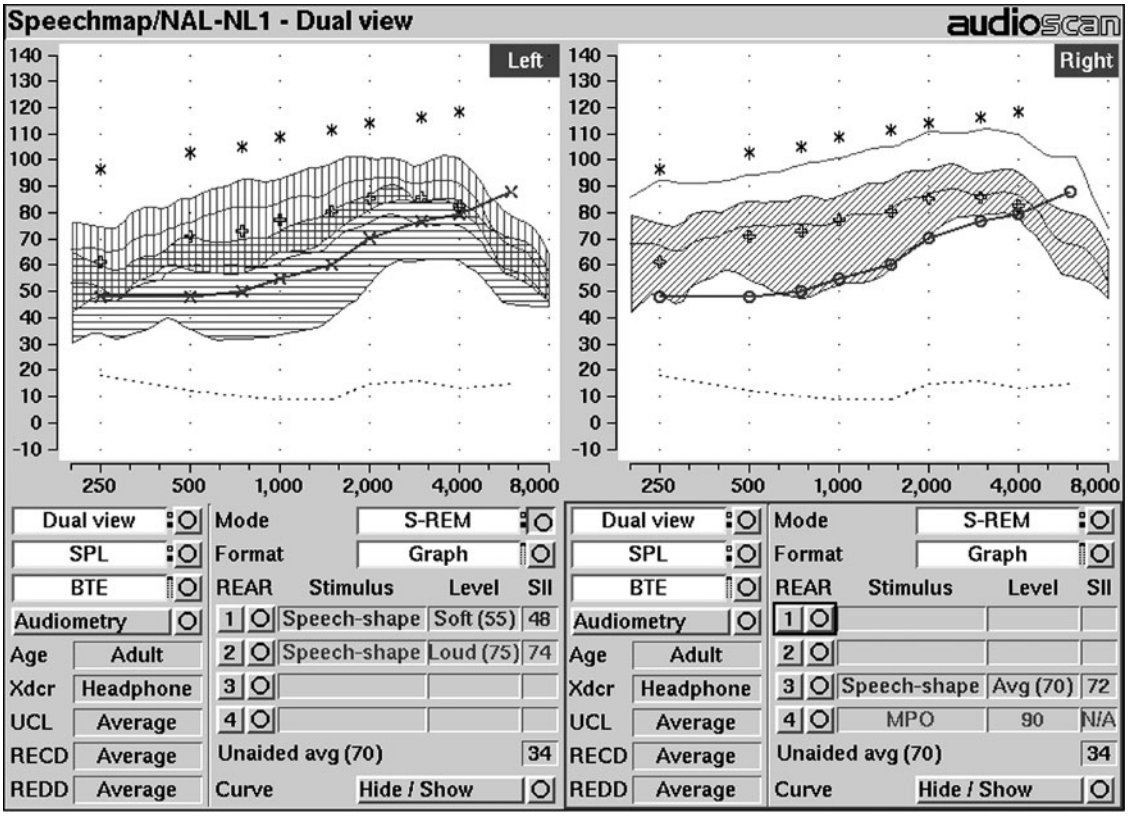


FIGURE 41.18 Example of REAR measures for a nonlinear hearing aid using the Audioscan Verifit. [Courtesy of Audioscan®.]

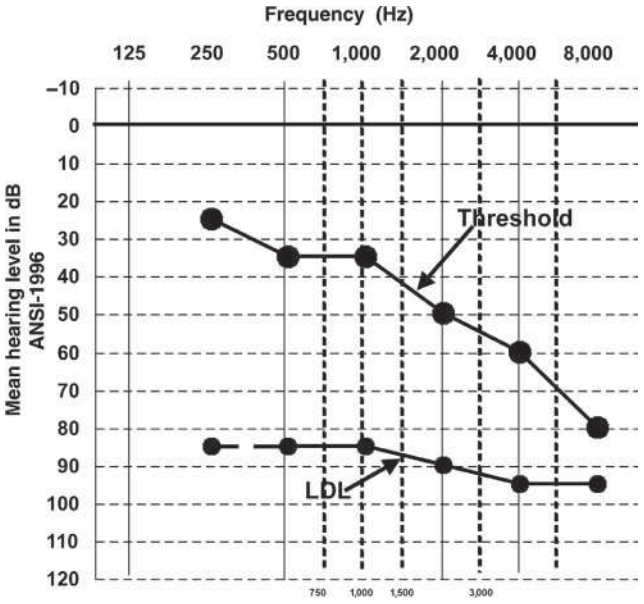


FIGURE 41.19 Measured threshold and LDL (in dB HL) for a patient.

and LDL, and the measured output to a high-input level (85 to 90 dB SPL) is below the LDL.

One problem associated with these devices is the reliance on predicted threshold (dB SPL) and LDL (dB SPL) based on the entered audiogram. After the audiologist enters the audiogram, the respective unit converts the entered thresholds (dB HL) into threshold in dB SPL by using average REDD transformations of dB HL to dB SPL. In addition, the software within these units predict the LDL in dB HL based on the data published by Pascoe (1988) and then transforms these values into dB SPL using average REDDs. Unfortunately, research has shown that the interparticipant variability of the combined factors of REDD and LDL can be as great as 40 dB wide (Pascoe, 1988; Valente et al., 1997). With such variability, it is difficult to imagine with any degree of confidence that the predicted threshold and LDL in dB SPL would be the same as individually measured thresholds and/or LDLs. If the advantage of these units is that the "target" is the patient's DR and these units can report how the hearing aid output fits within that DR, then it would seem appropriate that the DR appearing on the monitor of these units reflects the DR of the individual and not the DR of an average individual.

Figures 41.19 and 41.20 illustrate how this could be a potential problem. Figure 41.19 reports the measured hearing thresholds and LDLs in dB HL for the right ear of a patient. The LDLs are the same as those appearing in Figure 41.4 at 500 to 4,000 Hz. These audiometric thresholds were entered into the software of one commercially available real-ear system. From these entered thresholds, the software predicted thresholds in dB SPL by adding the average REDD. The result is the "predicted threshold" illustrated in Figure 41.20. The "measured threshold" is the result of adding the patient's measured REDD (values in parenthesis in Figure 41.4). As can be seen, in this case there was very little difference between "measured" and "predicted" thresholds (dB SPL). The upper curve in Figure 41.20 is the "predicted LDL" that was calculated by the software of the real-ear system predicting the LDL in dB HL from the entered threshold of the patient based on the results of Pascoe (1988) and adding the average REDD. The

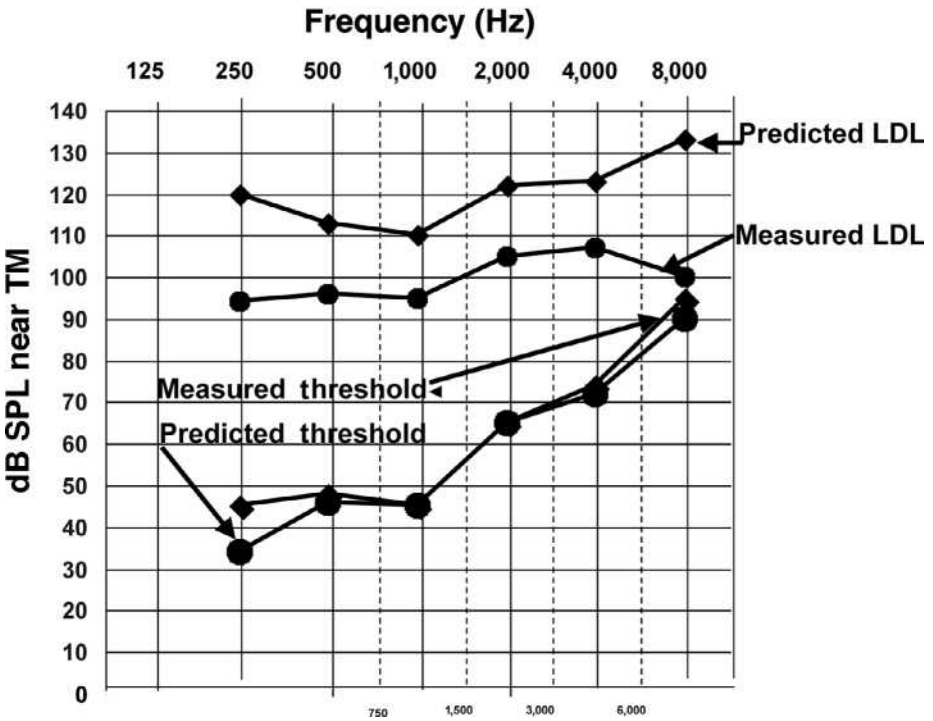


FIGURE 41.20 Measured and predicted threshold and LDL (in dB SPL) for the same patient.

lower upper curve in Figure 41.20 is the "measured LDL" that was measured at the time of the HAE, as described earlier and reported in the table in Figure 41.4. As the reader might recall, the LDL measured by Pascoe (1988) was based on a loudness judgment of "too loud," whereas the loudness judgment for the measured LDL was "loud, but OK." In addition, the "measured LDL" uses measured REDD and not predicted REDD. In this case, this is of minimal impact for this patient, as seen in the similarity of predicted and measured thresholds in the lower curves of Figure 41.20.

In this case, the measured and predicted thresholds were quite similar, but the differences between measured and predicted LDLs were quite large. Therefore, the differences in DR between the measured and predicted curves can clearly be seen. In this case, the predicted DR is significantly wider than the measured DR. This can have a significant impact on the selection and programming of the compression and output-limiting characteristics of the hearing aid. If the audiologist used the predicted LDL to determine where to place the $RESR_{90}$, it can easily be seen how this patient would probably find the output of the hearing aid to be excessively loud and possibly reject amplification.

Selection of Prescriptive Formulae

Prescriptive "Targets" for Linear Amplification

Although not as commonly used as in the past, for hearing aids providing linear amplification (i.e., constant gain for varying input levels), we verified if the measured REIG "matches" the prescribed REIG by presenting an input signal level of 65 or 70 dB SPL (i.e., REIG65-70). Figure 41.14 illustrates the measured REIG65 (thin curve) in relation to the prescribed NAL-NL1 REIG target ("A" in Figure 41.14). Notice how well the measured REIG65 compares to the prescribed target curve. If this goal is accomplished, we are reasonably assured that adequate amplification has been provided to allow average conversational speech in a "quiet" environment to be audible and comfortably loud. Please note we are not overly concerned if the measured REIG65-70 does not "hit" the prescribed REIG at each frequency, but are more concerned about whether the *shape* of the measured REIG65-70 "matches" the *shape* of the prescribed REIG. This is because the user has the ability to adjust the overall gain with the gain control.

Currently, the most popular prescriptive "target" for linear aids appears to be the NAL-NL2 (Dillon et al., 2011; Keidser et al., 2011) and DSL i/o V5.0 (Scollie et al., 2005).

Prescriptive "Targets" for Nonlinear Amplification

Typically, most hearing aids entering the commercial market use nonlinear signal processing (i.e., greater gain for low-input levels and less gain for high-input levels). Audiologists can now program crossover frequencies (Cf), compression threshold(s) (CT), compression ratio (CR), and time constants in one or more channels (bands) (see Chapter 35 for in-depth discussion of these parameters). Because of this, prescriptive procedures designed for linear signal processing (i.e., gain remains constant as input level changes) are no longer appropriate. To address this need, several prescriptive procedures for nonlinear signal processing have been introduced.

One approach incorporated into many of the new prescriptive procedures is to re-create the normal loudness patterns of speech and other complex sounds for the hearing impaired listener (i.e., "normalization" approach). This approach is the major fitting goal of hearing aids using wide DR compression (WDRC). Clearly, it may be difficult to normalize loudness patterns for complex sounds (i.e., speech) because abnormal loudness growth functions vary not only as a function of frequency, but also as a function of signal duration. An example of the new procedures using this approach includes DSL i/o V5.0 (Scollie et al., 2005) for variable CR circuit types.

Some professionals argue that the normalization approach may not maximize speech intelligibility when compared to procedures whose fitting goal is to assure that all the frequency bands of speech are amplified to be equally loud at the MCL (i.e., "equalization" approach). Examples of the new procedures using this approach include NAL-NL2 (Keidser et al., 2011) and DSL i/o V5.0 (Scollie et al., 2005) for fixed CR circuit types.

In this section, we provide a brief overview of the two procedures (NAL-NL2 and DSL i/o V5.0) that are the most widely used to verify the performance of nonlinear hearing aids.

NAL-NL2

NAL-NL2 is the most recent attempt to provide a tool to assist audiologists in fitting nonlinear hearing aids more accurately (Keidser et al., 2011). This procedure is reportedly based on the principle of providing a frequency-gain response that maximizes speech intelligibility, while keeping the overall loudness of the input signals at a level that is no greater than that perceived by a listener with normal hearing. In comparison to the previous prescriptive formula (e.g., NAL-NL1) that was described in the previous edition of this chapter, NAL-NL2

- Provides a lower CR when fitted with fast acting compression, but a higher CR can be used in hearing with slow-acting compression for patients with severe-to-profound HL.
- Provides overall gain that is 2 dB lower for female patients than male patients.
- Recommends correction for binaural summation of 2 dB for input levels less than 50 dB SPL and 6 dB for input levels greater than 90 dB SPL. Thus, a bilateral fit has a greater CR than a monaural fit.

- Provides gain adaptation (i.e., increasing gain over time) for new users with moderate-to-severe hearing loss.
- Prescribes less overall gain for an input level of 65 dB SPL than NAL-NL1 for mild-to-moderate hearing loss (i.e., NAL-NL2 provides slightly less gain and a higher CR than was prescribed by NAL-NL1).
- Prescribes a slightly greater low-frequency gain for patients whose primary language is "tonal" (e.g., Chinese, East Asia).
- Prescribes a slightly "flatter" frequency response with greater gain in the low and high frequencies and slightly less gain in the mid frequencies.

To use the NAL-NL2 program, the audiologist performs the following:

a. The audiologist first clicks the "Client Data" tab to enter the patient's gender, level of experience with hearing aids (new or experienced), language (tonal or nontonal), and date of birth. Based on these entries, "corrections" are made within the software to generate the prescribed gain (REIG) or output (REAR).
b. The next step is to click on the "Audiologic Input" tab and proceed to enter the transducer used to assess hearing thresholds (supra-aural earphone, insert earphone, loudspeaker), input value (dB HL, dB SPL in the ear canal or sound field, or nHL), hearing thresholds at 250 to 8,000 Hz for air conduction and bone conduction, predicted or measured real ear to coupler difference (RECD for threshold purposes or hearing aid purposes), REUG (predicted or measured), and REDD (predicted or measured). The RECD is the difference between the output of a hearing aid (or insert earphone) measured in the ear canal versus what is measured in a 2-cm^3 coupler. The RECD is affected by individual differences in ear canal volume and tympanic membrane impedance. Finally, the predicted (e.g., average) values for RECD and REUG vary as a function of age and gender.
c. The third step is to click on the "Section Screen" tab to enter the type of hearing aid (CIC, ITC, ITE, BTE), number of hearing aids being dispensed (unilateral or bilateral to correct for binaural summation), number of compression channels (1 to 18 to correct for channel summation), fitting depth (shallow, standard, or deep to correct for the remaining residual volume between the tympanic membrane and the tip of the hearing aid or earmold), type of tubing (No. 13, Libby 3 mm, Libby 4 mm, thin tube, or RIC), venting (occluded, tight, closed dome, 1, 2, 3 mm, open dome), and compression speed (slow, fast, dual, intermediate, or adaptive). In this section, the software provides suggestions on the "correct" manner to select tubing and venting based on the entered data.
d. The fourth tab is the "Target" screen. With this screen, the audiologist can "instruct" the software to display target curves for input levels of 50 to 90 dB SPL, type of measure (REIG, REAG, SPL-O-GRAM (e.g., REAR) real-ear input/output, coupler gain, coupler input/output, audiogram, ear simulator gain or input/output, aided sound-field thresholds, RESR, or coupler OSPL90), and Cf for as many as four channels if the number of compression channels of the dispensed hearing aid was documented in the "compression channel" selection in the "Selection Screen" tab. Finally, for each compression channel, the CT and CR are documented for the selected input levels which are typically 50, 65, and 80 dB SPL. Finally, the audiologist can view how the NAL-NL2 prescription compares to NAL-RP, POGO II (Figure 41.6), and IHAFF. There are past prescriptive formulae for linear (NAL-RP and POGO II) and nonlinear signal processing (IHAFF).
e. The final tab is the "Speech Screen" where the audiologist can view the calculated SII (0.00 to 1.0) based on how the aided speech spectrum falls above or below the patient's thresholds (i.e., audibility) for input levels from 55 (soft) to 75 dB SPL (loud).
f. Finally, clicking on the "Preferences" under "File" the audiologist can set
    a. The CT for wide dynamic compression between 0 and 100 dB SPL with the default set @ 52 dB SPL.
    b. The method of limiting between multichannel, wideband (default), or none.
    c. The status of the reference microphone of the real-ear analyzer to head surface/control microphone on (default) or undisturbed field/reference microphone off. Also, the audiologist may set the azimuth of the loudspeaker for REM to 0° (default) or 45°.
    d. The type of target to REIG (default) or REAG.
    e. The input levels to 40 to 90 dB SPL or 50 to 90 dB SPL (default).

DSL i/o Version 5.0

This is a comprehensive software-based program (Scollie et al., 2005) designed to help audiologists select and verify the performance of linear and nonlinear hearing aids. The primary goal of DSL i/o V5.0 is to place conversational speech in the patient's most comfortable listening range. The comfortable listening range targets are approximately midway between the participant's threshold of audibility and the predicted (or measured) upper limit of comfort (one standard deviation below LDLs, as reported by Pascoe, 1988).

To use the DSL i/o V5.0 program the audiologist follows the following procedure:

1. The audiologist accesses the "Assessment" tab to enter the patient's age (years or months), circuit type (linear or WDRC), patient type (pediatric or adult), style (BTE, ITC, ITE, CIC deep, CIC shallow, body), venting size (none, 1, 2, 3.5 mm, custom, or open), bilateral or unilateral fit, type of transducer (insert earphone using an E-A-R or immittance tip, insert earphone using a personal earmold, conventional TDH, loudspeaker at 0°, 45°,

or 90° or real ear in dB SPL), RECD type (HA-1 coupler tip, HA-2 coupler tip, HA-1 mold, or HA-2 mold), REUG (at 0°, 45°, or 90°), air conduction and bone conduction hearing thresholds (dB HL) at 250 to 6,000 Hz, LDL (dB HL) at 250 to 6,000 Hz, RECD, REDD, and REUG at 250 to 6,000 Hz, number of compression channels (1 to 17), and Cf.

2. The next step is to click on the "Target and Output Data" tab to select the input level for Target 1 (usually 50 dB SPL; alternative range is 45 to 59 dB in 1 dB steps), Target 2 (usually, 65 dB SPL; alternative range is 60 to 70 dB SPL), and Target 3 (usually 80 dB SPL; alternative range is 71 to 84 dB SPL), program 1 and 2 type (quiet and noise), verification signal (speech, speech noise, or puretone), and output (REAR, REAG, REIG, or sensation level). At this point the prescribed targets will be generated for the input levels that were selected, as well as the $RESR_{90}$ for a 90-dB input. In the lower section of this screen, targets will be created if the measures were made in a coupler (gain or output) for the same three input levels and for OSPL90 (output saturation level for a 90-dB input).

Once these data are entered, the software illustrates the auditory area between threshold (lower limit) and LDL (upper limit) in dB SPL near the tympanic membrane. In DSL i/o V5.0, the "target" is the auditory area in dB SPL that is measured near the tympanic membrane. The goal is to select hearing aids and then verify that the measured *output* is

- above threshold for a 50-dB SPL input signal,
- below LDL for a 80- to 90-dB SPL input signal, and
- between these two "targets" for a 60- to 70-dB SPL input signal.

"Smoothness" of REAR Measures for Multiple Input Levels

Earlier, we emphasized the need for hearing aid coupler measures to be as smooth for input levels of 80 to 90 dB SPL as for input levels of 50 to 60 dB SPL. The same goals also need to be achieved for hearing aid performance when actually worn by the patient. At this point, our emphasis shifts to observing the "smoothness" of the REAR at 50 to 80 dB SPL (Figure 41.16) for the goal of ensuring that the morphology of the $REAR_{80}$ curve is as smooth as the $REAR_{50}$ curve. If the $REAR_{80}$ curve is "jagged," then it has been suggested that the hearing aid is generating an excessive amount of intermodulation distortion (Revit, 1994).

$RESR_{90}$ (Real-Ear Saturation Response with a 90-dB Input)

With the hearing aid still in place and the volume control at the same position, we measure the $RESR_{90}$ using a 90-dB puretone sweep (200 to 8,000 Hz) to ascertain the SPL near the tympanic membrane. At the completion of the sweep, we observe if the measured $RESR_{90}$ is below the LDL measured at the time of the initial evaluation. If it is, this assures the audiologist that intense environmental sounds should not be perceived as uncomfortably loud (Munro and Patel, 1998). As mentioned earlier, Figure 41.5 illustrates the $RESR_{90}$ in relation to the measured LDL ("dots") at 500 to 4,000 Hz for a patient. Note, at each test frequency, the measured $RESR_{90}$ (thin line) is below the measured LDL. The "dots" with an arrow pointing up indicate that the measured LDL_{spl} was greater than 120 dB SPL at that frequency. According to Munro and Patel (1998), if the results reported in Figure 41.5 are achieved, the audiologist can be reasonably assured that environmental sounds at high input levels will not be judged as uncomfortable by the patient.

Verification of Performance of Directional Microphones

Figure 41.21 illustrates the use of 2-cc coupler measures and Figure 41.22 illustrates the use of REM to verify that the directional microphone is performing correctly. In the first author's experience, it is not uncommon to receive new hearing aids where either (a) the function of the microphones is reversed so that the rear microphone is amplifying and the front microphone is attenuating or (b) the rear microphone is not working at all, resulting in the hearing aid having only omnidirectional capability. In Figure 41.22, the top curve was measured with the patient facing the real-ear loudspeaker (0°) and the signal (modulated ANSI composite) presented at 65 dB SPL. With the signal remaining on, the patient is slowly rotated so the rear microphone is facing the loudspeaker. As the patient is rotated (making sure the distance between the loudspeaker and the microphone is the same for this measure as it was for the 0° measure), the audiologist views the real-ear system monitor to determine the azimuth where there is the least amount of amplification. In Figure 41.21, this occurred at 135° (hypercardioid polar design) showing a 20- to 25-dB decrease in gain when the rear microphone was facing the loudspeaker in comparison to when the front microphone was facing the loudspeaker. This verifies that the rear microphone is working properly. In cases where the rear microphone is not working, the front and rear measures will superimpose suggesting no reduction in gain caused by the activation of the rear microphone (omnidirectional performance). In the case where the microphone function is reversed, the 0° curve would be an example of a measure for the rear microphone and the 135° would be an example of a measure for the front microphone.

Verification of Feedback

Figure 41.23A to C illustrates how REM can be used to verify the presence of feedback. In Figure 41.23A, the REAR reports that no feedback was present. Figure 41.23B reports the beginning of the presence of feedback at ~4,000 Hz as

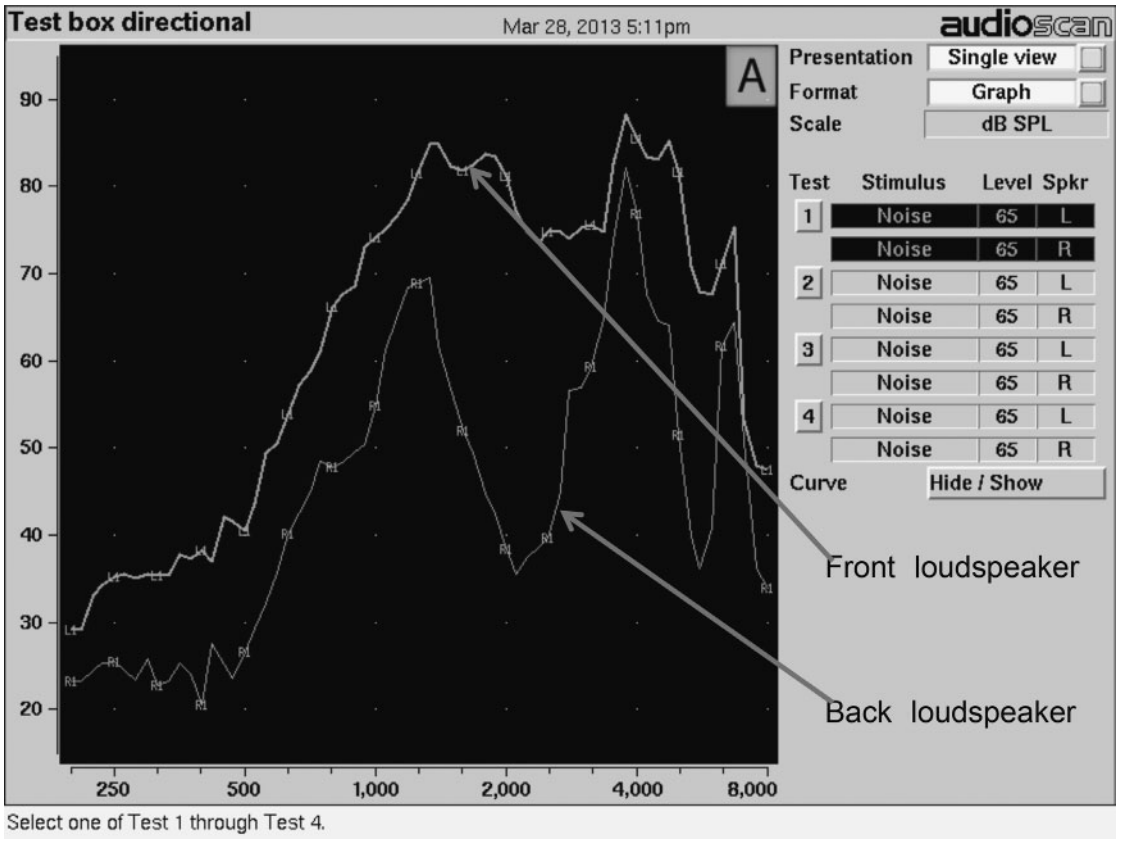


FIGURE 41.21 Using 2-cc coupler measures to verify the performance of a directional microphone.

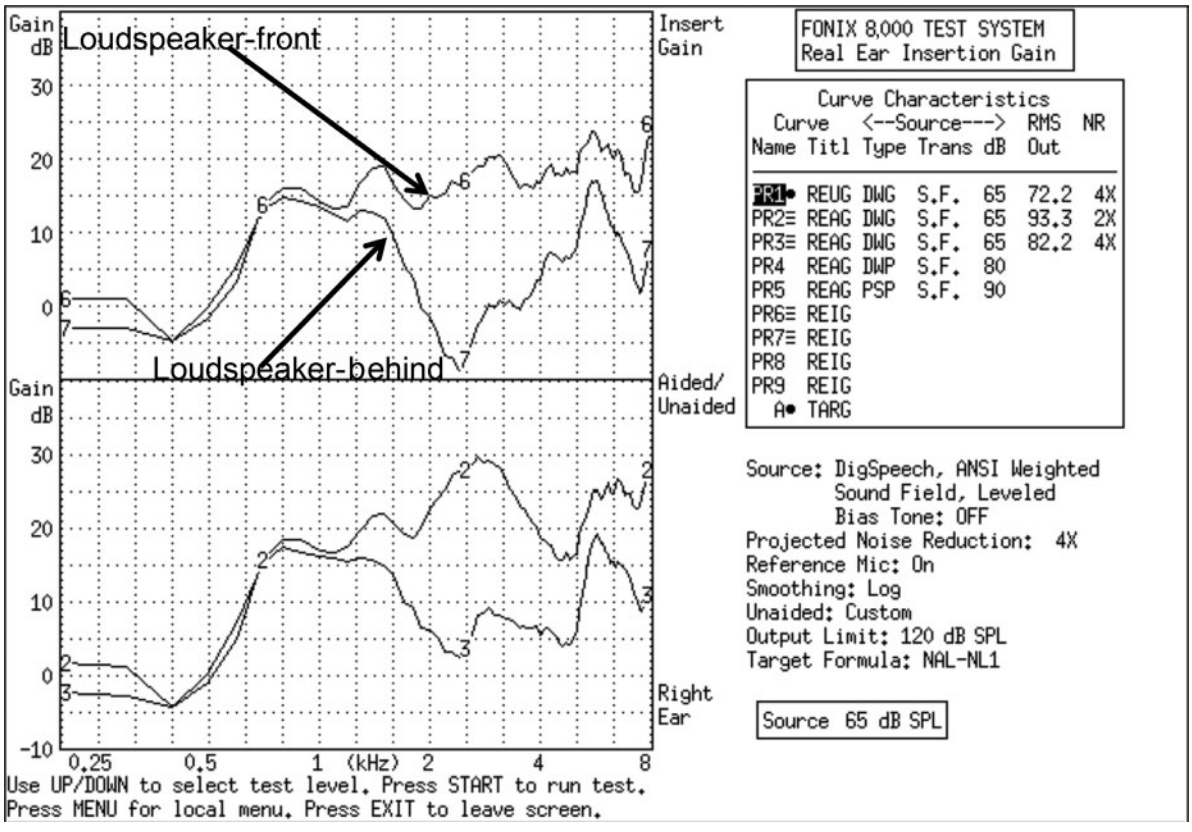
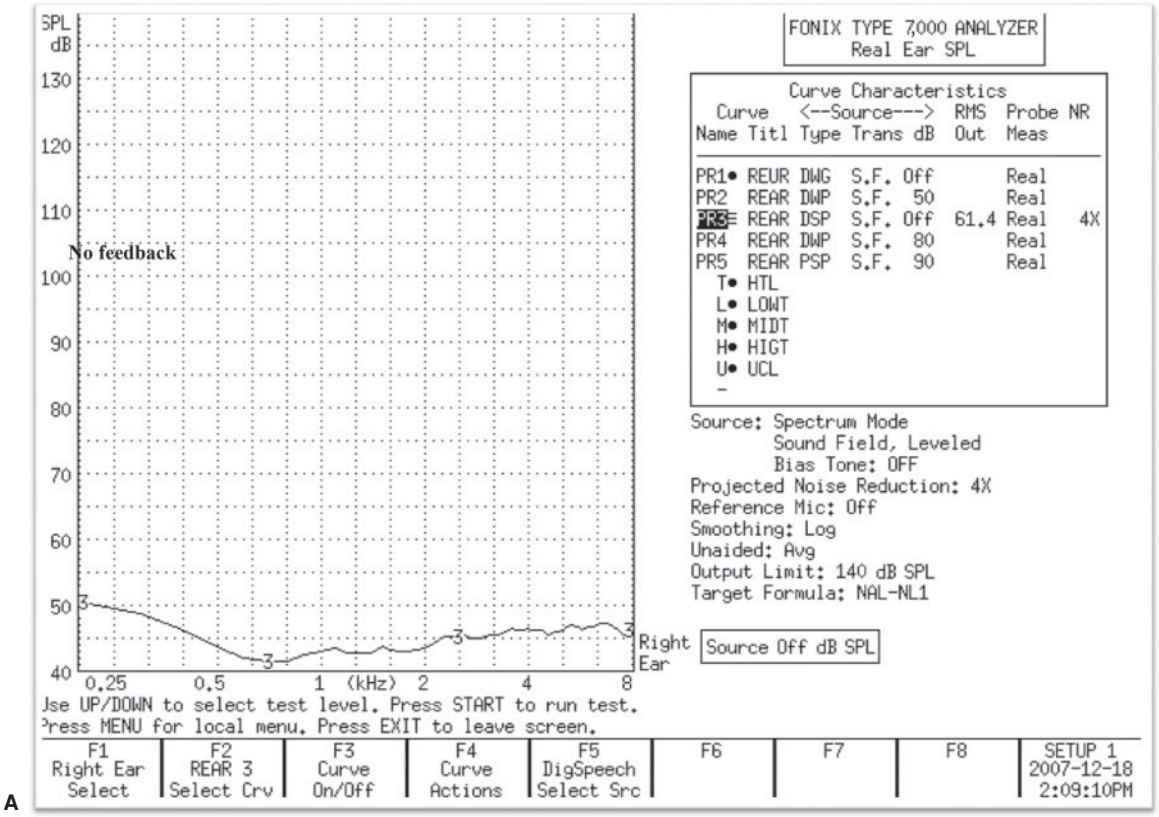
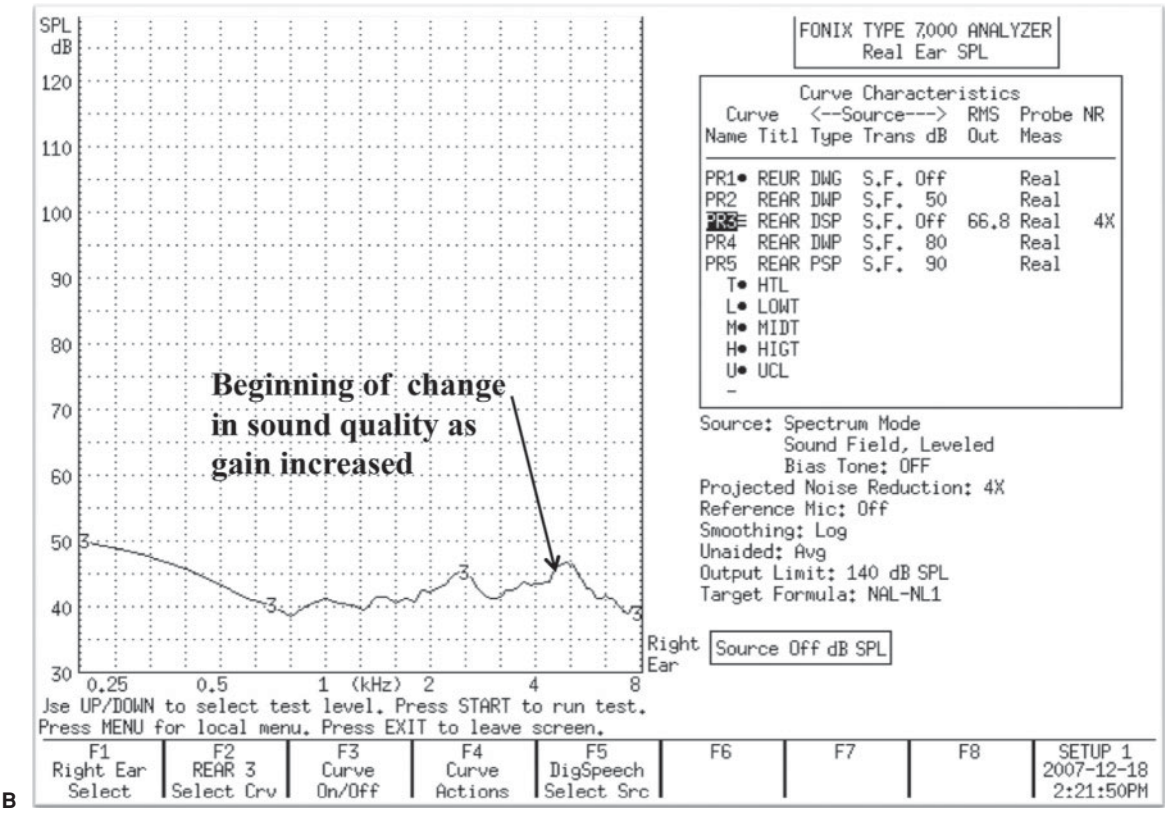


FIGURE 41.22 Using real-ear measures to verify the performance of a directional microphone.

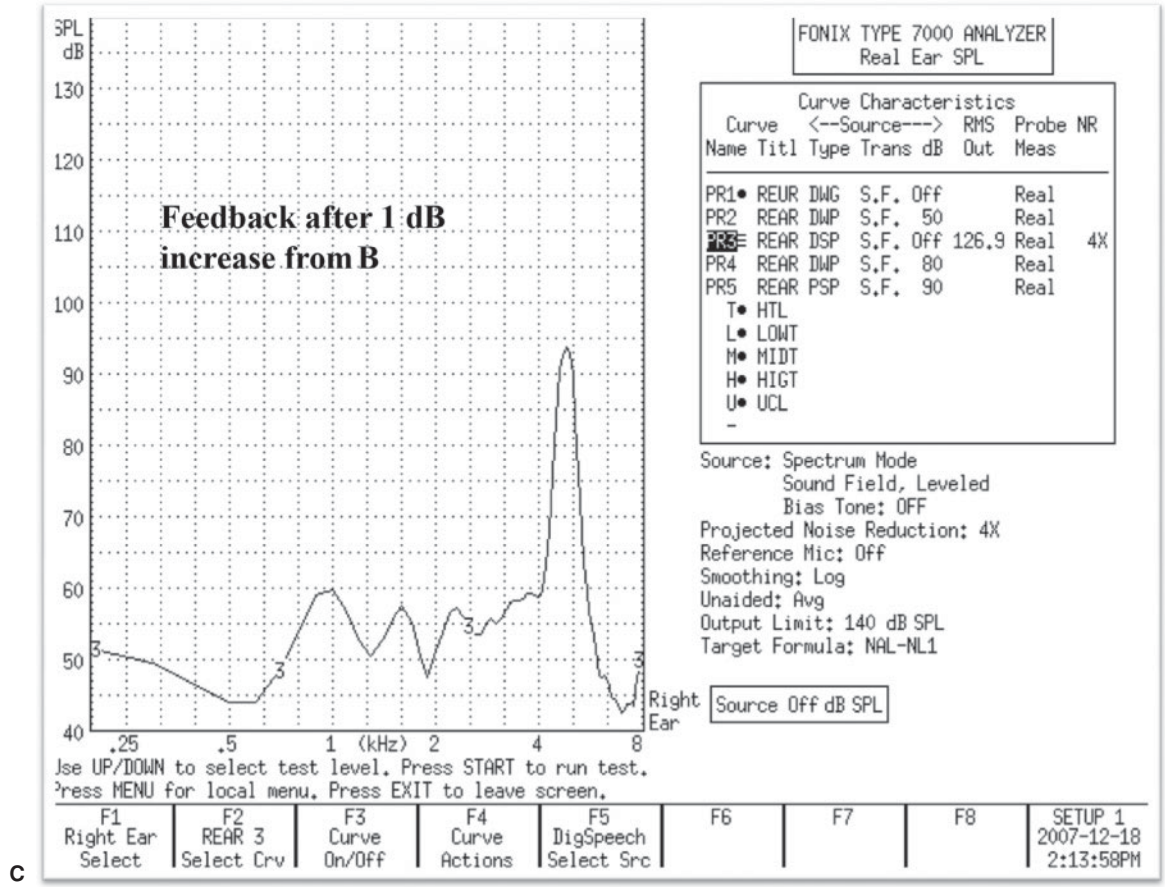


A



B

FIGURE 41.23 Using coupler measures to verify the presence of feedback for a DSP hearing aid. [**A**] The REAR with no feedback present. [**B**] The beginning of the presence of feedback at ~4,000 Hz as the volume control was increased. [*continued*]



C

FIGURE 41.23 [*Continued*] [**C**] The obvious presence of feedback at ~4,000 Hz as the volume control was increased by 1 dB.

the volume control was increased. Figure 41.23C reports the obvious presence of feedback at ~4,000 Hz as the volume control was increased by 1 dB.

Loudness Judgments for Speech

In the previous section, we described a method to validate that a frequency-specific signal (puretone sweep), presented at a high input level (90 dB SPL), was not uncomfortable to the patient. In the "real world" the listener is often exposed to varying levels of speech, which have a much broader bandwidth than frequency-specific stimuli. Therefore, it is important to include in the protocol a method to assess loudness judgments for a "speech-like" signal.

To accomplish this goal in a clinically efficient manner, we present speech composite–noise from a real-ear analyzer at 50, 65, and 85 dB SPL. While wearing both aids for a bilateral fit (or one aid for a monaural fit), we ask the patient to judge the loudness of the speech-weighted noise using the same loudness scaling categories described earlier. If the hearing aids are adjusted properly, the patient should rate the 50-dB SPL input as either "very soft," "soft," or "comfortable, but slightly soft." For an input level of 65 dB SPL, the patient should rate the loudness as "comfortable, but slightly soft," "comfortable," or "comfortable, but slightly loud." For the input level of 85 dB SPL, the patient should never report a rating of "uncomfortably loud." If the patient reports the high input level to be "uncomfortably loud," then the audiologist must consider reducing the output and/or CT. Another alternative would be to provide a more aggressive CR.

Aided Sound-Field Thresholds

One quick and reliable measure is to obtain aided sound-field thresholds using warble tones at 250 to 8,000 Hz presented in a calibrated (ANSI, 2004) sound field. The patient, facing the loudspeaker, is asked to press a button when he/she hears a sound no matter how loud or soft. Research has suggested that if the aided sound-field threshold is 20 dB HL or better, this is indicative that the patient can hear the softest components of speech (Skinner et al., 2002).

Hearing Aid Counseling: Use and Care of the Hearing Aids

Assuming that the fitting goals have been achieved, we then counsel the patient on the use and care of the hearing aids.

Items typically covered include informing the patient about the following crucial areas:

1. The length (1 to 3 years) and terms of the warranty (damage, or loss and damage). The patient is informed that in 1 year a card is sent as a reminder of the need to check his/her hearing and the function of the hearing aids.
2. The audiologist provides the patient with the option of being scheduled to return at 3, 4, or 6 months for a hearing aid check. At this visit, the hearing aid(s) are dehumidified, earmolds cleaned, thin tube replaced, filters replaced, battery compartment cleaned, and 2-cc coupler performance completed. The hearing aid performance is compared to the initial coupler analysis measured at the initial "user" setting.
3. It is important to remind the patient that the hearing aids are purchased on a 30-day trial (varying across different states) period. If the patient should decide to return the hearing aids, then the patient will receive a full refund minus a small "professional fee."
4. The patient is instructed regarding the operation of the volume control for those hearing aids that have volume controls.
5. The patient is instructed regarding the operation of the remote control (volume control, switching between programs, and other features) if the hearing aids use a remote control.
6. Counseling takes place related to operation of any buttons or switches that may be necessary to operate the hearing aids.
7. The patient is instructed regarding the insertion and removal of the batteries. The patient is counseled to be sure the voltage of the batteries is 1.45 V, because it has been the experience of the authors that batteries with a voltage less than 1.45 may lead to intermittent performance of the hearing aids.
8. The type of battery and expected battery life are explained. We also counsel the patient if the hearing aids have a feature informing when the battery drainage is low.
9. The patient is instructed regarding the insertion and removal of the shells or earmolds.
10. The patient is instructed regarding the problems related to moisture. Each patient is provided a Zephry® Dry & Store at the time of the fitting. If the patient has been dispensed BTEs, then an air blower is provided, which is used to quickly remove moisture from the tubing.
11. Problems related to cerumen plugging the vent and/or sound channel should be explained. Counseling is also extended to other options for combating cerumen. These may include extended receiver tubing, spring guards, cerumen filters, and tools to remove cerumen from the vent. The patient is also counseled on the correct use of the brush and wax removal device accompanying most custom products.
12. Use of the telecoil is an important area requiring counseling. Whenever possible, the patient is counseled on using the microphone of the hearing aid as an "acoustic" telecoil. Typically, we dial the local weather-line and observe as the patient typically moves the telephone receiver around the entrance to the ear canal and pinna. We counsel the patient on the need to place the receiver of the telephone adjacent to the microphone of the hearing aid. In many cases, this is the position typically preferred by patients for using the telephone with hearing aids. When this is not successful, we then counsel the patient on the operation of the t-coil switch on the hearing aids. Several programmable hearing aids allow the audiologist to program a stronger telecoil response. Recently, several manufacturers have incorporated the EasyPhone feature into their hearing aids. With this technology, the hearing aid automatically switches to telecoil when the circuit detects an electromagnetic signal and back to microphone when it detects that the electromagnetic signal is no longer present (Puttermann and Valente, 2012).
13. Discussion of HAT and aural rehabilitation (our facilities have full-time audiologists providing aural rehabilitation).
14. Finally, we will call the patient in 2 days to determine how the patient is doing; and the patient is scheduled to return within 4 weeks for a hearing aid assessment.

OUTCOME MEASURES (FOURTH VISIT)

Hearing Aid Assessment

At this visit, we are interested in the patient's overall satisfaction with the hearing aids. For example, we want to know how well the patient performed during the intervening 3 to 4 weeks, as he/she listened to speech in a variety of listening situations. Other questions relate to judgment of sound quality; presence/absence of feedback; ease of communication on the telephone; ability to remove and insert the earmolds, hearing aids, or batteries; the duration of battery life; issues related to the comfort of the hearing aids; and issues related to the presence or absence of the occlusion effect. It is during this interview process that decisions are made relative to the need for readjusting the electroacoustic characteristics or transmission line characteristics of the hearing aids.

At this visit, the patient is also asked to complete the "aided" portion of the COSI or Washington University questionnaires (Figure 41.1).

CONCLUSIONS

In 1998, ASHA published the "Guidelines for Hearing Aid Fitting for Adults." This chapter's first author had the honor

of chairing that committee. Recently, the first author had the pleasure of chairing a national Task Force for the American Academy of Audiology (AAA) to develop a national guideline for selecting, sitting, verifying, and validating the hearing aid performance in adults using evidence-based principles based on (Valente, 2006). In all of these guidelines, several important points were made:

1. REM is the preferred method for verifying the performance of hearing aids. Unfortunately, however, approximately 25% to 30% of audiologists do not routinely verify the performance of hearing aids using REM (Mueller and Picou, 2010). This is in spite of the fact that recent research (Abrams et al., 2012) clearly demonstrated that 15 of 22 participants preferred the fitting using REM with the hearing aid programmed to NAL-NL2 when compared to the manufacturer first-fit.
2. Patients need to be counseled on the realistic benefits to be derived from hearing aids.
3. LDLs should be directly measured using frequency-specific stimuli when possible to accurately assess/adjust the output and/or compression characteristics of the hearing aids.
4. Outcome measures need to be included in the hearing aid fitting process.
5. HAT needs to be integrated into the fitting and counseling process.

This chapter has provided a comprehensive overview of procedures to select and fit hearing aids for adults. Many of the requirements of the guidelines suggested by ASHA (1998) and AAA (2006) are included in the procedures outlined in this chapter. We feel that incorporating some or all of these suggestions has a high probability of resulting in a successful hearing aid fitting.

FOOD FOR THOUGHT

1. What is the difference between verification and validation?
2. What is the goal for varifying the increased RESR90?
3. What is the primary purpose of measuring ANSI S3.42-1992?

KEY REFERENCES

thePoint A full list of references for this chapter can be found at http://thePoint.lww.com. Below are the key references for this chapter.

Abrams H, Chisolm T, McManus M, McArdle R. (2012) Initial fit approach versus verified prescription: comparing self-perceived hearing aid benefit. *J Am Acad Audiol.* 23, 768–778.

American National Standards Institute. (1992) *American National Standard for Testing Hearing Aids with a Broad-Band Noise Signal (ANSI S3.42-1992).* New York: Acoustical Society of America.

American National Standards Institute. (2003; R 2009) *American National Standard: Specification of Hearing Aid Characteristics (ANSI S3.22-2003; R 2009).* New York: Acoustical Society of America.

American National Standards Institute. (2004) *American National Standard: Specification for Audiometers (ANSI S3.6–2004).* New York: Acoustical Society of America.

American Speech Language Hearing Association. (1998) Guidelines for hearing aid fitting for adults. *Am J Audiol.* 7, 5–13.

Cox RM, Alexander GC. (1995) The abbreviated profile of hearing aid benefit. *Ear Hear.* 16, 176–186.

Cox RM, Alexander GC. (2001) Validation of the SADL questionnaire. *Ear Hear.* 22, 151–160.

Dillon H, James A, Ginis J. (1997) Client oriented scale of improvement (COSI) and its relationship to several other measures of benefit and satisfaction provided by hearing aids. *J Am Acad Audiol.* 8, 27–43.

Dillon H, Keidser G, Ching T, Flax M, Brewer S. (2011) NAL-NL2 prescription procedure. *Phonak Focus.* 40, 1–10.

Erdman SA, Demorest ME. (1990) *CPHI Manual: A Guide to Clinical Use.* Simpsonville, MD: CPHI Services.

Gatehouse S. (1999) Glasgow hearing aid benefit profile: derivation and validation of a client-centered outcome measure for hearing services. *J Am Acad Audiol.* 10, 80–103.

Giolas TG, Owens E, Lamb S, Schubert E. (1979) Hearing performance inventory. *J Speech Hear Disord.* 44, 169–195.

Hawkins D, Montgomery A, Prosek R, Walden B. (1987) Examination of two issues concerning functional gain measurements. *J Speech Hear Disord.* 52, 56–63.

Jenstad L, Van Tasell D, Ewert C. (2003) Hearing aid troubleshooting based on patients' descriptions. *J Am Acad Audiol.* 14 (7), 347–360.

Keidser G, Dillon H, Flax M, Ching T, Brewer S. (2011) The NAL-NL2 prescription procedure. *Audiol Res.* 1324, 88–90.

Kochkin S. (2000) Customer satisfaction with single and multiple microphone hearing aids. *Hear Rev.* 7 (11), 24, 26, 28–29, 32–34.

Kuk F, Keenan D. (2005) Efficacy of an open-fitting hearing aid. *Hear Rev.* 12, 26–30, 32.

Lewis M, Crandell C, Valente M, Horn J. (2004) Speech perception in noise: directional microphone versus frequency modulation (FM) systems. *J Am Acad Audiol.* 15, 426–439.

Mueller G, Picou E. (2010) Survey examines popularity of real-ear probe microphone measures. *Hear J.* 63 (5), 27–32.

Munro K, Patel R. (1998) Are clinical measurements of uncomfortable loudness levels a valid indicator of real-world auditory discomfort? *Br J Audiol.* 32, 287–293.

Newman CW, Weinstein BE, Jacobson GP, Hug GA. (1990) The hearing handicap inventory for adults: psychometric adequacy and audiometric correlates. *Ear Hear.* 11, 430–433.

Pascoe D. (1988) Clinical measurement of the auditory dynamic range and their relation to formulas for hearing aid gain. In: Jensen J, ed. *Hearing Aid Fitting, Theoretical and Practical Views.* Copenhagen: Danavox Jubilee Foundation; pp 129–152.

Puttermann D, Valente M. (2012) Differences between the default telecoil (t-coil) and programmed microphone frequency response in behind-the-ear (BTE) hearing aids. *J Am Acad Audiol.* 23, 366–378.

Revit LJ. (1994) Using coupler tests in the fitting of hearing aids. In: Valente M, ed. *Strategies for Selecting and Verifying Hearing Aid Fittings.* New York: Thieme Medical Publisher; pp 64–87.

Scollie S, Seewald R, Cornelisse L, Moodie S, Bagatto M, Laurynagay D, et al. (2005) The desired sensation level multistage input/output algorithm. *Trends Amplif.* 9, 159–197.

Skinner M, Binzer S, Potts L, Holden L, Aaron R. (2002) Hearing rehabilitation for individuals with severe and profound hearing impairment: hearing aids, cochlear implants, and counseling. In: Valente M, ed. *Strategies for Selecting and Verifying Hearing Aid Fittings.* 2nd ed. New York: Thieme Medical Publishers; pp 311–344.

Stone MA, Moore BCJ. (2002) Tolerable hearing-aid delays: II. Estimation of limits imposed during speech production. *Ear Hear.* 23 (4), 325–338.

Stone MA, Moore BCJ. (2005) Tolerable hearing-aid delays: IV. Effects on participative disturbance during speech production by hearing-impaired participants. *Ear Hear.* 26 (2), 225–234.

Summerfield Q. (1992) Lipreading and audiovisual speech perception. *Philos Trans R Soc Lond Biol Sci.* 335 (1273), 71–78.

Valente M. (2006) Audiological management of adult hearing impairment. *Audiol Today.* 18 (5), 32–36.

Valente M, Fabry D, Potts L. (1995) Recognition of speech in noise with hearing aids using dual-microphones. *J Am Acad Audiol.* 6, 440–449.

Valente M, Mispagel K. (2008) Unaided and aided performance with a directional open fit hearing aid. *Int J Audiol.* 47, 329–336.

Valente M, Potts L, Valente M. (1997) Differences and interparticipant variability of loudness discomfort levels (LDL) measured in sound pressure level and hearing level for TDH-50P and ER-3 A earphones. *J Am Acad Audiol.* 8, 59–67.

Valente M, Valente M, Goebel J. (1991) Reliability and interparticipant variability of the real ear unaided response REUR. *Ear Hear.* 12, 216–220.

CHAPTER 42

Building and Growing an Audiologic Practice

Melanie Herzfeld

CHAPTER OVERVIEW

This chapter will describe the skills involved in building and growing an independent audiologic practice and will include the personal experiences of an audiologist who has successfully navigated the business of practice management. The process described here focuses on independent or private practice and also applies to an audiologist charged to create and maintain a practice within an existing setting (e.g., medical group, hospital, foundation). After a short history of the evolution of independent practice, attention is directed to some of the variables involved with establishing and growing a business: Personality types that do well in business, initial decisions (location, financing, taxes, insurance, etc.), marketing, and more. The business of audiology is an important specialty, and all audiologists should have a working understanding of the operational challenges of managing one's own practice.

A BRIEF HISTORY

To be in private practice in audiology seems to be a very coveted goal for many newer graduates. It was not always this way. In fact, for many years, there was no private practice in audiology. There were no hearing aid sales by audiologists, no products being dispensed. Historically, audiology began during World War II to serve the needs of the military (Hosford-Dunn et al., 1995). After the war, the profession moved to the universities, which led to a graduate school curriculum being developed; the first school offering training was Northwestern University in 1946. Even the term *audiologist* is of relatively recent vintage, having been coined in the late 1940s by Hallowell Davis (Galambos, 1998). It is sometimes hard to imagine that this relatively new field is now recognized as one of the most rewarding professions in the United States (CareerCast.com, 2013).

Audiologists in the 1940s, 1950s, and 1960s were involved in research, taught at universities, or delivered clinical services, but had nothing to do with dispensing products. The only professional organization at the time was the American Academy of Speech Correction (which is now the American Speech-Language Hearing Association (ASHA), n.d.). Hearing aids were fit and sold by hearing aid dealers. Audiologists were the testers, researchers, and rehabilitationists, but were not independent managers of a patient's hearing loss. Their role was less autonomous and, resulting in audiologists being assigned as a provider of ancillary services, which usually infers being under the supervision of a physician. At the time, audiology and speech pathology had much in common, so the two professions were teamed together in one organization.

During these years, audiologists were prohibited from dispensing hearing aids or from making any profit on sales and, interestingly, rehabilitation actually fell by the wayside as the primary focus of audiology became testing. Audiologists who bucked the system and began dispensing aids were stripped of their ASHA membership and considered unethical in practice. However, audiologists began to demand the right to exercise their professional responsibilities associated with dispensing amplification devices, and thus in 1977 the Academy of Dispensing Audiologists was formed (http://www.audiologist.org/about-ada). Around that same time period, ASHA agreed to retract its policy of considering the practice of selling hearing aids as a violation of ethics.

Audiologists also began emphasizing the need for further education, which led to the development of the clinical doctorate (American Academy of Audiology, 1991). Private practice grew exponentially from the time dispensing was allowed. However, in the past few years, the number of independent practices has declined as practices are joining big corporate groups. At one point, individually owned practices accounted for about half of the provider locations in the United States; however, as of 2011, this percent had declined to under 25%. Corporate affiliates and corporate-owned locations have become the largest percentage of provider locations (Smirga, 2011). But private practice still remains a goal for many of the students graduating from today's AuD programs. Their perception of private practice, however, has changed from total autonomy to allowing some affiliation with corporate partners.

PRIVATE PRACTICE AND PERSONALITY TYPES

There are presently many work options for audiologists. The advantages of private practice are that the audiologist

has more autonomy and can develop a practice that meets the needs of the independent practitioner. As attractive as this may sound, it is not for everyone.

Who should/should not be in private practice? Private practice requires the audiologist to function independently. This does not mean one must know everything, but instead that one is comfortable with the burdens resting solely on his or her shoulders. A practice demands a great deal of attention, interest, and stamina of the owner. It is the proverbial "the buck stops here" experience. One must be comfortable not only with dealing with patient needs and physician requirements, but also with handling financial services, negotiating leases and contracts, and managing employment issues and staff challenges. It is not for the faint of heart. But it can be a most rewarding job!

Personality testing might help the audiologist decide if private practice is appropriate as a career choice, although most people know how risk averse, how independent, and how comfortable they are with change and with responsibility. The vast majority of audiologists are in the caring field because they want to help others, but can they also accept risk? Can they function independently, deal with business complications, staffing problems, location issues, and supplies, and still manage the patients?

One might begin by taking the online Keirsey Temperament sorter (http://www.keirsey.com/sorter/instruments2.aspx?partid=0). This 71-question inventory identifies four temperament types: Artisan, guardian, idealist, and rational. Audiologists tend to be guardians (K. English, personal communication, June 10, 2013) because caring and belonging are trademarks of this personality type; for those who enter this field, these are typical traits. We want to help our patients! These personality types are then further divided into four divergent preferences: Extrovert/introvert, sensing/intuiting, thinking/feeling, and judging/perceiving (see Figure 42.1). Historically, students in health professions such as audiology fell into the intuiting/feeling temperament type but they have transformed, probably with the advent of private practice models, to the sensing/judging (SJ) type. Interestingly this seems to be true of many health professions (Baggs, 2012). The SJs present themselves as being reliable, delivering service well, and being respected. Those who are extroverted, sensing, thinking, and judging are often found in business. Those who are extroverted, sensing, feeling, and judging are often found in health care, dealing with people, but also may be business owners. Those who are introverted, sensing, feeling, and judging are the least likely to want to be leaders, although they enjoy helping make things run smoothly. It becomes clearer which personality type is most likely to succeed in private practice or perhaps even to want to take that step (Keirsey, 1998). In particular, extroversion seems to be a common trait, probably related to the social demands involved with marketing, staff management, attorney and accountant consultations, and landlord and contractor negotiations.

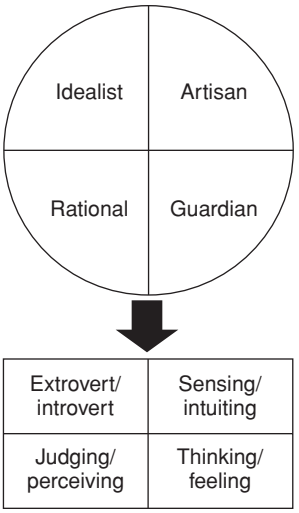


FIGURE 42.1 Personality and temperament: What is the right mix for business?

Like anything else, however, broad generalizations can only begin to help us understand complex issues such as practice ownership. Readers are advised to interview as many practitioners as possible when considering the option of practice management.

Audiologists in private practice have to be able to wear many hats. First and foremost they must be *competent,* that is, must have the requisite job skills to perform the task flawlessly. That is the technician part of their personality. Then they must be an *entrepreneur,* willing to look to the future, to plot and plan. And still they must be a *manager,* making sure that all business details are addressed and that the nature of the business success is defined and achievable (Gerber, 1995). Coming up with goals for the business which are realistic and manageable is key. It is not enough to say, "Business will be solicited," or "Time will be spent." It is necessary to consider each and every aspect of the business, to consider not just the test requirements but the patient needs, the referral source, and the community impact. Thus the personality tests may help give an overview of whether or not a particular audiologist has the necessary character traits to be a private practice owner or whether or not this person has the strength of character to deal with transforming his or her self to be able to do what needs to be done to be successful. Personality testing might offer some information of value, even if it is pointing toward skills which need development.

FIRST DECISIONS

Let's consider an audiologist who demonstrates the temperament to be in private practice or the strong will to try to succeed (which can be just as important). Where does he or she start? See Table 42.1 for a listing of many business considerations.

TABLE 42.1

Business Considerations for the Audiologist Considering Independent Practice

- Location
- Finances
- Space and equipment
- Personnel
- Marketing
- Local, state, and federal regulations

Location

Remember the real estate adage, "Location, location, location!" Location is one of the most important assets in a practice; to a very large extent, where one works determines one's patient base. The aspiring practice owner will first investigate practices already in the area, for instance, by drawing a circle on a map, conducting an Internet search, and driving around within that circle. How many practices exist, and who owns them? Consider their niches and what is unique about each practice. Find out if any of them are likely to be for sale in the near term. See if there is a missing element. For example, there may be 20 different hearing aid dispensing practices, but none specializing in pediatrics or central auditory testing. Perhaps tinnitus care is not provided in the area or perhaps vestibular testing is not in the mix. It is important to consider one's own professional interests, professional strengths, and the domains one would most likely want to practice. Once you have done that, look back around that drawn circle and at all of those practices. How close are they to medical offices, to highways? Perhaps it is advantageous to be near other professionals, for instance, a psychology practice or a pediatric office, or near the local otologist or the largest group of internal medicine providers, or to a senior center or senior housing, or even along a bus route. Think about how patients would get to each location and consider the positives and negatives of each; approach each of the existing practices from a patient's perspective. As an older patient, a younger patient, a driver, a nondriver: How would they get to that practice? Are there too many steps? Two flights up, no elevator? How convenient are they? How clean? How approachable?

Another way to approach this research is to put oneself in the mindset of a job search. Would you want to work here? Who would the patient base be? How would you get referrals? Would the patients you want to see want to come to this practice? What attracts you to want to work in this particular place? What makes you think this is not for you?

Financial Decisions

The audiologist considering independent or private practice will have a steep learning curve while adjusting to the business world. Table 42.2 lists just a few terms that represent "business vocabulary." Many decisions need to be made, including "to buy or not to buy."

TABLE 42.2

Important Business Vocabulary for the Private Practitioner

- Noncompete clause
- Practice valuation
- Buying groups
- Business plan
- Personal and professional liability

To Buy or Not to Buy?

Once one has seriously considered *where* one would like to practice, the next question is *how to obtain* that practice. Has the aspiring practitioner built up enough equity to start a practice from scratch or to purchase an existing practice? Is there a practice owned by an audiologist who might have an interest in selling sometime in the near future? Talking to the owners of existing practices might help in finding someone interested in selling in the next few years. That practice might be willing to take on an employee with an offer to buy into the practice over time. Just being involved in an ongoing practice one intends to purchase may allow the employee to develop a patient base, which might be helpful when it is time to talk about financing. This of course assumes the employee is becoming the owner of the same practice. At the same time, an employee must be aware of noncompeting clauses in contracts; frequently a condition of employment disallows starting a practice within a certain distance from the one in which the audiologist worked and is equally likely to disallow taking any patient records. However, buying into an existing practice has many advantages. Although it can be more difficult legally, it is certainly less risky to buy an existing practice.

Practice valuation for purchasing an existing practice requires the help of specific professionals. There are many ways to set a value on an existing practice, but this is best left to the experts.

Arranging Financing

Some practices are part of affiliated groups which will help the new owners finance the arrangements. There are buying groups which offer financing as well as guidance in running a practice. There are also franchise businesses. However, many practitioners need to obtain financing on their own. It is also possible that the current business owner will allow some financing help; again, this requires that the sale arrangement meet the legal requirements regarding interest and payment options.

To approach a bank through the Small Business Association (SBA), due diligence is required. To begin with, one cannot just show up and ask for a loan. In fact, audiologists have reported that they wanted to buy a practice already in existence, only to be told by the bank that they could not use that practice's statistics because they were not the owners and they had no track record with that practice. An audiologist planning to buy an existing practice needs to invest time in the practice in a way that is documented and transparent to financiers. It will be important to show a banker that he or she already has an established base of patients and is familiar with what needs to be done to keep and expand that base.

The Business Plan

The first item a financier will require is a business plan. Samples of business plans can be researched online for additional information on what is specifically needed or how best to present ideas. To get started, see "Helpful Websites" at the end of this chapter.

A business plan begins with a mission statement. The mission statement is a short statement of what one intends to do, why the business should be funded, and what the applicant has done to make sure that any investment in the practice would be a success. An analysis of the business opportunity, financial prediction, niche market approach, and potential referral sources are next. If there are 20 practices near the desired location, the applicant must indicate one's unique approach and the market trends that will support success. This is where research is vital. When buying a practice already in existence, what needs to be done to expand it? How much is the applicant personally investing?

My first foray into requesting money from SBA was for a practice in a shared space with some otolaryngologists. We had recently left a multispecialty medical group and were unable to contact our patient base because of legal requirements. Included in the business plan was a clear and concise description of how we would contact referring physicians, the advertising plan, and a very simple projection of income based on a review of our old data, including a "guesstimate" of the number of existing patients lost to location change, and a projection of new patient growth. Also included were all the costs in the practice analysis, including the amount the audiologist had to invest. Documentation was provided for new equipment and a rationale for the expenses. The banker was happy to discuss the plan, but ultimately indicated the plan was unlikely to be financed unless collateral was offered. However, the banker agreed to send the information along to the banking underwriters without mention of collateral. The banker was pleasantly surprised when the underwriters called her to say the author would receive the funds requested because the business plan was so specific and well written. They had decided the author was a good risk. According to the ADA Practice Model Task Force (2008), audiologists in private practice seem to have a very low loan failure rate, making audiologists a good loan risk.

When the author then decided 8 years later to venture into a completely independent practice, once again funding was needed, and the request was submitted with the same concise three-page documentation. However, this time the audiologist had a history of financial success and could show a profit and loss sheet for the past 8 years. More money was needed this time because of the need to pay for a complete build out (described in a later section), and once again, the banker was unsure if the loan request would be approved. Doubts arose not because of the track record but because of the current financial climate; money had become tight and he was not sure if the underwriters would fund the loan request without collateral. Once again, to the banker's surprise, the loan was funded and again the underwriters indicated the quality of the statement swayed them. This outcome reinforces the principle that the quality of the writing and the depth of information are vital to one's presentation.

Help from the Small Business Administration

The Small Business Administration has a tool to help with formulating a business plan. By registering on their site, one can use the SBA Business Plan Tool, which is a step-by-step guide to formulating a successful business plan. Categories or topics on the plan include the executive summary, the company description, market research, information on services and products, marketing plans, financial projections, and a summary statement (http://www.sba.gov/business-plan/2).

Professional Advice

The journey to independent practice must include help from other professionals. The importance of an experienced attorney, knowledgeable in the ways of starting up a business, and an equally qualified accountant, knowledgeable in projecting and writing a business plan, cannot be understated. These professionals will serve as a lifeline in the quest for private practice and will continue to help the audiologist even after the financing is complete. These professionals will explain why one would want to incorporate or not (discussed in the next section), and the differences within a limited liability company (LLC), a limited liability partnership (LLP), a subchapter S corporation (corporations that pass income, losses, deductions, and credit through to their shareholders for federal tax purposes), and incorporation in general (also detailed in the next section). They help predict tax consequences and plan for financial health. Some audiologists also use a real estate attorney to help them negotiate leases. The takeaway message is that hiring experienced and skilled professionals is invaluable. This is not the time to ask a favor of an uncle who does senior citizen tax returns or a patent attorney because he is a friend. Just as one would hope that

a patient would view the audiologist as the expert in one's area, so the audiologist will need to seek out help in matters of business, law, accounting, and other domains.

To Incorporate or Not

Speaking of incorporation, why incorporate? Again, this is a conversation best had with one's attorney because each state has different rules. In New York, for example, a professional wanting to have a corporate identity must apply to the state before becoming a professional corporation or limited liability professional corporation. The name of the corporation is tightly controlled and must be approved; the constitution of the corporate board (president, treasurer, secretary) is also tightly controlled. In a professional corporation in New York State, the shareholders are specified; a corporation can only offer shares to other audiologists or similarly licensed professionals. New York State business law is quite specific in this regard; in the business code section 1508, it states that "no individual may be a director or officer of a professional service corporation unless he/she is authorized to and engaged in the practice of his profession in that corporation." This is not a question of personal decision making but of state law, and thus must be considered in the decision-making process; again, having an expert on the team can make the difference in following laws and making appropriate decisions.

Limiting Liability and Tax Consequences

The general purpose for incorporating one's practice is to limit liability and tax consequences. Incorporating is intended to protect the owners in case of liability (e.g., malpractice, personal injury, damage to property); however, at this time it appears that liability limitations are limited more to financial obligations and less to personal liability. In a general incorporation, the tax rate is set at a specific level for the corporate entity and then any profit taken personally is taxed again at a personal rate; in essence this is a double taxation. However, if there is a financial loss, it cannot be passed onto the owners. Its advantages are that the business debt responsibility is limited to the business and not the shareholder and also that fringe benefits are deductible by the business. Shares offered in the corporation are always preferred shares.

In an S corporation, the profit is given to the shareholder personally and so taxes are paid only at the personal rate (http://www.irs.gov/Businesses/Small-Businesses-&-Self-Employed/S-Corporations). Again, there is limited personal liability for business debt unless the bank demands this, which they can do, by asking for a personal bond or guarantee. Fringe benefits are limited by law as to what can be a deductible expense and shares are common. Losses are limited to the extent of personal investment and the amount of income (http://smallbusiness.chron.com/personal-liabilities-s-corp-3696.html).

An LLP is a limited liability partnership and has an advantage in that liability is limited generally to the extent of investment in the partnership; in the case of malpractice, it protects the other partners from claims against the practicing partner. For example, if Dr. M is sued for malpractice, his partners in the LLP are not part of the suit; they did not see the patient who is suing and thus have no liability to pay in the case of a lost suit. However, depending on the state in which one practices, this type of corporation or professional arrangement may or may not be used by specific professionals. According to Legalzoom.com, an LLP must have two partners whereas an LLC may have only one overseer or owner. Clearly, obtaining an expert legal opinion on which status is appropriate for a practitioner's circumstances is quite important, since the decision has both financial and liability consequences. Although there are many ways to become incorporated, without professional guidance the wrong corporate identity might be selected.

Sole proprietorship is another option. This label applies to one owner of a business who has not chosen the route of incorporation. The name of the business may be a "DBA" (doing business as) and this might be the owner's name (e.g., Jane Smith, AuD, Audiologist) or a name designated to identify the business (Hearing and Tinnitus Center). The sole proprietor is still responsible for obtaining the appropriate licenses and registrations for both the business and the DBA. It is a simple way to run a business but has risks. Tax returns are simply done as a self-employed individual with all income and expenses listed as personal income/expense. There is no tax return due in the name of the business. The owner is, however, personally responsible for everything related to the business, whether debt or liability, for the problems associated with any employees, or with the business in general. It is also more difficult to obtain funding as a sole proprietor.

Regarding tax consequences, there are differences as well among these entities. Tax differences are not the only reason to select one option over another; it is simply a piece of the puzzle. For corporations which are not S corporations (not having shareholders), profits are taxed at the corporate rate and then taxed again when the profit is taken by the individual. For sole proprietorships and S corporations, the tax rate is the personal rate. What can and cannot be deducted depends on the business type, the number of employees, and state/federal laws. Again, the role of the accountant in helping chart a path through this accounting morass is invaluable.

SPACE REQUIREMENTS

To Share or Not to Share

After meeting with legal and financial advisors, the audiologist must decide where to house the practice. Options include sharing space with a medical practice—not only otology and otolaryngology, but also pediatrics, ophthalmology, optometry, and internist offices. One might open

an independent space or join a group of audiologists. Finding a psychologist or speech pathologist with whom to share space is also a possibility. Depending on the patient base, or projected base, these types of practice decisions might be mutually beneficial. A tinnitus practice with an in-house psychologist can be an advantageous joint venture, for example.

At the present time, several groups operate on a management basis or on a multilevel affiliation basis. There are buying groups, for example, which have a multilevel plan; some practitioners join and give full management rights to the buying group; others pay for the specific services they elect to use; and still others can simply be a user of the buying group services to obtain a discount on products. For some, joining with these groups can be helpful; they not only offer management support but may also offer funding opportunities in exchange for joining their buying group. Location is, however, still the most important decision to be made. As discussed previously, a private practice needs to be accessible, attractive, and appealing.

Size of the Space

The amount of space a private practice requires depends on what is available to some extent, and to some extent it has to be determined by the owner. It is certainly possible to open a small office and then relocate as the practice grows, but this transition can introduce problems, both with the actual relocation and with the risk of losing patients (possibly up to 20%).

Consider the following variables when deciding on space needs. One needs a room to house the sound-treated booth, a lab and storage area, and a waiting area and secretarial space. Closet space is needed for supplies, a server closet, and possible files. Bathrooms might also be necessary. A typical space might require approximately 1,200 square feet. However, often spaces are offered with a minimum greater than planned.

The search for the right space is similar to searching for the right residence: One might find a space in a desirable location but it exceeds one's budget; or the site is priced attractively but has no elevator. Windows are appealing for many businesses but for an audiologist, windows increase noise problems. Some audiologists may desire a small kitchen space. It is recommended to think long term: Would it be possible that eventually the practice might need more space, another booth room, a rentable office?

The audiologist might consider hiring an architect to draw out sample office layouts for consideration. The audiologist will have specs for booth size and the space needed for wheelchair access, as well as the estimated space needed for the waiting room, front desk, lab, and so on. The architect should have expertise in designing restroom facilities with wheelchair access; be aware of local codes and be mindful of the need for sinks where needed. Any sketched architectural layout is just an idea, but will help guide in computing the space needs of the office.

Lease and Build Out

The new practice owner must also consider the time frame for the lease and the cost of build out (installing walls, doors, cabinetry, plumbing fixtures, lighting, etc.). Again, an attorney is needed to ensure all contracts are fair and in the signee's favor as much as possible. Leases have options, for instance, placing the leasee responsible for paying for the cost of the heating and ventilation system including replacement if needed, or assuming responsibility for the annual service contract but not for replacement costs.

The length of the lease may also be subject to some negotiation. Let's imagine that the audiologist desires a 5-year lease with an option to renew, but the landlord wants a 10-year lease. The audiologist would rightly be concerned that if circumstances did not merit staying open for 10 years, he or she would be personally responsible for the lease payments for the full lease. An attorney may or may not be able to eliminate the personal liability clause, but might be able to negotiate a compromise so that the audiologist is fully responsible for the first 5 years, but after that, if deciding not to remain open, he or she would only be responsible for paying the remaining build out costs that the landlord incurred divided over each year of the remaining 5 years. This is just one example of how attorneys can be a financial benefit even when considering their fees; initially the landlord may not have agreed to that compromise but with skillful negotiations, the middle ground has a chance to be placed into the contract.

The build out on the part of the landlord may include the outside structure, the cost of the carpeting and flooring, the ceiling tiles and a standard number of sprinklers as well as the bathrooms, the office walls, doors, and cabinetry. These details must be confirmed in writing. The architect will then draw up a floor plan, and then submit a quote from a contractor for the costs of the build out. There is always a choice unless the lease prohibits it; one can select one's own contractor or use the landlord's contractor (advantage: One may not have to post a bond for the work done by the landlord's contractor; disadvantage: One has less control over the situation).

Many small details can be missed, but sharing one's plans with a colleague who has gone through this process can help. Having a second set of experienced eyes can result in identifying smaller issues upfront and avoiding an onerous expense later. Something as simple as not seeing that the correct kind of washable paint is being used on the walls can result in increased expense and the need to repaint the office sooner rather than later.

Rent Charges

Calculating an affordable rent is straightforward. A base rent (priced per square foot) for a year is defined in the

contract, as well as annual increases. Rent may or may not include utilities. Some contracts may stipulate that when taxes increase above the base tax at the time of initial contract, that increase is added to the rent. A cap on fees may or may not be negotiable. In some cases, the landlord will not provide build out at all, whereas others may provide an allotment (tenant improvement funds), and any upgrades over the allotted amount are the tenant's responsibility. Parking space agreements should be included in the contract, as well as maintenance. The landlord is typically responsible for maintaining the outside of the building, the common spaces, and the parking area, and those responsibilities should be clearly defined as part of the rental agreement.

EQUIPMENT NEEDS AND SPACE CONSIDERATION

Equipment needs are a critical consideration and must be calculated before designing space or even selecting a space; a practitioner cannot decide on how much space is needed until he or she knows how the space will be occupied. An audiologist setting up a practice will need to decide on videonystagmography (VNG) equipment, double-walled sound booth, using one or two booth rooms, and so on. These decisions are often staggered as short-term and long-term needs. What equipment is necessary at start up, and what can be expanded in the future? One's niche market considerations drive these decisions. For instance, if planning to test the younger population, a larger, double-walled booth is worthy of consideration because space is needed for speakers for sound field testing, as well as accommodating children with a parent or another adult. In addition, since frequently live voice and sound field are used, the more insulated booth is beneficial. If planning to conduct VNG assessments, one will need a room dedicated to those procedures.

Testing Booths

Booths come in different formations: Single-walled booths, single wall with a control booth, single walled with extra quieting panels, double-walled and double-walled with a control booth. The disadvantage of single-walled booths is the compromise to a quiet testing environment; for instance, children tested in sound field can hear the tester through the wall if testing is done via live voice. However, the cost of a double-walled unit is almost double. Control rooms can be added onto the booth to help maintain an acceptable noise level (American National Standard Institute, 1999). Remember that the patient referral system is dependent on satisfied patients who spread the word that your practice is worthy. Consider patient's response to the selection of the booth and what impact that might have on a budding practice.

Audiometers and Impedance Audiometry

Audiometers are available from one and a half channel to two full channel devices. They range in price from a few thousand dollars to almost $20,000 when fully equipped with sound field, high-frequency testing, and visual reinforcement audiometry. The audiologist will want to consider headphones: Insert headphones, standard circumaural cushions, high-frequency headphones.

For impedance testing, consider whether you will need to include options for Eustachian Tube dysfunction testing or B/G tympanograms. If testing newborns, a 1,000-Hz probe tone would be needed for tympanometry. An option for reflex testing would include manual or automatic control.

Additional Testing Equipment

The audiologist will have considered the cost and reimbursement rates for advanced diagnostic equipment, as well as equipment associated with hearing aid fittings (analyzers, probe mic systems, etc.). Remember that a niche practice will need to invest in equipment specifically for satisfying that population. Thus whereas a practice specializing in tinnitus and hyperacusis will have to invest in high-frequency headphones and in a toolbox full of tinnitus managers, a practice specializing in vestibular testing must include the cost of VNG equipment in the budget and in the space allocation.

Portable Equipment

A portable audiometer would be necessary if testing in nursing homes or assistive living facilities perhaps as well as when testing babies; then a portable audiometer can be brought into the sound booth to work directly with the young child. Some new options include hearing aid analyzers with portable testing equipment; some are networkable and compatible with electronic medical records. If conducting ototoxic monitoring, you will need high-frequency testing capabilities.

Financing Equipment

Equipment can be purchased or obtained with a leasing agreement. There are advantages and disadvantages to both options, so the decision must be carefully considered. In addition, used equipment is frequently available for purchase, and some manufacturers offer leasing or purchase over time. Equipment needs include not only audiometric instrumentation, but also computers, fax machines, telephones, even refrigerators, and coffee pots. When not outright purchasing, frequently the costs increase as payment is made over time; however, for a new practice, it may be

preferable to pay monthly costs rather than borrowing larger sums of money.

CASE STUDY: CALCULATING THE COST OF BUSINESS

Although at this point we have not considered all the variables of starting up a practice, it is never too early to start thinking like a business person and consider the "cost of business." Following is an example of a basic calculation:

Dr. Mayer, an audiologist, locates an attractive office property which is 1,000 square feet, paying $20 per foot rent and $2 per foot for utilities. Dr. Mayer plans on investing $25,000 in the build out costs, which includes sinking the booth into the floor and putting up cabinets in the lab and in the front desk area. She wants to outfit with an audiometer, a single-walled booth with extra sound paneling, insert phones, and VRA. She selects an impedance bridge with reflex testing and reflex decay as well as 1,000-Hz tympanograms. She plans to purchase an OAE machine and a probe mic speech mapping system, and also plans to obtain office furniture and computers.

Dr. Mayer determines that the overall budget including legal fees will be about $85,000. But she also needs money for inventory and supplies, so the more reasonable estimate is about $100,000 in start-up costs. Dr. Mayer has $20,000, and she will approach a bank for an $80,000 loan and a possible line of credit.

What is needed to make the monthly payments to the bank and to the landlord? Assume the loan will require $1,500 a month; rent will be $2,200; telecommunications (phone and internet) will cost $200. Utilities are $300 per month. Without paying herself a salary or hiring front desk personnel, Dr. Mayer's fixed expenses then are going to be about $4,200 a month. Also assume that she will be reimbursed for CPT codes 92557 (full audiogram) and 92550 (tympanograms and reflexes both ipsilateral and contralateral), which yields a payment of $50 per patient. Simple math indicates that she will need to perform 80 audios a month to cover fixed expenses. Proceeds remaining after the first 80 audiograms are applied to salaries and expenses.

If Dr. Mayer purchased less expensive equipment, she may only need $50,000 in start-up expenses and can manage to cover costs that much sooner, perhaps with only 10 audiograms per week. Dr. Mayer can increase her revenue-after-expenses with OAE and other kinds of testing and, of course, hearing aid fittings. Additionally Dr. Mayer might consider approaching her equipment vendor about a leasing deal; she might consider a lease with the option to buy so that if she found herself doing well sooner than anticipated, she could outright purchase the equipment.

Needless to say, this case study will continue to unfold as the practice develops and as Dr. Mayer evaluates the outcome of each decision. Every audiologist in independent practice must understand what is meant by the cost of business, how to calculate it, and how to manage the variables that affect it.

FIRST IMPRESSIONS: THE OFFICE AS A MARKETING TOOL

The general appearance of the practice is important and has been discussed at length elsewhere (e.g., see Carroll, 2012; Irene, 2011). Details to consider include

- Furniture and flooring: Comfortable, durable, easy to keep clean
- Lighting and decorations
- Reading materials (general interest magazines as well as materials on hearing and overall wellness)
- Brochures and demonstration areas (especially handy for various streamers, television remotes, captioned telephones, FM systems, alerting systems, and other assistive technology for patient education and hands-on practice).

Suffice it to say, a patient's initial impression of an audiologist's professionalism may depend more on "waiting room appeal" than the framed diploma and license. If not particularly talented in managing visual appearances, hire someone! Perhaps that person can also manage some business details.

PERSONNEL

The audiologist will need to answer several questions about personnel, such as who will run the office? Is it worthwhile to hire a front desk person? A billing person? What can one do independently and where is help needed? Some work can be "outsourced" such as billing, payroll, and making appointments. If sharing space with another professional, perhaps sharing personnel is also an option.

The prospect of hiring support staff can be daunting but help is available. Typical salary requirements for staff can be found through searches of online sites such as www.salary.com, which can provide regionally relevant information. Information such as salary range for years of experience is included in the graphed results. Be advised that salary is not the only consideration; other expenses associated with having an employee include worker's compensation, state disability, medicare and social security taxes on that income, the cost of sick/vacation days, and possibly health insurance. Conventional wisdom indicates an employer should plan on adding 10% to salary for these benefits. See Table 42.3 for a summary of these contributions.

Small businesses can obtain health insurance through brokers, national organizations, or local organizations. Some groups require that the insurees all be members of that professional group; others allow employees to be insured as long as the owner is a member of said group. Some require at least two to be insured for the small group rates to apply. The healthcare industry is in flux right now as the legislation

TABLE 42.3

The "Salary + 10%" Contributions to Personnel

- Workers compensation
- State disability
- Medicare
- Social Security
- Vacation/sick days
- Health insurance

regarding the new healthcare laws is changing as this is written. There may be state insurance pools or pools across state lines in the future. Additionally, quotations can be obtained online through groups like www.ehealthinsurance.com.

Once the practice is up and running, pensions can be considered. The US Department of Labor has a site defining all the pension options available for small businesses (http://www.dol.gov/dol/topic/retirement/). Some are tied to profits and thus in profitable years a set amount will be included but in unprofitable years no deposit may be made. Other options are based on contributions by the employee at a percentage the employee is comfortable making, whether matched or not by the employer. There can also be a stock ownership plan where the employee is given an amount of stock in the corporation. All of these need careful explanation by legal and accounting advisors as well as checking into state requirements and laws.

MARKETING

After the audiologist has found and built up the office space, obtained furniture and equipment, hired a front desk person, and contracted for health insurance, it is time to advertise. Initial steps are to create a brochure, business cards, and a webpage/Internet presence. The audiologist will want to put a personal stamp on the business, but may not have the personal skills to attract patients and referrals with marketing materials. One might want to ask for referrals from trusted colleagues for marketing experts. Different brochures may be needed for different audiences, for instance, a general brochure for internists, pediatricians, or patient groups and a specialized brochure (e.g., tinnitus care) for internists, psychiatrists, and TMJ specialists.

With written materials in hand, and web page functioning, the audiologist will dedicate considerable time delivering these brochures along with business cards to different audiences, including vision specialists, gerontologists, pediatricians, and psychologists (Duldudoa, 2012).

Within the community, marketing opportunities can include visits to senior citizen–assisted living centers, activity centers, nursery schools, and after-school care centers. If interested in providing services to schools, education administrators can be contacted as well. One can offer seminars in the local libraries, presenting on hearing loss prevention, assistive technologies (captioned phones, relay services, e.g., as well as hearing aids), communication strategies, and so on. Audiologists have a wealth of information to share with the public, and that information raises awareness for audiology in general, and one's practice in particular.

Advertising

The audiologist will want to consider advertising in local newspapers or on local radio or TV, in weekly disposable papers or local magazines, or via direct mail. Some of these options allow one to advertise his or her practice and also provide an interview for an article about the practice. An "advertorial" is especially valuable as it includes information to raise public awareness of your practice plus information the reader might find helpful and interesting. There is no guarantee of the most effective advertising method for your expertise, interest, and region; trial and error helps one find what works well for that particular practice.

Providing educational seminars or community events open to the public or by reservation is another way to promote one's practice. This can be done in the office, at a local library or school, or in a nicer setting such as a catering hall or restaurant. In many towns, if the purpose of the seminar is to educate, rather than to sell product, the local libraries allow the use of their community room at little or no cost. Regardless of how advertising is managed, it will be a large part of the budget until the practice becomes established.

PRICING

A key concern for the new business owner is how to set prices for one's services. In the current era of managed care, the audiologist needs to realize that he or she will most likely be paid the amount the managed care company (insurance) pays for that service. Most patients have some kind of insurance, and rates are typically set by insurers. Sometimes it is in the best interest of a practice to consider which insurances should be accepted and which declined (Brady, 2007). Medicare does not pay for hearing tests for the benefit of purchasing a hearing aid, so in a hearing-aid-only practice, the audiologist can either set a charge for the test or include it in a bundled price.

What is Bundling?

Before the era of managed care, each service or each product had a separate fee. For example, an audiologist might charge a patient $100 for a full evaluation and $1,500 for the hearing aid fit on the second visit. The hearing aid fee covered up to three office visits for hearing aid orientation, electroacoustic evaluations, batteries, and supplies (in other words, all services and products were bundled or combined

into one fee). The patient paid the bill in full and left with a statement to submit to the insurance company.

In the 1970s, health maintenance organizations began to come into prominence because of the federal Health Maintenance Organization Act of 1973 which required employers with 25 or more employees to offer options of HMO plans to employees (Hall et al., 2008). More and more, the traditional insurance plans disappeared as Preferred Provider Organizations developed. Because healthcare costs were rising, insurance companies began to look closely at fees charged and worked to control the reimbursement process to physicians who functioned as accepting providers (Kongstvedt, 2009). The change from a bundled charge to an unbundled charge came about because of what is and what is not covered by insurers. For instance, instead of charging $1,500 per aid, one might only charge $1,000 plus $250 for orientation, $100 for probe mic testing, $50 for batteries and supplies, and $100 for earmolds. If one decides to "unbundle," the audiologist will bill the insurance company for each office visit.

"To bundle or not to bundle" is a topic discussed at length on audiology forums and among online patient and patient-advocate groups. For patients who believe they will use fewer services than average, it would appear an unbundled fee schedule will save some money. Whether because of insurance benefit or because patients want to know what they are paying for and what benefit that payment will have, audiologists are now leaning toward the unbundling of their price structures. The largest benefit of unbundling has to do with valuing the services of the audiologist; when bundled, there is no value to the rehabilitation services provided (Coverstone and Miller, 2013).

THE OFFICE IS OPEN AND STILL MORE PAPERWORK!

When the office finally opens and the first patient comes in, the audiologist and/or staff need to ensure that referrals are appropriate, that insurance is valid, that a copy of official identification has been collected, and that HIPAA forms are signed. An up-to-date HIPAA agreement must be prominently displayed and the patient must sign a statement acknowledging that the agreement was seen (US Department of Health and Human Services, n.d.). In addition, with the latest version of HIPPA which was implemented in September 2013, a patient must actively agree to accept any marketing to be sent to that patient. Although one can send an unsolicited birthday card, or an educational newsletter, anything which has marketing intent must have patient permission prior to being sent.

Keeping patient data secure is another mandatory part of a practice. Each practice must have a security policy in writing, a security officer, and a compliance officer. A breach notification policy must be in place. In this age of electronic records, email and faxes, laptops and portable backup drives, it is vital that there be a record of how to ensure security. Even something as simple as a policy of how to wipe an old computer clean must be documented. Training on privacy issues is a requirement and must be documented.

Business Agreements

Currently under HIPAA, it is mandated that business agreements be kept with every vendor or party who will see protected personal data, including cleaning service, shredding vendors, accountants, hearing aid manufacturers, and equipment representatives. Details of this mandate can be found at http://www.hhs.gov/ocr/privacy, as well as a sample business agreement.

Material Data Sheets and Emergency Plans

Material data sheets accompany all cleaning and decontaminating products, as well as products used to modify hearing aids. They list the ingredients which are active and hazardous and how to handle the scene if the product is ingested or splashed into eyes, for example. Check-off sheets are signed to verify that employees have been trained to handle hearing aids and to clean up medical waste. Workers must be provided with training on an annual basis, and vaccines (e.g., for hepatitis) must be offered to all employees at no cost.

Emergency plans are written documentation of how to handle accidents, how to deal with bodily fluids and who has been trained to do so, as well as what to do in cases of physical emergencies. For example, what clean-up procedures are required when a patient has a bathroom accident or a vomiting incident? What plans go into affect if a patient becomes ill during a test procedure? These plans are legal requirements.

In many states, a practice must keep all material data sheets and emergency action plans in a metal file box in a set location, which must be easily found in an emergency. An evacuation plan has to be included in that box as well.

CONCLUSION

From the ADA (2008) document, "Ensuring Audiology's Future in Healthcare," it is clear that the students, faculty, and practitioners questioned agree that private practice ownership is where the vast majority see audiologists in the future. Besides the issue of autonomy, the income reported in this document as well as in ASHA's Audiology Survey 2010 Private Practice is greatest for private practitioners compared to other settings. Although the effort to open and run a private practice might be greater than that of being an employee, the benefit is real. In addition, owners of private practice are more likely to use validation and verification techniques, provide counseling, and demonstrate assistive technology (ASHA, 2010). In other words, the private

practitioner is generally providing much needed and high-quality services. Who can argue that this is an outstanding career choice?

HELPFUL WEBSITES

- Keirsey Temperament Sorter: keirsey.com/sorter/instruments2.aspx?partid=0
- How to create a business plan: sba.gov/business-plan/2
- An employer's guide to health insurance: ehealthinsurance.com
- An employer's guide to retirement plans: dol.gov/dol/topic/retirement/
- HIPAA regulations: http://www.hhs.gov/ocr/privacy/

FOOD FOR THOUGHT

1. What is the impact of consolidators (those who are bringing a variety of practices together under one umbrella) and hearing aid manufacturers buying practices?
2. Is there any ethical dilemma in having a hearing aid manufacturer provide funding to a practitioner who wants to purchase a practice?
3. What impact will the retail model or the future of Internet sales have on private practice?

REFERENCES

Academy of Doctors of Audiology. (2008) Practice Model Task Force: Ensuring audiology's future in healthcare: owning the profession through a culture of practice ownership. (Feedback (suppl), 19 (3).

American Academy of Audiology. (1991) The professional doctorate (AuD). Available online at: http://www.audiology.org/resources/documentlibrary/Pages/AuD.aspx.

American National Standards Institute. (1999) *Maximum Permissible Noise Levels for Audiometric Test Rooms (ANSI/ASA S3.1 [R2008])*. New York: Author.

American Speech-Language-Hearing Association. (n.d.) History of ASHA. Available online at: http://www.asha.org/about/history/.

American Speech-Language-Hearing Association. (2010) Audiology Survey 2010: private practice. Available online at: http://www.asha.org/uploadedFiles/10AudSurveyPrivatePractice.pdf.

Baggs T. (2012) Understanding personality: a key to supervisory success. *Perspect Admin Superv.* 22 (1), 4–11.

Brady G. (2007) The toughest competitor. *Adv Audio.* 9 (6), 80.

CareerCast.com. (2013) Jobs rated 2013: ranking 200 jobs from best to worst. Available online at: http://www.careercast.com/jobs-rated/best-worst-jobs-2013.

Carroll B. (2012) Pardon the wait: how to enhance the waiting room experience. *Audiol Today.* 24 (1), 18–19.

Coverstone J, Miller E. (2013) An analysis of fee-for-service models in audiology practices. Paper presented at AudiologyNOW! 2013, Anaheim, CA.

Dudulao M. (2012) Promoting your practice in challenging times. *Audiol Today.* 24 (3), 14–15.

Galambos R. (1998) *Hallowell Davis, 1896–1992: A Biographical Memoir.* Washington, DC: National Academies Press.

Gerber M. (1995) *The E-myth Revisited: Why Most Small Businesses Don't Work and What to Do About It.* New York: Harper Business.

Hall M, Bobinski M, Orentlicher D. (2008) *The Law of Health Care Finance and Regulation.* New York: Aspen Publishers.

Hosford-Dunn H, Dunn D, Harford E. (1995) *Audiology: Business and Practice Management.* San Diego, CA: Plural Publishing.

Irene T. (2011) Enhancing patient experience through first impressions. *Audiol Today.* 23 (5), 12–13.

Keirsey D. (1998) *Please Understand Me, II: Temperament, Character, Intelligence.* Del Mar, CA: Promethius Nemesis Books.

Kongstvedt P. (2009) *Managed Care: What It Is and How It Works.* 3rd ed. Sudbury, MA: Jones and Bartlett Publishers.

New York BSC. Law § 1508: NY Code – Section 1508: directors and officers. Available online at: http://codes.lp.findlaw.com/nycode/BSC/15/1508.

Smirga D. (2011) Are we (still) asleep at the wheel? An update from seven years ago. Available online at: http://www.audiologyonline.com/audiology-ceus/course/we-still-asleep-at-wheel-19303

US Department of Health and Human Services. (n.d.) The Health Insurance Portability and Accountability Act (HIPAA) privacy and security rules. Available online at: http://www.hhs.gov/ocr/privacy/.

CHAPTER 43

Implantable Hearing Devices

Teresa A. Zwolan

INTRODUCTION

Treatment options for hearing loss now include several implantable devices, including bone-anchored hearing systems, implantable middle-ear devices, cochlear implants (CIs), and auditory brainstem implants (ABIs). This chapter will provide information regarding how such devices work, descriptions of the surgical procedures used to implant these devices, and information regarding contemporary devices, including their Food and Drug Administration (FDA)-approved claims and candidacy statements.

BONE-ANCHORED HEARING DEVICES

Background

Bone-anchored hearing devices successfully transmit sound information to patients with chronic ear disease, external ear canal problems, external ear malformations, and, more recently, patients with single-sided deafness (SSD). They were first introduced by Anders Tjelsstrom in 1977 (Mudry and Tjellstrom, 2011). This early device consisted of a 4-mm titanium screw that was inserted into the bone behind the ear and attached to a bone-conduction hearing device. The screw, also referred to as an abutment, osseointegrates with the skull, meaning that bone grows and fuses with the screw, integrating it into the skull without the involvement of intervening fibrous tissue. A sound processor is then attached to the abutment. The microphone of the sound processor picks up sound from the environment and transmits it to the abutment. Vibration of the abutment results in transmission of sound to the inner ear via direct bone conduction, bypassing both the outer and middle ear. Direct attachment of the abutment to the skull provides several advantages over a traditional bone-conduction hearing aid, including greater comfort, less pain, and improved transmission of sound to the inner ear.

Contemporary Bone-Anchored Hearing Devices

Presently, there are three bone-anchored hearing devices available in the United States (Figure 43.1): the Baha (produced by Cochlear Americas), the Ponto (produced by Oticon), and the Alpha 1 (produced by Sophono). The Ponto utilizes an implantable abutment while the Sophono is an abutment-free aid that is placed in the bone behind the ear. The Baha has both an implantable (Baha 4 Connect) and an abutment-free (Baha 4 Attract) device that stays coupled via magnetic attraction. The Baha 4 sound processor is compatible with either Baha device. The Ponto has three sound processors available: the Ponto, the Ponto Pro, and the Ponto Pro Power. All three processors may be used with either the Ponto implant system or with specific compatible Baha abutments/implants from Cochlear Bone Anchored Solutions (BAS). Similarly, some of the Baha sound processors can be used with the Ponto implant system. The Alpha 1 and Alpha 2 speech processors are used with the Sophono Alpha Magnetic Implant.

Hard and soft headbands are available for Baha, Ponto, and Sophono processors, making it possible for young children, and occasionally adults, to use the bone-conduction hearing systems without having surgery.

Surgery

The surgically implanted abutment of the Baha and Ponto may be implanted in one or two stages. When done in two

FIGURE 43.1 Photos of the Baha, Ponto, and Sophono bone-conduction hearing aids.

TABLE 43.1

A List of Manufacturer Websites for Obtaining Additional Information Regarding Implantable Hearing Devices Mentioned in this Chapter

Category	Device	URL
Bone-anchored hearing devices	Cochlear Americas Baha bone-anchored hearing system	http://www.cochlear.com/wps/wcm/connect/us/home/treatment-options-for-hearing-loss/bone-conduction-implants
	Oticon Medical Ponto bone-anchored hearing system	http://www.oticonmedical.com/Medical/Our-Products/The%20Ponto%20System/what-is-ponto.aspx?gclid=CISelfvA_L0CFa9c-Mgodyw8ALQ
	Sophono Alpha 1 bone-anchored hearing system	www.sophono.com/products/alpha-1
Implantable middle-ear devices	Esteem Envoy hearing implant system	http://envoymedical.com
	Vibrant Soundbridge hearing implant system	http://www.medel.com/us/vibrant-soundbridge-middle-ear-implant/
Nonsurgical implantable hearing devices	Phonak Lyric2 hearing aid	http://www.phonak.com/com/b2c/en/products/hearing_instruments/lyric/overview.html
Cochlear implants	Advanced Bionics cochlear implant system	www.advancedbionics.com
	Cochlear Americas cochlear implant system	www.cochlear.com
	MED-EL cochlear implant system	www.medel.com/us/

stages, the first procedure involves placement of a fixture into the bone located behind the pinna. The second procedure is performed about 3 to 6 months later, allowing time for osseointegration of the fixture, and involves connecting the abutment to the fixture. With a single-stage procedure, which is primarily used with adults and older children, the fixture and the abutment are implanted at the same time. Surgery for the Sophono and the Baha Attract involves surgical placement of an abutment-free, completely implantable titanium device that is placed in the bone behind the ear. The implanted device houses two magnets that enable the sound processor to attach magnetically to the skull where it sits directly over the implanted device. The sound processor delivers acoustic information across the skin to the implanted device where sounds are then transferred directly to the inner ear via bone conduction. Complications for all three types of bone-conduction hearing systems are rare, but may include infection and inflammation at the implant site or failure of the abutment to osseointegrate with bone (Dun et al., 2011). Additional information about each type of device can be found on each manufacturer's website, as listed in Table 43.1.

Candidacy for Bone Anchored Hearing Devices

All three of these devices received approval in the United States by the FDA for use in patients (1) who have conductive or mixed hearing loss and can still benefit from sound amplification, (2) with bilaterally symmetric conductive or mixed hearing loss (and both may be implanted bilaterally), (3) with sensory/neural deafness in one ear and normal hearing in the other (i.e., SSD), and (4) who are candidates for an air-conduction contralateral routing of signals (AC CROS) hearing aid but who cannot or will not wear an AC CROS device (http://www.fda.gov/medicaldevices/productsandmedicalprocedures/homehealthandconsumer/consumerproducts/hearingaids/ucm181482.htmdevices). Additionally, all three devices are FDA-approved for use in adults and children aged 5 years and older.

CONTEMPORARY IMPLANTABLE MIDDLE-EAR DEVICES

Contemporary implantable hearing devices available in the United States are displayed in Figure 43.2. The Esteem® Hearing Implant is an active ossicular prosthesis manufactured by Envoy Medical and is FDA-approved for use in adults 18 years of age or older who have a moderate to severe sensory/neural hearing loss.

Surgery to implant the device typically involves an outpatient procedure that includes a postauricular incision to access the middle-ear space, disarticulation of the ossicular chain, connection of the sensor to the incus, connection of the driver to the stapes, and placement of the sound

Esteem

Vibrant Soundbridge

FIGURE 43.2 Photos of the Esteem and Vibrant Soundbridge implantable hearing aids.

processor behind the ear (underneath the scalp). The speech processor is connected to the sensor and driver via insulated wires. The sensor converts vibrations from the tympanic membrane and middle-ear ossicles into electrical signals that are sent to the sound processor. The sound processor amplifies and filters the signal based on the patient's hearing needs. The driver receives the signal from the sound processor and converts it back to vibrations that are then transmitted to the inner ear where they are perceived as sound. The Esteem differs from other devices in this chapter because all of its components are implanted, making it waterproof, as well as invisible to others.

The Vibrant Soundbridge is manufactured by MED-EL Hearing Technology GmbH and is FDA-approved for use in adults with moderate to severe sensory/neural, conductive, and mixed hearing losses. It consists of a surgically implanted transducer called the vibrating ossicular prosthesis (VORP) that includes a component known as the floating mass transducer (FMT), which is surgically attached to a vibratory structure of the middle ear. The VORP receiver is placed underneath the skin behind the ear and contains a magnet that helps attach the externally worn Amadé audio processor to the user's head. The microphone of the Amadé picks up sound from the environment and sends a signal across the skin to the implanted VORP for transmission to the inner ear via the FMT.

Nonsurgical Implantable Hearing Aids

A nonsurgical alternative to improved hearing is the Lyric2 produced by Phonak and consists of a hearing aid that is implanted deeply into the ear canal. It is suitable for people

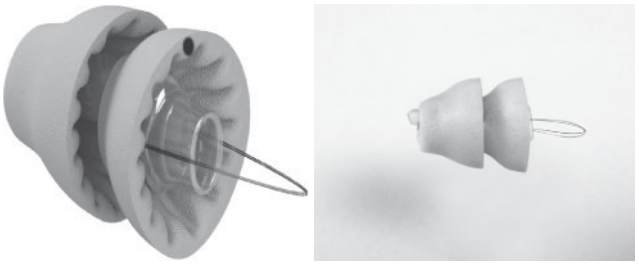


FIGURE 43.3 Photos of the Lyric2 (Phonak) nonsurgical implantable hearing aid.

with mild to moderately severe hearing losses (Figure 43.3). The aid is worn in the canal for a period of up to 3 months and can be worn while performing various activities such as showering, exercising, and sleeping and is removed/replaced every few months when the battery wears out. Additional information about this device can be found on the manufacturer's website: www.lyrichearing.com.

COCHLEAR IMPLANTS

Background

Cochlear Implants (CIs) were originally designed to replace the function of the profoundly impaired inner ear. Candidacy was initially limited to adults and children with bilateral profound sensory/neural hearing losses. Today, adults and children with moderate-to-profound hearing losses receive CIs. Various clinical trails are evaluating the expansion of candidacy to include the use of hybrid devices designed for patients with mild to moderate low-frequency hearing coupled with a severe to profound hearing loss in the high frequencies, to evaluate the use of CIs in patients with greater preoperative speech recognition, and to evaluate the use of CIs as a treatment for SSD. Such expansions in CI candidacy have been fostered by improvements in technology and revision of traditional surgical techniques to allow greater preservation of residual hearing.

There are three manufacturers who provide CI systems in the United States: Advanced Bionics (AB) of Sylmar, CA (Figure 43.4), Cochlear Americas of Centennial, CO (Figure 43.5), and MED-EL of Innsbruck, Austria (Figure 43.6). All of the systems developed by these manufacturers include a surgically implanted device and an externally worn speech processor.

FIGURE 43.4 Photo of the cochlear implant system presently offered by Advanced Bionics (AB).

FIGURE 43.5 Photo of the cochlear implant system presently offered by Cochlear Americas.

Internal Device Components

Contemporary internal devices consist of several common components, including a receiving coil/internal processor, magnet, electronics package, extracochlear electrodes, and an electrode array containing tiny contacts (electrodes) that deliver the electric signal to the inner ear.

The receiving coil is the largest portion of the internal device and is surgically placed in the mastoid bone. It is composed of a magnet (for attachment of the external headset) and an antenna that receives the transmitted signal. All devices use a special plastic casing to house the receiving coil/internal processor.

All of the currently available systems are multichannel and take advantage of the tonotopic organization of the cochlea providing differently processed information to electrodes positioned at different locations within the cochlea. When stimulated, these electrodes provide localized excitation of cochlear nerve fibers, resulting in place pitch information used to understand speech. The number of stimulating electrodes used in current systems varies from 12 to 22. Several different configurations of electrode arrays are offered by each manufacturer to customize the fit of the electrode into the cochlea. Such arrays vary in several ways, including total length (longer, shorter, and compressed arrays are available) and configuration (i.e., thinner arrays with smaller diameters for improved hearing preservation).

FIGURE 43.6 Photo of the cochlear implant system presently offered by MED-EL.

External Components

Currently available CI systems have several common external components that work together to collect, process, and transmit auditory information to the internal components. External components include a microphone, speech processor, connecting cables, and a transmitter coil (Figure 43.7). The microphone captures sound from the environment and sends it to the speech processor, where it is converted into digital information that is coded for transmission to the internal device. The signal is then sent via a wire to the transmitter located on the outside of the implant user's head. The transmitter is aligned with the internal receiving coil and is held in place by external and internal magnets. The integrated circuit within the CI internal processor receives the information, decodes the signal, and delivers electrical stimulation to the implanted electrodes. All of the currently available devices use transcutaneous transmission, which means the signal is delivered across intact skin using a radio frequency link.

Currently available devices offer several options that enable the clinician to provide optimum efficiency and fit of the external equipment for individual patients. Cords come in a variety of lengths, and magnets come in a variety of strengths to accommodate the needs of both adults and children. Additionally, speech processors, cords, and headset components are offered in a variety of colors to maximize cosmetic appearance.

Processor Features

Early CI systems included large body-worn processors that were powered by AA batteries. Present-day systems continue to include body-worn processors but also include ear-level speech processors. Ear-level processors are modular and include a battery pack that utilizes either rechargeable or disposable batteries and can be attached to the processor and worn behind the ear, or attached to a body-worn battery pack that can be attached to the recipient's clothing. All processors are able to store multiple programs and have special features that decrease their susceptibility to electrostatic discharge (ESD). The number of memories or programs that the processor is able to contain varies from three to nine. Having more than one memory is advantageous as it provides the user with the ability to experiment with

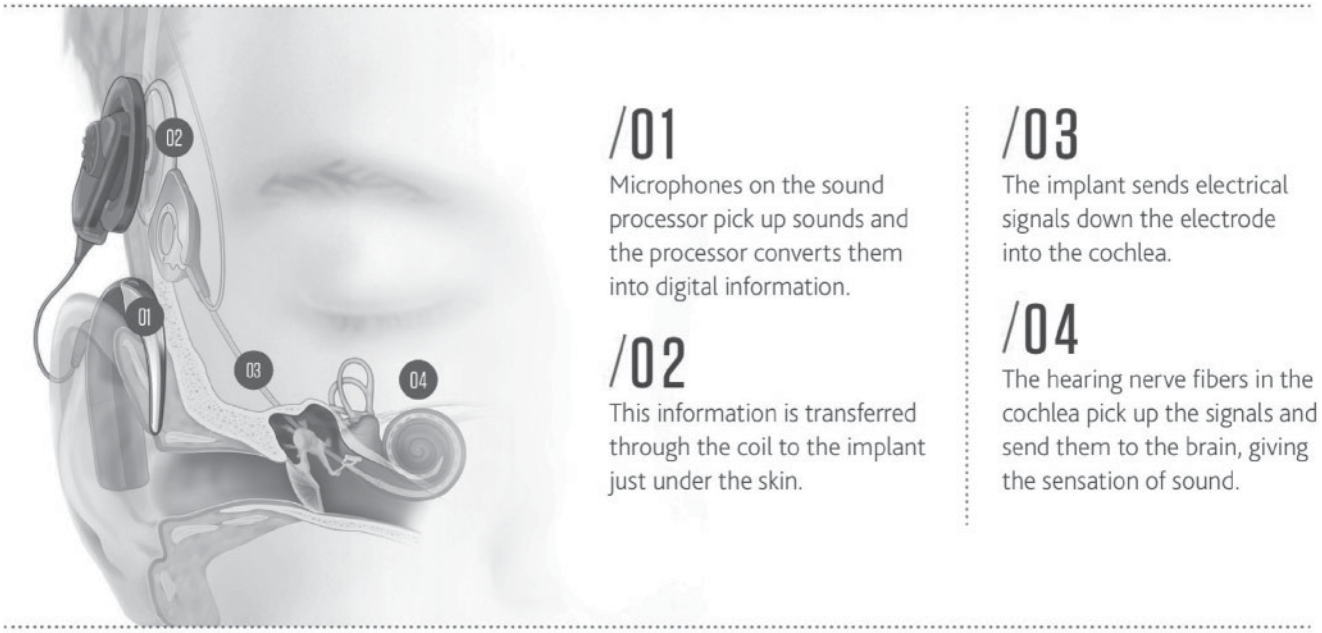


FIGURE 43.7 Schematic diagram of how a cochlear implant works. [Courtesy of Cochlear Americas.]

different programs and/or different processing strategies outside the clinic setting that helps decrease the number of follow-up appointments needed to adjust the patient's program.

Additional device features include processor dials or remote controls to adjust the settings of the processor, private and public alarms, "lock" features that prevent children from changing the settings on the speech processor or having access to the speech processor batteries, connection to various assistive listening devices and FM units using telecoil, streaming, or Bluetooth technology, and either standard alkaline or rechargeable battery compartments to power the device.

The way the speech processor delivers information to the inner ear is determined by the speech processing strategy. Several different strategies are available with each device and provide clinicians with the ability to customize and maximize patient outcomes. Table 43.2 includes a summary of the various speech processing strategies that are available with contemporary devices. The reader is referred to Wolfe and Schafer (2010) for a detailed description of contemporary speech processing strategies.

Specific features of currently available CI systems will be briefly described below. Additional information can be obtained from each manufacturer's website; see Table 43.1.

Advanced Bionics

The HiResolution Bionic Ear System is manufactured by Advanced Bionics (AB) Corporation of Sylmar, CA. The first commercial device (Clarion) was introduced in 1987 by MiniMed Technologies, which later became Advanced Bionics Corporation in Valencia, CA.

Components of the contemporary AB system are displayed in Figure 43.4. This system includes the HR 90K implant, the Naida CI speech processor, and the recently introduced Neptune—the industry's first waterproof processor. There are three speech processing strategies presently available with the AB device: HiResolution (HiRes-S, HiRes-P, HiRes-S with Fidelity 120, HiRes-P with Fidelity 120), Continuous Interleaved Sampling (CIS) (Wilson, 2000), and Multiple Pulsatile Sampler (MPS).

Cochlear Pty. Ltd.

Cochlear Pty. Ltd. ("proprietory limited") was formed in 1981 in Sydney, Australia. In 1985, the Nucleus CI22 received FDA approval for use in adults and received approval for use in children in 1990.

Components of the contemporary CI system produced by Cochlear Americas are displayed in Figure 43.5. This system includes the Nucleus Freedom (CI24RE) implant and the N6 (CP910) speech processor, a modular device that can be worn on either the body or behind the ear. The speech processing strategies currently available with the Cochlear system include Spectral Peak (SPEAK), Advanced Combined Encoder (ACE), and CIS.

MED-EL

The MED-EL device was first introduced in Europe in the 1970s and was first introduced in the United States in the

TABLE 43.2

Speech Processing Strategies Available with Contemporary Devices

Strategy	Descriptor	Advanced Bionics	Cochlear Americas	MED-EL
NofM	Unlike CIS strategies (described below), which stimulate all active electrodes during each stimulation cycle, NofM strategies evaluate the acoustic energy present in each *m* channel; stimulation is then only delivered to the *n* channels with the highest amplitude inputs. The *n* is typically referred to as maxima and remains constant for each stimulation cycle			X
SPEAK	SPECTRAL PEAK: An NofM-type strategy that typically uses up to 8 maxima. Electrodes are typically stimulated at a rate of 250 pp. Unlike NofM, the number of maxima may change during stimulation cycles depending on the energy of the incoming signal		X	
ACE	ADVANCED COMBINED ENCODER: Also considered an NofM strategy, but utilizes faster stimulation rates and is capable of using more maxima than SPEAK. Similar to SPEAK, the number of maxima during each stimulation cycle may change depending on the energy of the incoming signal		X	
CIS	CONTINUOUS INTERLEAVED SAMPLING: The acoustic signal is digitally filtered and sent through a bank of bandpass filters. Each electrode is stimulated sequentially during each stimulation cycle. Amplitude of stimulation depends on the energy present in each band (greater energy = greater amplitude)	X	X	X
CIS+	A version of CIS that utilizes expanded frequency bands			X
HDCIS	HIGH DEFINITION CIS: A version of CIS that, in addition to sequential stimulation, utilizes sequential stimulation between neighboring stimulation sites to elicit a percept that falls between the percept elicited by the two electrodes			X
FSP	FINE STRUCTURE PROCESSING: A CIS-type strategy that additionally modulates the timing of pulse bursts in the low-frequency channels to provide improved temporal cues and provides information regarding intermediate pitches by using overlapping bandpass filters			X
HIRES	HI RESOLUTION: A version of CIS that utilizes 16 channels, stimulates at higher maximum stimulation rates, and has higher cutoff frequencies for low-pass filters than traditional CIS	X		
HIRES 120	HI RESOLUTION 120: Incorporates current steering to attempt to provide an increase in the number of perceptual channels to 120	X		
HIRES S	HI RESOLUTION–SEQUENTIAL: A HIRES strategy that utilizes sequential stimulation of electrodes	X		
HIRES P	HI RESOLUTION PARTIAL SIMULTANEOUS: A HIRES strategy that provides partial simultaneous stimulation	X		
MPS	MULTIPLE PULSATILE SAMPLER: A partially simultaneous strategy that stimulates two or more nonadjacent electrodes at the same time	X		
SAS	SIMULTANEOUS ANALOG STIMULATION: Stimulates electrodes with continuous analog waveforms. Unlike CIS (which stimulates all electrodes sequentially), SAS stimulates each electrode simultaneously during each cycle of stimulation	X		

1990s. Components of the contemporary MED-EL system are displayed in Figure 43.6 and include the Concert implant, and the Opus 2 and Rondo speech processors. The Rondo is the first CI speech processor that can be worn on the head but off the ear; it was introduced in 2013. Speech processing strategies currently available with MED-EL devices include HDCIS and FSP.

HYBRID COCHLEAR IMPLANT DEVICES

Hybrid devices use both electric and acoustic stimulation in the same ear, by presenting high-frequency information via the surgically implanted electrode array, and low-frequency acoustic information via an ipsilateral hearing aid. These electric acoustic stimulation (EAS) devices are based on the premise that it is possible to preserve low-frequency hearing after CI surgery when one uses careful surgical techniques and certain electrode designs that minimize trauma to inner-ear structures. Previously, it was assumed that patients would experience a total and complete loss of residual hearing following insertion of the electrode array. However, Gstoettner et al. (2006a, 2006b) reported long-term hearing preservation rates of 70% and 83%, respectively, with EAS devices, indicating that hearing preservation is possible following CI surgery.

The Nucleus Hybrid Hearing Implant was FDA approved in 2014 and is suitable for patients with low frequency thresholds no poorer than 60 dB HL up to and including 500 Hz and a PTA (2,3,4 KHz) that is greater than or equal to 75 dB HL in the ear to be implanted. Additionally, the ear to be implanted should demonstrate a CNC word recognition score between 10 and 60% correct while the CNC score for the non-implanted ear should be equal to or less than 80% correct and have a PTA (2,3,4 KHZ) greater than or equal to 60 dB HL. Recipients of this device utilize a hybrid sound processor that delivers both acoustic information (via a hearing aid portion connected to the processor) and electric information (via the sound processor). The Nucleus 6 processor, which was recently approved by the FDA, includes a commercially available acoustic component integrated into the processor and is available for use with hybrid patients.

MED-EL's FLEX^{eas} electrode is a 20.9-mm electrode array with a flexible tip designed to reduce the force placed on the cochlea during electrode insertion, increasing the chances for preservation of residual hearing (Hochmair et al., 2006). In 2005, MED-EL introduced the DUET EAS hearing system, which combines the features of the MED-EL TEMPO+ speech processor with digital acoustic amplification circuitry. The MED-EL EAS system is currently undergoing clinical trials in the United States and is not yet FDA approved for widespread use.

DETERMINING CANDIDACY FOR A COCHLEAR IMPLANT

Candidacy requirements for a CI have changed greatly since CIs were first introduced. In the United States, the FDA oversees the selling, distribution, labeling, and marketing of medical devices and determines if the specific wording used in device labeling, including information regarding indications for its use, or candidacy, is appropriate following completion of a clinical trial. They also include wording related to both audiometric and speech recognition criteria. FDA-approved indications differ for adults and children and are often dependent on when the device received FDA approval. Candidacy criteria for contemporary devices are provided in Table 43.3.

When CIs were first introduced, only patients who demonstrated no benefit from amplification (i.e., scored 0% correct on open-set tests of sentence recognition) were considered candidates for a CI. Today, patients may be considered a candidate to receive an FDA-approved CI if they demonstrate various levels of open-set speech recognition. Because of broader candidacy criteria, it is important for clinicians to recommend a CI only if it is believed that the patient will demonstrate improved communication skills with implant use.

Preoperative Testing to Determine Candidacy for a Cochlear Implant

The primary purpose of the preoperative evaluation is to determine if the patient is medically and audiologically suitable for a CI. Additionally, such information should be compared to postoperative results to evaluate the recipient's progress and device efficacy. The determination of candidacy for a CI is most often made by a group of qualified professionals, including a surgeon, an audiologist, and a speech-language pathologist. The opinions of other professionals, such as that of a psychologist or educator, may also be taken into account.

Test procedures commonly included in the preoperative process for determining implant candidacy are described below.

Medical Evaluation

During the preoperative medical evaluation, the physician obtains a complete medical history, performs a physical examination, and ensures that the candidate is up to date on all immunizations (see http://www.cdc.gov/vaccines/vpd-vac/mening/cochlear/dis-cochlear-gen.htm for information regarding recent Centers for Disease Control and Prevention recommendations). The surgeon attempts to identify the cause of the hearing loss if it is not already known and determines if treatment options other than a CI are more

TABLE 43.3

FDA-approved Candidacy Criteria for Contemporary Cochlear Implant Systems

Adults (18 years of age or older)

Advanced Bionics HiRes 90K

18 years of age or older with severe to profound, bilateral sensory/neural hearing loss (>70 dB HL) with postlingual onset of severe or profound hearing loss. Limited benefit from appropriately fit hearing aids, defined as scoring 50% or less on a test of open-set sentence recognition (HINT sentences)

Cochlear Americas Nucleus Freedom (CI24RE)

Bilateral pre-, peri-, or postlinguistic, sensory/neural hearing impairment and obtain limited benefit from appropriate binaural hearing aids. These individuals typically have moderate-to-profound hearing loss in the low frequencies and profound (≥90 dB HL) hearing loss in the mid to high speech frequencies. Limited benefit from amplification is defined by test scores of 50% correct or less in the ear to be implanted (60% or less in the best-aided listening condition) on tape-recorded tests of open-set sentence recognition

MED-EL Concert

Adults of 18 years of age or older who have bilateral, sensory/neural hearing impairment and obtain limited benefit from appropriately fitted binaural hearing aids. These individuals typically demonstrate bilateral severe to profound sensory/neural hearing loss determined by a puretone average of 70 dB or greater at 500, 1,000, and 2,000 Hz. Limited benefit from amplification is defined by test scores of 40% correct or less in best-aided listening condition on DC-recorded tests of open-set sentence recognition (Hearing in Noise Test [HINT] sentences)

Children

Advanced Bionics HiRes 90K

Age from 12 months through 17 years with profound, bilateral sensory/neural deafness (>90 dB HL). Use of appropriately fitted hearing aids for at least 6 months in children 2–17 years of age, or at least 3 months in children 12–23 months of age. The minimum duration of hearing aid use is waived if X-rays indicate ossification of the cochlea. Little or no benefit from appropriately fit hearing aids. In younger children (<4 years of age), lack of benefit is defined as a failure to reach developmentally appropriate auditory milestones (such as spontaneous response to name in quiet or to environmental sounds) measured using the Infant-Toddler Meaningful Auditory Integration Scale or Meaningful Auditory Integration Scale or <20% correct on a simple open-set word recognition test (Multisyllabic Lexical Neighborhood Test [MLNT]) administered using monitored live voice (70 dB SPL). In older children (>4 years of age), lack of hearing aid benefit is defined as scoring <12% on a difficult open-set word recognition test (Phonetically Balanced Kindergarten Test) or <30% on an open-set sentence test (HINT for Children) administered using recorded materials in the sound field (70 dB SPL)

Cochlear Americas Nucleus Freedom (CI24RE)

Intended for use in children 12–24 months of age who have bilateral profound sensory/neural deafness and demonstrate limited benefit from appropriate binaural hearing aids. Children 2 years of age and older may demonstrate severe to profound hearing loss bilaterally. In younger children, limited benefit is defined as lack of progress in the development of simple auditory skills in conjunction with appropriate amplification and participation in intensive aural habilitation over a 3–6 month period. It is recommended that limited benefit be quantified on a measure such as the Meaningful Auditory Integration Scale or the Early Speech Perception test. In older children, limited aided benefit is defined as ≤30% correct on the open-set MLNT or Lexical Neighborhood Test (LNT), depending on the child's cognitive and linguistic skills. A 3–6 month hearing aid trial is recommended for children without previous aided experience

MED-EL Concert

Children aged 12 months to 17 years 11 months must demonstrate a profound, bilateral sensory/neural hearing loss with thresholds of 90 dB or greater at 1,000 Hz. In younger children, little or no benefit is defined by lack of progress in the development of simple auditory skills in conjunction with appropriate amplification and participation Intensive aural habilitation over a 3–6 month period. In older children, lack of aid benefit is defined as <20% correct on the MLNT or LNT, depending on the child's cognitive ability and linguistic skills. A 3–6 month hearing aid trial is required for children without previous experience with hearing aids. Radiologic evidence of cochlear ossification may justify a shorter trial with amplification

suitable, such as performing a stapedectomy in cases of far-advanced otosclerosis. The candidate's general health is also evaluated to determine if he or she is healthy enough to participate in the surgery required for cochlear implantation.

Cochlear Imaging

Computed tomography (CT) or magnetic resonance imaging (MRI) of the temporal bone is routinely performed as part of the preoperative evaluation process to visualize development of the mastoid and inner-ear structures. These procedures identify any inner-ear anomalies, determine if cochlear ossification is present, and help determine the most suitable ear for implant if a unilateral procedure is recommended. Knowledge regarding the presence of cochlear anomalies is important prior to surgery because it may affect the type of electrode array selected for surgery (i.e., compressed, straight, or split array), the surgical approach used to access the cochlea, the insertion depth of the electrode array, selection of the ear for implantation, and final placement of the electrode array. Cochlear malformations rarely preclude placement of a CI (Balkany et al., 1996; Tucci et al., 1995), although severe anomalies may limit the insertion depth of the electrode array to a degree that would compromise the anticipated benefit from the device. Furthermore, associated anomalies of the temporal bone, particularly absence of the eighth nerve, may render cochlear implantation unwise. To avoid unnecessary risks to the patient, disappointment for the family, and delay in the initiation of alternative communication modes, the preoperative assessment of patients with anomalous temporal bones should include MRI and electrically evoked auditory brainstem response (EABR) testing (Kileny and Zwolan, 2004), which will allow the clinician to assess both the structural and functional integrity of the auditory neural pathways.

Audiologic Evaluation

The primary purpose of the preoperative audiologic evaluation is to determine the type and severity of hearing loss. This evaluation typically includes unaided testing of air- and bone-conduction thresholds, speech recognition threshold (SRT), speech detection threshold (SDT), word recognition, otoacoustic emissions (OAEs), and tympanometry and acoustic reflexes. As noted in Table 43.4, FDA guidelines differ across manufacturers with regard to recommended audiometric criteria to receive a CI.

Electrophysiological Testing

Auditory brainstem response (ABR) testing is a vital part of the CI candidacy process as it is used to verify behavioral audiometric test results, helps to identify patients with auditory dyssynchrony (ANSD), and helps to rule out the possibility of functional deafness. Additionally, some centers perform EABR (Kileny and Zwolan, 2004; Kileny et al., 1994) testing prior to implantation to verify electric stimulability of an ear. Such testing is usually performed in the operating room immediately prior to CI surgery and involves the presentation of current pulses via a transtympanically placed promontory needle electrode (Stypulkowski et al., 1986). The EABR is particularly useful when questions arise regarding the presence or stimulability of the eighth nerve in the ear to be implanted (e.g., when a patient presents with a history of cochlear anomaly coupled with no responses to audiometric testing).

TABLE 43.4

Tests Commonly Used to Evaluate Speech Perception Skills of Pediatric Cochlear Implant Users

- Early Speech Perception Test (ESP) (Moog and Geers, 1990)
- Meaningful Auditory Integration Scale (MAIS) (Robbins et al., 1991)
- Infant-Toddler Meaningful Auditory Integration Scale (IT-MAIS) (Zimmerman-Phillips et al., 1998)
- Word Intelligibility by Picture Identification (WIPI) Test (Ross and Lerman, 1979)
- Northwestern University-Children's Perception of Speech (NU-CHIPS) Test (Elliott and Katz, 1980)
- Phonetically Balanced Kindergarten (PBK)-50 Word List (Haskins, 1949)
- Bamford-Kowal-Bench (BKB) Sentences (Bench et al., 1979)
- Glendonald Auditory Screening Procedure (GASP) (Erber, 1982)
- Lexical Neighborhood Test (LNT) (Kirk et al., 1995)
- Multisyllabic Lexical Neighborhood Test (MLNT) (Kirk et al., 1995)

Hearing Aid Evaluation and Speech Perception Testing

The preoperative hearing aid evaluation (HAE) is necessary to verify that the candidate's device is appropriate for his/her level of hearing loss. Although recommended hearing aid settings vary among clinicians, the amount of gain a candidate receives from amplification should be verified using a probe microphone approach that is referenced to ear canal SPL (Valente et al., 2006). Once an appropriate hearing aid has been selected for the evaluation, testing to evaluate speech recognition skills when using the appropriately fit hearing aids may begin.

When evaluating candidacy for a CI, it is important to consider the amount of time the candidate has used appropriate amplification. This is particularly true with children, prelingually deafened adults, and adults and children who

demonstrate some open-set speech recognition as experience with amplification may influence auditory detection and speech recognition skills. In most cases, a 1- to 3-month trial with appropriate amplification is recommended to ascertain the potential for aided benefit. With children, feedback provided by a speech-language pathologist who works with the patient during the trial period can be valuable. A shorter trial with amplification is justified if there is radiologic evidence of cochlear ossification or if the patient clearly receives no benefit from amplification. A longer trial or extended speech and language therapy may be needed if the clinician observes good improvement in auditory skills during the trial period.

Speech perception testing should be performed in a sound field using recorded test materials whenever possible (this is often not feasible, however, when testing small children). Most clinics and most clinical trials presently use a presentation level of 60-dB sound pressure level (SPL) for test stimuli (Firszt et al., 2004).

When administering speech perception tests, patients should be seated in a sound-treated room that contains minimal visual and auditory distractions. The presentation level should be calculated using a calibration microphone placed at the center of the listener's head. Test materials should be presented a single time only, and feedback should not be provided.

There are several different speech perception materials available for use with children and adults. When testing adults, most clinics follow recommendations provided in the manual of the Minimum Speech Test Battery (MSTB) (http://www.auditorypotential.com/MSTBfiles/MSTB-Manual2011-06-20%20.pdf), which was revised in 2011 and includes a recommendation to administer the following tests preoperatively and 3, 6, and 12 months postoperatively: One 20-sentence list of AzBio sentences (Peterson and Lehiste, 1962) presented in quiet, one 20-sentence list of AzBio sentences presented in noise, one 50-word list of CNC words, one 16-sentence list-pair (8 sentences per list) of the BKB-SIN test (Bench, 1979) preoperatively, and one 20-sentence list-pair (10 sentences per list) of the BKB-SIN postoperatively. A portion of the testing should be performed with each ear aided separately as well as in a binaurally aided condition to compare pre- versus postoperative performance. Testing ears individually also helps with other decisions, such as the selection of which ear to implant in cases of unilateral CI and determination of binaural advantage received from using two devices.

It is important to keep in mind that speech recognition scores provided in Table 43.3 are guidelines approved by the FDA for particular products, and that specific wording of each product is influenced by several factors, including the timeframe of FDA approval and design of the clinical trial. Thus, some patients have received devices "off label," meaning that they demonstrated preoperative speech recognition scores that are better than current FDA-approved recommendations for that device. Factors that may contribute to a decision to recommend an implant to someone outside FDA recommendations include the effect that reduced speech recognition has on the patient's ability to function in educational, occupational, and social settings. In some instances, insurers will only preauthorize payment for the device if the patient meets FDA indications, limiting any flexibility the clinic may have regarding candidacy.

It is also important to note that Medicare, the national health insurance program that provides health insurance to US citizens aged 65 and above, has specific criteria that clinics need to follow if a patient is enrolled in Medicare. Presently, these criteria differ from those of the FDA. On its website (www.cms.hhs.gov), the Centers for Medicare and Medicaid Services (CMS), which administers the Medicare program, presently indicates that implantation is for treating bilateral pre- or postlinguistic, sensory/neural, moderate-to-profound hearing loss in those who get limited benefit from amplification (i.e., ≤40% correct for the best-aided listening condition using recorded tests of open-set sentence recognition). CMS also states that individuals with hearing test scores of 40% to 60% can receive a CI if both the provider and patient are enrolled in an FDA-approved category B IDE (Investigational Device Exemption) clinical trial, a trial under the CMS Clinical Trial Policy, or an upcoming, controlled comparative trial that is approved by CMS as meeting the requirements for National Coverage Analyses and also meets their specific quality standards. Additional information regarding this matter can be obtained from the CMS website (http://www.cms.hhs.gov/).

Selection criteria for children have also been expanded to include candidates with minimal open-set speech perception skills. Like adults, the specific wording used in FDA-approved guidelines differs for currently approved devices. FDA-approved indications for use of all devices state that, in younger children, limited benefit is defined as lack of progress in the development of simple auditory skills in conjunction with appropriate amplification and participation in intensive aural rehabilitation over a 3- to 6-month period, and all recommend that hearing aid benefit be quantified in young children using measures such as the Meaningful Auditory Integration Scale (MAIS) (Robbins et al., 1991) or the Early Speech Perception (ESP) test (Moog and Geers, 1990). With older children, all three manufacturers recommend use of the open-set Multisyllabic Lexical Neighborhood Test (MLNT) or Lexical Neighborhood Test (LNT) (Kirk et al., 1995), depending on the child's cognitive and linguistic skills. Like adults, the particular score used to define lack of benefit varies. For the Nucleus Freedom, lack of aided benefit is presently defined as a score ≤30% correct, whereas the Advanced Bionics HiResolution and the MED-EL Pulsar systems define this as a score ≤20% correct on such measures. With both children and adults, preoperative

speech perception testing should include tests that measure a variety of speech perception skills.

Pediatric speech perception tests can be broken down into three general areas: (1) Closed-set tests that measure prosodic cue, speech feature, or word perception; (2) open-set word and sentence tests that provide an estimate of the child's ability to communicate in the "real world"; and (3) auditory development scales, such as the MAIS (Robbins et al., 1991), that use parental report to evaluate the child's listening skills in his or her daily environment. Speech perception tests commonly used with children in pre- and postoperative CI evaluations are listed in Table 43.4. For a comprehensive review of these test procedures, the reader is referred to Gifford (2013).

Speech and Language Evaluation

Speech-language evaluations are performed at most centers as part of the pediatric pre- and postoperative evaluations. This evaluation is essential because it provides information regarding the child's development of speech and language skills—a critical factor when considering pediatric candidacy for a CI. During this evaluation, the speech-language pathologist may evaluate the child's developmental expressive and receptive language skills, articulation skills, and speech intelligibility. Importantly, the child's communicative status is evaluated with respect to normative models of language development, and this provides information regarding expectations for speech and language improvements that may or may not be seen following intervention with a CI. Occasionally, the speech-language pathologist may recommend participation in structured therapy before making a recommendation regarding the patient's candidacy for a CI. Such therapy may be particularly helpful in borderline cases, in cases where the child has received little therapeutic intervention or has been inappropriately fit with amplification, and also in cases of auditory neuropathy spectrum disorder (ANSD).

Similar to speech perception materials, the specific tests used in the speech and language evaluation are dependent on the child's age and language level. Postoperatively, children with CIs should continue to participate in regularly scheduled speech and language therapy and evaluations. These evaluations help determine which speech cues are or are not being perceived by the child, help identify lack of progress that may be because of internal device failure, provide information that may aid in programming the child's device, and help determine auditory training goals that can be focused on during speech and language therapy sessions.

Psychologic Evaluation

The psychologic evaluation is primarily performed with pediatric patients but may also be necessary with adults who present with concerns regarding cognitive status or mental function. With children, a preoperative evaluation should include nonverbal assessment of cognitive, social, emotional, and adaptive abilities to determine if factors other than hearing impairment are hindering the child's auditory development. Depending on the patient's age and the presenting concerns, the ability of the child to attend to and remember information may also be assessed, and recommendations may be made regarding educational services. The presence of a cognitive impairment may impact the child's ability to develop spoken language skills and will influence the counseling provided to parents regarding expected outcomes. Parents of very young children should be informed that some psychologic deficits (e.g., autism) are not typically identified until the child is 2 years of age or older and that performance with the device may be hindered if additional disabilities are identified.

In recent years, greater numbers of children presenting with a disability in addition to their hearing loss have received CIs (Donaldson et al., 2004; Waltzman et al., 2000; Wiley et al., 2005). This change has increased the need for preoperative psychologic evaluations that will help determine how effectively the child will be able to utilize the auditory signal. The input of the psychologist is essential when determining if referrals to other professionals are necessary prior to and after the child receives a CI.

Patient Education

Patient education is an ongoing part of the pre- and postoperative evaluation process. When the patient is first seen, information should be provided regarding device options, CI technology, candidacy requirements, expectations of performance, appointments involved in the evaluation process, and financial obligations. Many centers make arrangements for patients to meet with a CI recipient or with the parents of a pediatric CI recipient to discuss the implant evaluation process.

Coordination of Services with the Child's Educational Setting

Ideally, parents should inform the child's school about the plans for the child to receive a CI as it is important for educators to understand how to use and troubleshoot the device. An in-person visit from the implant audiologist or speech–language pathologist is ideal as it allows the clinician to observe the child in his/her classroom setting and may foster recommendations being made regarding management of the child's auditory needs in the classroom.

Determination of Implant Ear

Over the past few years, bilateral CIs have become increasingly common for both children and adults. Numerous insurers and various professional organizations recognize

bilateral CIs as the standard-of-care treatment for individuals with bilateral severe to profound hearing loss. When it is determined that a patient is a candidate for a CI, one of the decisions that needs to be made is determination of which ear to implant (if it is determined the patient will receive a unilateral implant), which ear to implant first (if the patient is receiving sequential bilateral implants), or if the patient should receive simultaneous bilateral CIs. If being provided with a unilateral implant, the determination of which ear to implant can be influenced by several factors. Many clinics routinely implant the ear with the least amount of residual hearing, whereas other clinics routinely implant the patient's "better" hearing ear. Some clinics determine the ear of implant on a case-by-case basis, whereas others leave this decision up to the patient and/or the parents.

Recent studies indicate numerous benefits of bilateral implantation, including benefits for speech perception resulting from overcoming the head shadow effect, improved speech understanding in noise, improved sound localization (Patrick et al., 2006), binaural squelch (Buss et al., 2008), and binaural summation (Dorman et al., 2011). Similar findings have been reported for bilateral implants in the pediatric population (Litovsky et al., 2006), along with more rapid language acquisition (Wie, 2010) and improved speech recognition skills (Wolfe et al., 2007).

Summary of Preoperative Testing

In summary, preoperative testing is used to determine if a patient is a suitable candidate for a CI. Three primary questions that should be addressed in the preoperative evaluation include the following: (1) Can we increase the patient's auditory detection skills? (2) Can we improve the patient's speech understanding when compared to that obtained with a hearing aid? (3) In the pediatric population, is there a good chance that implant use will facilitate or improve spoken language more than would be expected with continued hearing aid use?

It is important for clinicians to remain current regarding candidacy guidelines as the specific criteria used to determine candidacy frequently change and will continue to evolve as technologic advancements are made and patient outcomes continue to improve. Updates regarding current CI candidacy can be obtained by contacting local CI programs or by accessing CI manufacturer websites.

SURGICAL PROCEDURE

Surgery begins with administration of general anesthesia. Hair is shaved above and behind the ear, the skin is prepared with an antiseptic solution, and sterile drapes are placed around the ear. A postauricular incision is made, and a well is created in the skull behind the mastoid bone to accommodate the receiver-stimulator portion of the internal device. The surgeon drills through the mastoid air cells and removes bone between the tympanic membrane and the facial nerve until the round window and the cochlear promontory are visualized. An opening (called a cochleostomy) is made into the basal turn of the scala tympani just anterior to or through the round window, and the electrode array is inserted into the scala tympani. If a ground electrode is attached to the receiver, it is then placed under the temporalis muscle. The receiver-stimulator is placed and secured into the well behind the mastoid, the incision is closed, and a pressure dressing is placed over the ear for 24 hours. Some surgeons may forego the use of the well and instead place the implant receiver in a tight pocket of skin behind the mastoid.

Some special considerations are needed when implanting young children because surgical intervention with this age group requires specific knowledge of temporal bone anatomy and the impact of skull growth on the implanted device. Although temporal bone growth has been shown to continue through adolescence, anatomy of the facial recess is fully developed at birth (Eby, 1996). The most significant developmental changes are in the size and configuration of the mastoid cavity. Eby and Nadol (1986) recommend that the surgeon leave approximately 2.5 cm of additional electrode lead in the mastoid area to accommodate for head growth. Additionally, modifications to the surgical technique or the use of specialized electrode arrays may be necessary when the patient presents with anatomic anomalies, such as cochlear ossification or malformation.

CI surgery typically lasts between 2 and 5 hours depending on the surgeon's experience, the device selected, and the complexity of the anatomy encountered in each patient. Most clinics perform this as an outpatient procedure, enabling patients to return home in less than 24 hours. Children and adults return to their normal routines when they feel well enough to do so, often within 1 week of surgery.

Cochlear implantation has the same risks as other procedures conducted under general anesthesia and those of other surgeries of the middle or inner ear. In centers with considerable experience performing CI surgery, such risks are quite limited and are greatly outweighed by the advantages that the properly selected patient will obtain from the implant. Risks include a remote possibility of infection, temporary or permanent facial paralysis on the operated side, mild temporary taste disturbances, tinnitus, and vertigo. In traditional CI surgery, one may expect loss of any residual hearing in the implanted ear, as well as mild pain and numbness at the site of the incision following the surgery. Removal of the CI may become necessary if the internal device suffers electrical or mechanical damage, if an infection at the site cannot be successfully treated with medication, or if the device or the electrode array becomes displaced. Such surgical intervention is similar in scope to initial placement of the device, although generally less risky because of the drilling having been completed at the original operation.

CI recipients must avoid various medical/surgical procedures that could damage the implanted device or the

functioning auditory nerve fibers that transmit the electrical signal to the brain. The use of monopolar electrosurgical instruments in the region of the head or neck, diathermy, neurostimulation, ionizing radiation therapy involving the area of the implant, electroconvulsive therapy, and MRI must all be avoided because they can cause excessive magnetic and electromagnetic interference, which may result in demagnetization of the internal magnet, displacement of the device, and/or disruption of the device electronics. Two commercial CI devices, the Nucleus Freedom and Advanced Bionics HR 90K, are manufactured with a removable internal magnet and may be preferable for patients who are expected to need MRI in the future, such as those who suffer from multiple intracranial tumors related to neurofibromatosis type II. The MED-EL Cochlear Implant System, which does not have a removable magnet, is FDA-approved for use with MRI at a maximum strength of 1.5 Tesla.

POSTOPERATIVE MANAGEMENT: PROGRAMMING THE DEVICE

After surgery, patients return to their implant center for initial programming of their device. The recommended amount of time between surgery and device programming varies; some centers activate the device within days, whereas others may wait several weeks. Waiting for device activation allows for healing and reduction of swelling around the incision, which will enable the headset magnet to adhere properly. The particular procedures followed for the activation will vary depending on the patient's age as well as on the device that was implanted.

For all devices, initial programming begins with connection of the patient's speech processor to the audiologist's computer. This interface of hardware to the internally implanted device enables the clinician to perform objective and subjective measurements needed to appropriately set the device. For all devices, telemetry is performed at the beginning of the appointment and provides valuable information regarding the status of the internal device. Telemetry systems work by sampling, digitizing, and reporting back to the clinician information about the voltage generated on the internal electrodes during stimulation. Telemetry measurements provide information about electrode impedances, short-circuited electrodes, and voltage compliance for each of the electrodes in the array (Abbas et al., 2006). Such measurements guide clinicians in their determination of which electrodes to include or exclude from the recipient's program.

Next, the clinician works with the patient to determine the softest level of sound that results in hearing for each of the electrodes. This level is referred to as "threshold" and is recorded on a computer using programming software. Additionally, the upper level of stimulation is determined for each active electrode. Depending on the type of device used, the level of stimulation is increased until the patient reports the sound is "most comfortable" (M level) or is loud but comfortable (C level). This process results in creation of a speech processor program tailored to the hearing of the individual being tested.

Programming of the electrodes and creation of the speech processor program can be particularly challenging with young children and with patients who are unable to provide feedback regarding the perceptibility and loudness of the electrical signal. With young children, traditional behavioral test techniques, such as behavioral observation audiometry (BOA), visual reinforcement audiometry (VRA), and conditioned play audiometry (CPA) (see Chapter 24), are used to determine threshold and comfort-level measurements. Additionally, objective measures, such as neural response telemetry (NRT), neural response imaging (NRI), auditory nerve response telemetry (ART), and measurement of electrically evoked stapedial reflexes (ESRT), may aid in determination of programming levels. The reader is referred to Hughes (2013) for additional information regarding the use of objective measures to develop speech processor programs for CI recipients.

Various programming techniques are used to refine the patient's psychophysical measures, such as loudness balancing, pitch scaling, and sweeping of the active electrodes. The finalized psychophysical data are applied to a particular speech processing strategy using programming software. Once it is determined that the program is appropriate, it is transferred to the patient's speech processor, and the patient takes it home to use following completion of the initial programming session.

The total number of electrodes used by a patient may be less than the number of electrodes available on the array. Determination of which electrodes to use may be influenced by factors such as telemetry test results, mode of stimulation, the patient's response to electrical stimulation, encoding strategy, and surgical placement of the electrode array. For example, the patient may experience discomfort, dizziness, or facial nerve stimulation when one or more of the electrodes are stimulated. When this occurs, the electrode is simply deactivated, which means it will not be used in the patient's program and will not cause discomfort. Additionally, the optimal number of electrodes differs for the various speech processing strategies. Thus, several of the electrodes along the array may be deactivated to use only the optimal number needed for a particular speech processing strategy. Lastly, an incomplete insertion of the electrode array may limit the number of electrodes that can be stimulated. Such incomplete insertions may occur with cochlear malformations or cochlear ossification.

The number of times patients return to their implant center for continued programming varies greatly and can be affected by factors such as the patient's psychophysical/programming needs, distance traveled to reach the implant facility, implant center protocols, and occupational demands. In many clinics, children are seen twice a month for the first

3 months, monthly for the next 3 months, and then every 6 to 12 months to program their speech processor. During such visits, warble tone thresholds should be obtained in a sound field while the patient utilizes the speech processor to verify the appropriateness of the patient's program. At many centers, speech perception testing is performed every 6 to 12 months.

Adult patients are typically seen twice a month the first month, and then 3, 6, and 12 months postactivation. Most patients are then seen annually for programming and speech perception testing. Such testing is valuable because it can be compared to previous speech perception test results to determine if performance has improved or declined. Decreases in scores are unusual and may indicate that the current program is not optimal for the patient or that there is a problem with the external equipment or the internal device.

(RE)HABILITATION

The goal of postoperative rehabilitation and training is to maximize oral communication (OC) skills with the device. The amount of rehabilitation provided to recipients varies greatly and will depend on several factors, including performance with the device, length of deafness prior to implantation, primary communication mode, and availability of services offered at school and by the implant program. With children, speech and language therapy is essential to maximize spoken language outcomes received with a CI.

Postlingually Deafened Adults

Most postlingually deafened adults have only mild rehabilitative needs following adequate adjustment of their device. Such rehabilitation primarily focuses on training in the proper care and use of the device, utilization of assistive listening devices with the CI, and training to maximize communication ability in difficult listening situations, such as listening in the presence of background noise and using the device on the telephone. Some adults enroll in classes to improve their speech-reading skills. Additionally, there are many computer-assisted programs and online aural rehabilitation resources that adults may use to improve their outcomes with the device.

Prelingually Deafened Adults

Prelingually deafened adults demonstrate greater rehabilitative needs than postlingually deafened adults. Because they tend to demonstrate a higher nonuse rate than postlingually deafened adults, it is recommended they receive preoperative counseling to ensure they have realistic expectations regarding performance and that they be seen more often than postlingually deafened adults for the first year following activation. Such visits provide an opportunity for ongoing counseling regarding issues related to dissatisfaction with the device. Additionally, adjustments can be made to patients' programs to maximize the clarity of the signal they are receiving. This is often needed because prelingually deafened adults may demonstrate more frequent changes in their hearing ability than postlingually deafened adults. These changes often include increased consistency in their ability to detect sound, gradual increase in the upper level of the dynamic range (increased comfort or most comfortable levels), and an improved ability to do loudness balance scaling. Many prelingually deafened adults report that initial stimulation of their device results in a sensation of feeling, rather than hearing. This feeling may occur in the forehead, sternum, throat, or the area around the ear. Increased experience with sound, however, often facilitates a transformation from feeling to hearing.

There are several rehabilitative programs designed for use by both pre- and postlingually deafened adults. All three implant manufacturers offer computer programs for purchase that are aimed at improving the speech perception skills of adults and children. Thomas and Zwolan (2006) found that use of the Rosetta Stone language learning software (Fairfield Language Technologies, Harrisonburg, VA) fostered statistically significant improvements for speech intelligibility, auditory comprehension, and reading level for prelingually deafened adults when the program was used consistently at home for a period of 3 months. Home-based computer programs appear to be particularly beneficial for prelingually deafened adults because they are self-paced and provide the implant recipient with a nonthreatening learning partner.

Children

The (re)habilitative needs of children with CIs are great. This is an important part of the implant process that must be provided if the child is to receive maximum benefit from the device. Such (re)habilitation should include parent training and parental involvement; speech perception training and assessment; speech and language assessment and training that includes speech production and receptive and expressive language; and involvement of the child's teachers. In many instances, the child's school is the primary provider of (re)habilitative services. The audiologist and/or speech–language pathologist on the CI team may additionally work with the child on rehabilitative tasks and make recommendations to the child's school regarding (re)habilitative needs. In some cases, children attend private therapy in addition to that provided by their school system. For specific information regarding rehabilitation therapy techniques commonly used with children with CIs, the reader is referred to Allum (1996) and Estabrooks and Birkenshaw-Fleming (2006). Additionally, each CI manufacturer provides excellent resources for parents, educators, and hearing professionals regarding pediatric aural (re)habilitation and device troubleshooting.

Use of a Hearing Aid in the Contralateral Ear

CI recipients receive a variety of recommendations regarding use of a contralateral hearing aid with a CI. Such recommendations should be made on a case-by-case basis and take into consideration the amount of hearing in the nonimplanted ear, appropriateness of the hearing aid for the patient's loss, the willingness of the recipient to continue using the contralateral aid, and the patient's early level of performance with the CI. Continued use of the aid following CI activation may be recommended by some clinicians, whereas others may recommend removal of the contralateral hearing aid as they feel it may increase the amount of time it takes to adjust to the sound quality of the implant. Some clinicians may recommend reintroduction of the aid once the recipient has accepted the sound quality of the CI. It is important to note that continued use of the device on the contralateral ear may foster bimodal benefit and may also positively impact the performance of that ear if it receives a sequential CI later on.

AN OVERVIEW OF CURRENT SPEECH PERCEPTION AND SPEECH AND LANGUAGE PERFORMANCE

Adults

Performance of adults with CIs is greatly affected by several factors, including age at onset of deafness, length of deafness, and primary communication method. Patients who lost their hearing prior to the development of speech and language skills (prelingually deafened adults) typically demonstrate poorer speech perception skills with an implant than postlingually deafened adults (Skinner et al., 1992; Waltzman and Cohen, 1999; Zwolan et al., 1996). Postimplant changes in speech recognition scores vary greatly for prelingually deafened adults. Some demonstrate progress, whereas others demonstrate little or no change in scores over time, even when combined with intensive rehabilitation (Yang et al., 2011). Because of these factors, prelingually deafened adults demonstrate a higher device nonuse rate than postlingually deafened adults. However, many prelingually deafened adults use their device regularly, report that they are satisfied with their device, and report that using the CI improves both their expressive and receptive communication skills (Zwolan et al., 1996).

The speech recognition performance currently obtained by postlingually deafened adults far exceeds that envisioned when CIs were first introduced as well as the results obtained by prelingually deafened adults. At minimum, most postlingually deafened adults demonstrate greatly enhanced lipreading skills when using their CI, and most hear so well that they are able to converse interactively over the telephone. Results of the most recent multichannel clinical trial for the Nucleus Freedom device in adults indicate a mean group score of 57% correct for CNC words, a mean score of 78% correct for HINT sentences in quiet, and a mean score of 64% correct for HINT sentences in noise (Balkany et al., 2007).

Children

Several factors are known to impact children's performance with a CI, including age at onset of profound deafness, age at which the child receives the implant, status of the cochlea, amount of residual hearing prior to implantation, presence of additional disabilities, and the child's educational environment, to name a few. Recent publications indicate that many children who receive CIs at a young age approach levels of speech perception and speech–language performance similar to those attained by children with normal hearing (Eisenberg et al., 2006; Geers et al., 2003; Taitelbaum-Swead et al., 2005). Such outcomes far exceed those anticipated when CIs were first introduced.

Age at Implant

Current FDA guidelines for the three commercially available devices indicate that it is appropriate to implant children as young as 12 months of age. There appear to be several arguments that support such an early age for implantation. First, providing an implant at an early age maximizes the amount of auditory information available to the child during the critical period for learning language (Cairns, 1986; Dale, 1976) and providing the child with sound decreases the length of auditory deprivation, which has a positive effect on the outcome (Young, 1994). Numerous investigators report that patients implanted at younger ages attain better speech perception scores and oral language capabilities than children implanted at older ages (Miyamoto et al., 2003; Waltzman et al., 2002). Recent research suggests that the optimal age for children to receive an implant is prior to their second birthday as children implanted prior to 2 years demonstrate significantly better expressive and receptive language skills than children who receive the device after their second birthday (Markman et al., 2011; Niparko et al., 2010). Even earlier implantation is supported by Houston et al. (2012), who found that after about 2 to 6 months of CI use, infants who received a CI at earlier ages (7 to 15 months of age) performed similar to infants with normal hearing on a preword learning task, whereas infants who received their implants at later ages (16 to 25 months of age) demonstrated poorer performance than their same-aged peers with normal hearing.

Status of the Cochlea

Preoperative CT or MRI provides important information regarding the status of the cochlea and is used when

providing preoperative counseling to families regarding anticipated outcomes with a CI. Recipients who present with abnormal cochleae, such as those with congenital malformations or cochlear ossification, may experience reduced performance because of incomplete insertion of the electrode array, insufficient loudness growth, or deactivation of electrodes because of electrical stimulation of the facial nerve. However, patients who present with such anomalies are often considered to be candidates for a CI (Balkany et al., 1996; Tucci et al., 1995). El-Kashlan et al. (2003) found that, although prelingually deafened children with postmeningitic hearing loss and ossified cochleae received significant benefit from CIs, their mean overall performance was poorer than that of children with CIs and nonossified cochleae. Eisenman et al. (2001) found that some children with radiographic cochlear malformations performed as well as their matched counterparts with normal cochleae, although their improvements occurred more slowly over time and that children with more severe malformations demonstrated poorer performance than children with mild abnormalities or normal cochleae. CIs are contraindicated for patients with an absent auditory nerve whereas patients with a hypoplastic nerve may develop an ability to detect sound with a CI but often demonstrate reduced outcomes. Buchman et al. (2011) advocate for considering alternative forms of intervention, such as ABIs, when patients with severe anomalies fail to receive benefit from a CI.

Additional Disabilities/Diagnoses

Some children may demonstrate disabilities secondary to their deafness that may affect performance with a CI. Noncognitive disabilities, such as blindness and cerebral palsy, are not likely to impact the decision to implant or to impact the child's eventual performance with a CI. Cognitive disabilities, on the other hand, are likely to impact performance (Waltzman et al., 2000) and may affect the decision regarding candidacy. These conditions include children diagnosed with learning disabilities secondary to meningitis and children diagnosed with mental impairments such as autism (Donaldson et al., 2004). If a child with cognitive disabilities receives a CI, appropriate expectations must be set at home and at school regarding expected performance with the device.

Recently, there has been a large increase in the number of children with ANSD who receive CIs. Auditory neuropathy spectrum disorder (ANSD) is a term used to describe an auditory disorder characterized by recordable OAEs and/or cochlear microphonics, absent or atypical ABRs, and speech recognition skills that are poorer than would be expected based on the audiogram (Rapin and Gravel, 2003). Determination of candidacy for a CI in patients with AD can be difficult because such patients may meet candidacy criteria based on their poor speech recognition skills but may fail to meet criteria based on their audiometric thresholds being better than current criteria indicate. Determination of CI candidacy for patients with AD is additionally complicated by the finding that some patients demonstrate improvement of detection and speech recognition skills over time without a CI (Neault, 2003) and that many children who present with AD also present with additional medical diagnoses that may affect expected performance outcomes (Rance et al., 1999).

Several investigators have reported that CI recipients with ANSD demonstrate postoperative outcomes that are similar to those obtained by more traditional CI recipients (Budenz et al., 2013; Sininger and Trautwein, 2002) and hypothesize that cochlear implantation is successful in such recipients because it provides a supraphysiological electrical stimulation to the auditory nerve, with the hope of reintroducing synchronous neural activity that cannot be achieved with acoustic stimulation (Mason et al., 2003). Because of the potential loss of residual hearing, determination of implant candidacy for a child with AD should be made carefully and should be determined on a case-by-case basis. One factor that should receive strong consideration is the child's spoken language skills, as the presence of a severe language delay demonstrates a need for intervention.

Communication Methodology

When a child is identified with a hearing loss, parents must select a treatment plan that determines the child's primary method for communicating with others. These communication options include American Sign Language (ASL), simultaneous (total) communication (TC), oral communication (OC), and the auditory–verbal (AV) approach (see Chapter 44). Such options vary greatly in the amount of emphasis they place on the child's use of spoken language, and therefore, can affect outcomes with a CI.

Several investigators have compared the speech perception and speech and language skills of children with CIs who use these various methods of communication. Many recent studies report that children trained to use either AV or OC demonstrate more rapid gains in spoken communication abilities than children who use TC (Tobey et al., 2004). Geers et al. (2003) compared speech and language and speech perception measures of children who received a CI prior to the age of 2 and found that most children with average learning ability who used OC produced and understood the English language at a level comparable to that of their peers with normal hearing.

With regard to educational placement, there is good agreement that children will perform optimally with a CI if their school supports the child in his or her use of the device, offers aggressive auditory management and treatment, and provides an optimal auditory environment that promotes and encourages auditory development.

SURGICAL COMPLICATIONS AND DEVICE FAILURES

Occasionally, patients will experience failure of the internal device or surgical complications will occur that will necessitate surgical revision or replacement of the internal device. Possible reasons for such intervention include mechanical device failure, less than optimal electrode placement, skin flap complications, need for technologic upgrade, or intratemporal pathologic conditions (Donatelli et al., 2005). Mechanical failures of the internal device are often referred to as either "hard failures," which are unequivocal device failures identified or verified by integrity testing performed by the device manufacturer, or "soft failures," which occur when the implant recipient experiences discomfort or an unexplained decrease in clinical benefit, even though manufacturer-conducted integrity testing indicates the device is functioning within normal limits. The "hard" failure rates reported by clinics vary and range from a low of about 3% (Donatelli et al., 2005) to a high of 11% of all patients, and 15% of pediatric patients (Parisier et al., 1991). Fortunately, many studies have found that reimplantation following device failure is a viable option and that many CI recipients show improved or stable benefit following reimplantation (Buchman et al., 2011; Donatelli et al., 2005; Ray et al., 2004).

FUTURE DIRECTIONS

Future advances in the field of CIs are highly anticipated and expected. Some implant manufacturers are evaluating techniques for drug delivery to the cochlea via the electrode array. Such drugs could prevent further degeneration of the auditory system or may help keep electrode impedances and power requirements low by preventing tissue growth within the cochlea following implantation (Hochmair et al., 2006). Internal and external components of CI systems will continue to decrease in size whereas their design, flexibility, and function will continue to improve. Additionally, it is likely that completely implantable CI systems will be available in the future. Implant recipients will continue to demonstrate even greater speech recognition skills, enhanced music perception, and improved hearing enjoyment in quiet and in noise as new and improved speech processing strategies are developed.

FOOD FOR THOUGHT

1. Implant candidates are scanned to examine the integrity of the cochlea and to identify any structural abnormalities, including the presence or absence of ossification. What would cause ossification, and to what degree would it impact the decision to implant?
2. As mentioned earlier, cochlear implantation is conducted under general anesthesia. What concerns would the surgeon consider regarding the use of general anesthesia?
3. The FDA has approved CIs for children as young as 12 months of age. Many centers provide CIs to children who are younger than 12 months. What are the possible reasons for implanting sooner, and also, what are some advantages of implanting sooner than 12 months of age? What types of concerns might an audiologist have regarding provision of an implant to a child is who less than 12 months?

KEY REFERENCES

thePoint A full list of references for this chapter can be found at http://thePoint.lww.com. Below are the key references for this chapter.

Abbas PJ, Brown CJ, Etler CP. (2006) Electrophysiology and device telemetry. In: Waltzman SB, Roland JT, eds. *Cochlear Implants.* 2nd ed. New York: Thieme Medical Publishers.

Balkany T, Hodges A, Menapace C, Hazard L, Driscoll C, Gantz B, et al. (2007) Nucleus Freedom North American clinical trial. *Otolaryngol Head Neck Surg.* 136, 757–762.

Buchman CA, Teagle HF, Roush PA, Park LR, Hatch D, Woodard J, et al. (2011) Cochlear implantation in children with labyrinthine anomalies and cochlear nerve deficiency: implications for auditory brainstem implantation. *Laryngoscope.* 121 (9), 1979–1988.

Budenz CL, Telian SA, Arnedt C, Starr K, Arts HA, Zwolan TA. (2013) Outcomes of cochlear implantation in children with isolated auditory neuropathy versus cochlear hearing loss. *Otol Neurotol.* 34, 477–483.

Buss E, Pillsbury HC, Buchman CA, Pillsbury C, Clark MS, Haynes DS. (2008) Multi-center U.S. bilateral Med-El cochlear implantation study: speech perception over the first year of use. *Ear Hear.* 29 (1), 20–32.

Donaldson AI, Heavner KS, Zwolan TA. (2004) Measuring progress in children with autism spectrum disorder who have cochlear implants. *Arch Otolaryngol Head Neck Surg.* 130, 666–671.

Donatelli A, Zwolan TA, Telian S. (2005) Cochlear implant failures and revision. *Otol Neurotol.* 26, 624–634.

Dorman MF, Yost WA, Wilson BA, Gifford R. (2011) Speech perception and sound localization with bilateral cochlear implants. *Semin Hear.* 32, 73–89.

Dun CA, Faber HT, de Wolf MJ, Cremers CW, Hol MK. (2011) An overview of different systems: the bone-anchored hearing aid. *Adv Otorhinolaryngol.* 71, 22–31.

Eisenman DJ, Ashbaugh C, Zwolan TA, Arts HA, Telian SA. (2001) Implantation of the malformed cochlea. *Otol Neurotol.* 22, 834–841.

El-Kashlan HK, Ashbaugh C, Zwolan T, Telian SA. (2003) Cochlear implantation in prelingually deaf children with ossified cochleae. *Otol Neurotol.* 24, 596–600.

Firszt JB, Holden LK, Skinner MW, Tobey EA, Peterson A, Gaggl W, et al. (2004) Recognition of speech presented at soft to loud levels by adult cochlear implant recipients of three cochlear implant systems. *Ear Hear.* 25, 375–387.

Geers AE, Nicholas JG, Sedey AL. (2003) Language skills of children with early cochlear implantation. *Ear Hear.* 24, 46S–58S.

Gifford R. (2013) *Cochlear Implant Patient Assessment: Evaluation of Vandidacy, Performance, and Outcomes.* San Diego, CA: Plural Publishing.

Gstoettner W, Helbig S, Maier N, Kiefer J, Radeloff A, Adukna O. (2006a) Ipsilateral electric acoustic stimulation of the auditory system: long term preservation of acoustic hearing. *Audiol Neurootol.* 11 (suppl 1), 49–56.

Gstoettner W, Van de Heyning P, O'Connor AF, Morera C, Sainz M. (2006b) Results from a multi-centre EAS clinical investigation. *Wien Med Wochenschr.* 156 (suppl 119), 121.

Houston DM, Stewart J, Moberly A, Hollich G, Miyamoto R. (2012) Word learning in deaf children with cochlear implants: effects of early auditory experience. *Dev Sci.* 15, 448–461.

Hughes M. (2013) *Objective Measures in Cochlear Implants.* San Diego, CA: Plural Publishing.

Kileny PR, Zwolan TA. (2004) Perioperative, transtympanic electric ABR in pediatric cochlear implant candidates. *Cochlear Implants Int.* 5 (suppl 1), 23–25.

Litovsky R, Parkinson A, Arcaroli J, Sammeth C. (2006) Simultaneous bilateral cochlear implantation in adults: a multicenter clinical study. *Ear Hear.* 27 (6), 714–731.

Markman TM, Quittner AL, Eisenberg LS, Tobey EA, Thal D, Niparko JK, et al.; CDACI Investigative Team. (2011) Language development after cochlear implant: an epigenetic model. *J Neurodev Disord.* 3 (4), 388–404.

Miyamoto RT, Houston DM, Kirk KI, Perdew AE, Svirsky ME. (2003) Language development in deaf infants following cochlear implantation. *Acta Otolaryngol.* 123, 241–244.

Mudry A, Tjellstrom A. (2011) Historical background of bone conduction hearing devices and bone conduction hearing aids. *Adv Otorhinolaryngol.* 71, 1–9.

Niparko JK, Tobey EA, Thal DJ, Eisenberg LS, Wang NY, Quittner AL, et al.; CDACI Investigative Team. (2010) Spoken language development in children following cochlear implantation. *J Am Med Assoc.* 303 (15), 1498–1506.

Patrick JF, Busby PA, Gibson PJ. (2006) The development of the Nucleus Freedom cochlear implant system. *Trends Amplif.* 10, 175–200.

Rapin I, Gravel J. (2003) "Auditory neuropathy": physiologic and pathologic evidence calls for more diagnostic specificity. *Int J Pediatr Otorhinolaryngol.* 67, 707–728.

Ray J, Proops D, Donaldson I. (2004) Explantation and reimplantation of cochlear implants. *Cochlear Implants Int.* 5, 160–167.

Sininger YS, Trautwein P. (2002) Electrical stimulation of the auditory nerve via cochlear implants in patients with auditory neuropathy. *Ann Otol Rhinol Laryngol Suppl.* 189, 23–31.

Taitelbaum-Swead R, Kishon-Rabin L, Kaplan-Neeman R, Muchnik C, Kronenberg J, Hildesheimer M. (2005) Speech perception of children using Nucleus, Clarion or Med-El cochlear implants. *Int J Pediatr Otorhinolaryngol.* 69, 1675–1683.

Thomas E, Zwolan T. (2006) Benefit of language software for prelingual/teen adult CI users. Paper presentation at the Ninth International Conference on Cochlear Implants and Related Sciences, Vienna, Austria.

Tobey EA, Rekart D, Buckley K. (2004) Mode of communication and classroom placement impact on speech intelligibility. *Arch Otolaryngol Head Neck Surg.* 130, 639–643.

Waltzman SB, Cohen NL, Green J, Roland JT. (2002) Long-term effects of cochlear implants in children. *Otolaryngol Head Neck Surg.* 126, 505–511.

Waltzman SB, Scalchunes V, Cohen NL. (2000) Performance of multiply handicapped children using cochlear implants. *Am J Otol.* 21, 329–335.

Wie OB. (2010) Language development in children after receiving bilateral cochlear implants between 5 and 18 months. *Int J Pediatr Otorhinolaryngol.* 74, 1258–1266.

Wiley S, Jahnke M, Meinzen-Derr J, Choo D. (2005) Perceived qualitative benefits of cochlear implants in children with multi-handicaps. *Int J Pediatr Otorhinolaryngol.* 69, 791–798.

Wilson BS. (2000) Strategies for representing speech information with cochlear implants. In: Niparko J, ed. *Cochlear Implants: Principles and Practices.* Philadelphia, PA: Lippincott Williams & Wilkins.

Wolfe J, Schafer E. (2010) *Programming Cochlear Implants.* San Diego, CA: Plural Publishing.

Yang WS, Moon IS, Kim HN, Lee WS, Lee SE, Choi JY. (2011) Delayed cochlear implantation in adults with prelingual severe-to-profound hearing loss. *Otol Neurotol.* 32 (2), 223–228.

CHAPTER 44

Intervention, Education, and Therapy for Children with Hearing Loss

Christine Yoshinaga-Itano and Kristin M. Uhler

INTRODUCTION

The role of the audiologist in the management of families of children who are deaf or hard of hearing (D/HH) has evolved markedly since the beginnings of newborn hearing screenings. The audiologist is often the first point of contact for families whose child does not pass a newborn hearing screening and is in a unique position to ensure their child's hearing health and also overall well-being. Audiologists counsel the family regarding the impact of hearing loss (HL*) on the child's development and, where appropriate, may raise the question of an additional disability from their observations, developmental screenings, or concerns voiced by parents. As children grow, audiologists continue to play a vital role in their patients' academic, social, and emotional development.

This chapter will begin with a review of the federal laws that define and support services to children with HL and their families. We will then consider current evidence-based practices in intervention, education, and therapy, some fundamental guidelines and principles and best practices for family-centered care, and quality indicators of educational services to school-aged students.

SPECIAL EDUCATION LAWS

Individuals with Disabilities Education Act

As described in Chapter 26, children with disabilities are protected by the Individuals with Disabilities Education Act (IDEA) (US Department of Education, 2006). "Hearing impairment" and "deafness" are categorized as disabilities under this law. The IDEA is divided into sections; for our purposes we will consider Parts B and C.

Part C provides early intervention for children with disabilities from birth to age 3. The following services are included under this section: (1) Home visits, family training, and counseling; (2) special instruction; (3) audiology and speech pathology services; (4) sign language and cued language services; (5) vision services; (6) assistive technology, including hearing aids, and other services.

The law also requires the assignment of a **service coordinator,** who in most situations is the first contact. If the coordinator has limited background in HL, the audiologist can play a major role in educating him/her and the entire Part C system about the child's needs and development, including the urgency of aggressive intervention (Yoshinaga-Itano et al., 1998).

Part C requires that all services be described and monitored with an individualized family service plan (IFSP), which must be completed within 45 days after the referral of the child to EI services. The IFSP involves a comprehensive multidisciplinary evaluation (with at least two different disciplinary areas of expertise) as well as an identification of services needed. The IFSP is evaluated annually in the first 3 years of life and reviewed every 6 months if needed.

The evaluation must include the assessment of (1) cognitive development, (2) physical development (including vision and hearing), (3) communication development, (4) social-emotional development, and (5) adaptive development. It also includes a family-directed assessment of the family's resources, priorities, and concerns and identification of the supports needed by the family to meet their child's needs. Children with permanent bilateral HL are eligible for Part C services; however, each state determines specific eligibility criteria, so children with unilateral HL, children with minimal HL (mild, high frequency, low frequency, borderline), auditory neuropathy/dyssynchrony, and children with medically manageable HL may be excluded from eligibility. Audiologists should play a major role in defining eligibility for services if their state's Part C system does not include all children who are D/HH.

Transition to Part B Services

When children reach age 3, they are re-evaluated to determine their eligibility for support in a preschool program. To

*In this chapter HL refers to hearing loss and not hearing level.

help plan ahead, families are notified of a transition planning conference no less than 90 days before the child's third birthday. Every state has different criteria for inclusion in Part B services for children who are D/HH (Johnson and Seaton, 2012).

Part B is applicable to children aged 3 to 21 and addresses the academic, developmental, and functional needs of the child. As described in Chapter 26, schools are required to provide an eligible child with a free appropriate public education in the least restrictive environment as described in the child's individualized education plan (IEP). The IEP is a document that describes learning goals and responsible service providers.

An important refinement of Part B includes several "special factors" provisions, including one tailored to D/HH students: "[i]n the case of a child who is deaf or hard of hearing, consider the child's language and communication needs, opportunities for direct communications with peers and professional personnel in the child's language and communication mode, academic level, and full range of needs, including opportunities for direct instruction in the child's language and communication mode." This requirement helps the IEP team address the unique communication and learning challenges experienced by children who are D/HH.

Section 504 of the Rehabilitation Act of 1973

Children who do not qualify for special education services under Part B of IDEA may qualify for services guaranteed by Section 504 of the Rehabilitation Act of 1973 (Johnson and Seaton, 2012). A 504 plan will provide the student with accommodations so that the child may access the information and content in the educational setting. The 504 plan may indicate that the child is in need of assistive technology such as FM devices, real-time captioning, sign language interpreters, and/or note-takers. Section 504 can be considered the educational equivalent of the Americans with Disabilities Act, and as such assures that the child who is D/HH has access to communication. However, because they are functioning at grade level, children under 504 plans do not receive any specialized educational instruction or special education direct services. See Chapter 26 for more discussion on 504 plans.

GUIDELINES AND BEST PRACTICES

In addition to federal laws, pediatric audiologic practices are informed by guidelines and descriptions of evidence-based best practice. The following section will review several materials developed to guide families and children through the educational "maze."

The Joint Commission on Infant Hearing (JCIH) Position Statement (2013)

Unlike other disabilities, EI services provided to infants/children who are D/HH and their families are designed to prevent delay in most of the children identified through universal newborn hearing screening (UNHS) programs. This distinction, which places children who are D/HH in a "risk prevention" category, is unique and in contrast to special education services that are initiated only when a significant delay is demonstrated. Therefore, it was necessary to delineate the characteristics of quality early intervention for infants/children who are D/HH and their families. The JCIH responded to this need. The JCIH has representatives from the fields of audiology, pediatrics, education, otolaryngology, and speech-language development. In 2013, the JCIH updated its 2007 position statement with the following 12 goals:

Goal 1. All children who are deaf or hard of hearing (D/HH) and their families have access to timely and coordinated entry into EI programs supported by a data management system capable of tracking families and children from confirmation of HL to enrollment into EI services.

Goal 2. All children who are D/HH and their families experience timely access to service coordinators who have specialized knowledge and skills related to working with individuals who are D/HH.

Goal 3. All children who are D/HH from birth to 3 years of age and their families have EI providers who have the professional qualifications and core knowledge and skills to optimize the child's development and child/family well-being.

- Goal 3a. Intervention services to teach American Sign Language (ASL) will be provided by professionals who have native or fluent skills and are trained to teach parents/families and young children.
- Goal 3b. Intervention services to develop listening and spoken language will be provided by professionals who have specialized skills and knowledge.

Goal 4. All children who are D/HH with additional disabilities and their families have access to specialists who have the professional qualifications and specialized knowledge and skills to support and promote optimal developmental outcomes.

Goal 5. All children who are D/HH and their families from culturally diverse backgrounds and/or from non-English-speaking homes have access to culturally competent services with provision of the same quality and quantity of information given to families from the majority culture.

Goal 6. All children who are D/HH should have their progress monitored every 6 months from birth to 36 months of age, through a protocol that includes the use of standardized, norm-referenced developmental evaluations,

for language (spoken and/or signed); the modality of communication (auditory, visual, and/or augmentative); social-emotional and cognitive issues; and fine and gross motor skills.

Goal 7. All children who are identified with HL of any degree, including those with unilateral or slight HL, those with auditory neural HL (auditory neuropathy), and those with progressive or fluctuating HL, receive appropriate monitoring and immediate follow-up intervention services where appropriate.

Goal 8. Families will be active participants in the development and implementation of EHDI systems at the state/territory and local levels.

Goal 9. All families will have access to other families who have children who are D/HH and who are appropriately trained to provide culturally and linguistically sensitive support, mentorship, and guidance.

Goal 10. Individuals who are D/HH will be active participants in the development and implementation of EHDI systems at the national, state/territory, and local levels. Their participation will be an expected and integral component of the EHDI systems.

Goal 11. All children who are D/HH and their families have access to support, mentorship, and guidance from individuals who are D/HH.

Goal 12. As best practices are increasingly identified and implemented, all children who are D/HH and their families will be assured of fidelity in the implementation of the intervention they receive.

These goals will serve as foundations for discussions on "continuous improvement" among pediatric practitioners for years to come.

Best Practices for Family-Centered Early Intervention

A second document identifies a set of best practices in the area of early intervention for families and their children (Moeller et al., 2013). This document was developed by EI providers and parents from a number of countries at an international conference. Readers are encouraged to obtain this article, which includes topics not addressed in the JCIH document, including informed choice and decision-making, family/provider partnerships, family social and emotional support, family/infant interaction, use of assistive technology and supporting means of communication, and collaborative teamwork.

Quality Standards for Educational Services for School-Aged Children Who Are D/HH

An example of quality standards for provision of educational services for children who are D/HH is the *Colorado Quality Standards for Programs and Services for Children who are Deaf or Hard of Hearing* document. Examples of programmatic quality standards include these:

- programs will provide a clear statement of purpose, including outcomes for expected learning, communication competency, and social/emotional well-being, and
- programs will provide training to general education personnel regarding accommodations, modifications of the curriculum, and understanding of the impact of HL on development and learning.

Predeveloped standards such as these help programs know what "quality" looks like.

Deaf Child's Bill of Rights

The Deaf Child's Bill of Rights has been adopted by a growing number of states and includes some additional IEP considerations for children who are D/HH. The Deaf Child's Bill of Rights includes using a communication plan for children who are in general education. Rights include the following:

1. The IEP team cannot deny instructional opportunity based on the amount of the child's residual hearing, the ability of the parent(s) to communicate, or the child's experience with other communication modes. When discussing these issues, the following questions may be helpful to clarify the child's needs: When considering the child's primary communication mode, is there just one? Does the child use a combination of modes? What mode do the parents use with their child? What mode does the child use to communicate with his/her friends?
2. The plan must include a statement documenting that an explanation was given of all educational options provided by the school district and available to the child. When considering all educational options, are the options available in the school district?
3. The plan must include a statement documenting that the IEP team, in addressing the child's needs, considered the availability of D/HH role models and a D/HH peer group of the child's communication mode or language. Because of the low incidence of a hearing disability, many students who are D/HH find themselves without contact with other children with HL. Additionally, 95% of these children are born into families with normal hearing (NH), which can further increase isolation. Questions to ask: Is there an opportunity to hold weekly "chats" online with other D/HH kids? Does the family know about regional activities designed for D/HH children? Explore all known opportunities.
4. The plan must include a statement that the teachers, interpreters, and other specialists delivering the communication plan to the student must have demonstrated proficiency in, and be able to accommodate for, the child's primary communication mode or language. Discuss the

communication proficiency of the child/student's service providers and write a statement of the needs of the staff. Is training/inservice/mentoring a possibility? Is there an accommodation not being utilized?

5. The Plan must include a statement of the communication-accessible academic instruction, school services, and extracurricular activities that the student will receive. These questions may help to clarify the student's needs: Is the student enjoying full access to academic instruction and services? To extracurricular activities? Audiologists should pay special attention to the IEP checklist for Recommended Accommodations and Modifications. Are TTY's, captioned television, interpreters for field trips, and so on being utilized?

More information on the Deaf Child's Bill of Rights can be found at this website: http://www.nad.org/issues/education/k-12/bill-of-rights.

Colorado Individual Performance Profile

The Colorado Individual Performance Profile (CIPP) was developed as a tool for the IEP placement process. Use of the CIPP assures that there is uniform decision-making across the state for placement decisions into a specific type of educational setting. Without a placement tool such as the CIPP, there is no guarantee that decisions for placement are evidence based or that children who are D/HH will receive appropriate services (Yoshinaga and Ruberry, 1992).

The CIPP sets child performance criteria for placement in one of six possible educational settings:

1. Indirect services via monitoring only (typically 504 plans);
2. Indirect services via consultation with the teacher of the mainstream classroom requiring an IEP;
3. Direct services: More than 60% of the time in the mainstream classroom with 1 to 4 hours per week of specialized instruction from a teacher of the deaf or other special education provider;
4. Direct services: 21% to 60% of the time in the mainstream classroom with 1 to 2 hours of daily specialized instruction;
5. Direct services: Less than 20% of the time in the mainstream classroom with 3 to 4 hours of daily specialized instruction; and
6. Direct services in a separate facility.

Students are assessed in Reading, Language, Math, Social Skills, and Cognitive Skills on assessments that are norm referenced so that children who are D/HH can be compared to their peers with NH. Further information about the CIPP can be found on the Colorado Department of Education website: http://cospl.coalliance.org/fedora/repository/co:2157/ed14408d342002internet.pdf

Placement and Readiness Checklists

Audiologists who participate in IEP meetings may also find the Placement and Readiness Checklists (PARC) helpful (http://www.handsandvoices.org/pdf/PARC_2011.pdf). The PARC are designed to assist in the IEP process.

There are two parts to the PARC. Part 1 addresses essential skills needed by the student to actively and meaningfully participate in the general education classroom. It includes four checklists: General Education Inclusion Readiness, Interpreted/Transliterated Education Readiness, Captioning/Transcribing Readiness, and Instructional Communication Access. These checklists can be used in combination or independently, depending on the student and the purpose of the review.

Inclusion in the general education classroom is evaluated through the General Education Inclusion Readiness Checklist. Identification of skills needed to benefit from services in the general education classroom is evaluated through the Interpreted/Transliterated Education and the Captioning/Transcribing Readiness checklists. Analysis of how a student accesses instruction is evaluated through the Instructional Communication Access Checklist. The Readiness checklists can also be used to identify IEP goals that will assist a student with acquisition of the necessary skills as well as a tool to monitor the acquisition of the desired skills.

Part 2 is the Placement Checklist, which evaluates the accessibility and appropriateness of the general education setting, including the physical environment, the general learning environment, the instructional style of the teacher, the school culture, and how well the learning environment is matched to the student's communication, language, and social needs. The Placement Checklist can be used as often as the classroom environment changes or other needs suggest monitoring.

SUPPORTING FAMILIES

Of course, we must always keep in mind that audiologic services are provided to each child within the context of the family. A child's learning success is strongly correlated to family support, and family support is enhanced when the audiologist takes every opportunity to "stay in step" with the family during its journey. Following are important considerations to include in the audiologic care.

Setting the Stage for Healthy Development

Counseling strategies are critically important for diagnostic audiologists. As bearers of difficult news, our skill in delivering the information and supporting parents through their emotional adjustment to the confirmation that their infant/child is D/HH is of utmost importance (English et al.,

2007). Emotional anxiety can be reduced if the family receives information in a timely manner and when the early intervention support provided is immediate, skilled, and knowledgeable about early childhood deafness (JCIH, 2013). A multilayered system of support should include the audiologist, educators, speech-pathologists, pediatricians, other parents, and D/HH professionals.

Bonding and Celebration of the New Family Member

The most important job of the first contact is to refocus families on the special time of getting to know their newborn. Bonding of mothers to babies is an important factor in the rate of language development (Pressman et al., 1999), and audiologists should actively inquire about the child's overall development. For example, what is the baby's personality? What seems to calm or upset the baby? Has the baby had his/her first smile? Does the baby smile back when the mother/father smiles at him/her? Does the baby seem to be visually alert? What have they learned about this new member of their family? Are they recording important developmental progress through diaries and pictures? Does the baby initiate interaction with parents, siblings, or pets? How does the baby react if someone is sad or angry? Does the baby react to facial expressions and to tone of voice?

We do not want families to miss the amazing development that takes place in the first year of life. It is important for parents to be emotionally available to their child and to observe whether their babies are emotionally available to them. We are trying to teach parents to observe and know their babies. This will help when we begin to ask them to observe responses to auditory stimuli, especially their voices.

Immediately after the birth, some mothers suffer from postpartum depression (Hoffman and Drotar, 1991). Audiologists and EI providers need to know the boundary between grieving as a direct result of the diagnosis of deafness or HL and more severe clinical depression that requires referral to mental health professionals.

Reducing and Preventing Stress

Parental stress levels are strongly associated with language development of children who are D/HH (Hintermair, 2007; Pipp-Siegel et al., 2001). Parents report lower stress levels when they are provided with immediate support after the confirmation of HL and have the opportunity to explore their opportunities and options. The important message for the parents is that babies react to their emotions. The more they take care of themselves, the happier their baby will be, and the less parental stress, the faster the language development (Pipp-Siegel et al., 2001). With appropriate EI services, parents are usually able to deal with stress without requiring support from mental health professionals, and in fact tend to cope as well as parents with typically developing children do, even as they contend with multiple appointments, hearing aids, new information, and weekly EI services. It is also important to remember that stress does not disappear as a child grows up—it just changes. Each new stage of a child's life (first day of school, first sport try-out, first driving lesson, first summer job) can bring a new type of stress to the family. Audiologists will want to heed warning signs and have a referral process at hand if it appears parents are struggling more than expected.

Counseling Strategies

Audiologists need to learn a variety of counseling strategies. Delivering information in a manner that is the most effective for diverse populations is an important skill. Hersey and Blanchard's (1993) model can be adapted to provide a framework for an audiologist's understanding of the family's skill/resources and readiness/motivation to participate in audiology or intervention services. The model identifies four different styles of counseling: (1) Telling, (2) selling, (3) collaborating/participating, and (4) delegating.

Telling

Families with the lowest levels of involvement and readiness may respond best to directive counseling approaches. In this approach, the professional provides the family with information about "what to do next." The goal of this type of counseling is to assist and support the families' development from one style of counseling to one that provides the family with more responsibility for decision-making. Cultural differences may also impact the choice of counseling techniques. In cultures where families have the expectation that professionals know best about what to do, they may expect to be told what to do next. In fact, they may interpret the presentation of a number of options as indication that the professional is not knowledgeable or skilled (Jezewski and Sotnik, 2001). As families become more acculturated, learn and adapt to the culture of the country in which they now live, they may respond very differently to other types of counseling styles.

Selling

For families with higher levels of knowledge/involvement but lower levels of readiness/motivation, the counseling approach that may be most beneficial is one that provides the family with the motivation to pursue a specific plan of action, such as keeping hearing aids on the child or learning sign language. If the parents feel convinced that the child is more responsive when wearing the hearing aids or by learning sign language, their motivation to follow-through may be significantly enhanced.

Collaborating/Participating

For families with higher levels of readiness and lower levels of knowledge/involvement, the family may respond best to collaborative counseling. In these situations, the family does not need encouragement for motivation, but it does need more information and support from the audiologist.

Delegating

For families that are at the highest levels of knowledge/involvement and readiness/motivation, the counseling strategy that may be most effective is one that provides the families with the information about resources and delegates them to proceed, checking back when they need additional information or direction.

The same model can also be useful for counseling children as they grow. They too have different levels of knowledge/involvement and readiness/motivation and these levels can change with age. For example, the counseling approach that worked best when the child was younger, highly motivated, and very knowledgeable may cease to be the approach of choice when the child is older, for example, social situations change, and the child is no longer convinced that wearing the hearing aid is beneficial/necessary or is important enough to overcome perceived social judgment and negative social circumstances. Arming the child with the information necessary may require a "selling" or "collaborating" technique.

When Families Choose a Goal for Visual Communication

Because an audiologist is a specialist in hearing, when a family chooses a goal for communicating through visual and/or tactile-kinesthetic communication, the audiologist should refer the family to the appropriate specialist with expertise in early childhood deafness and HL. These professionals may include educators of the deaf, speech-language pathologists (with training in both visual communication and auditory spoken language), and/or professionals who are fluent in sign language. Today, families may choose combinations of different communication modes and methods and the audiologist should work in partnership with these professionals.

A significant proportion of children who are D/HH rely on visual communication support even when their primary language access is through auditory spoken language. Auditory access to spoken language can be compromised by suboptimal auditory learning environments (e.g., noisy classrooms, noisy public environments such as supermarkets or parks), breakdowns in technology (e.g., batteries, cords), distance and low intensity of the speech of the conversational partner, structural abnormalities in the sensory system, and/or auditory perceptual disorders. Visual communication support may include adequate lighting, speech reading, natural body language and facial expressions, avoidance of visual noise/clutter, real-time captioning for older children, sign language, and cued speech.

American Sign Language (ASL) is an indigenous sign language of the United States. ASL, like all other languages has its own syntax, grammar, phonology, semantics, and pragmatics. ASL is not a communication approach; it is a language like English, Spanish, Mandarin, or Arabic.

There are two communication approaches that involve ASL. One is the **Bi-Bi (Bilingual-Bicultural)** approach, which refers to communication between people with ASL and written English. A related approach is called **bimodal-bilingualism**. This communication approach involves the use of spoken English and ASL.

Simultaneous communication is a communication approach often mistakenly referred to as total communication. Total communication uses many methods (ASL, spoken English, audition, gestures, fingerspelling, writing) separately or in combinations, in no particular order but as needed to communicate successfully. Simultaneous communication is an approach in which parents and teachers consistently speak and sign simultaneously.

Manually coded English is similar to simultaneous communication. It employs spoken English accompanied by sign language in English word order and often with the use of signs that designate English morphology, such as word endings like /s/, /ed/, /ing/. *Signed English, Seeing Exact English (SEE I), Seeing Essential English (SEE 2), or Linguistics of Visual English (LOVE)* are specific sign systems used in a simultaneous communication approach. They were developed in the 1970s when the use of sign language was first used in public programs for educating children who were D/HH. Signed English is the closest to ASL because it consists of ASL vocabulary and signs in English word order.

Cued Speech is a cueing system that can be used simultaneously with spoken English. The cues have the advantage of being learned rapidly, often in a few days, and are designed to provide visual hand cues to receiver that facilitate the use of speechreading cues. Since only 40% of the phonemes of English are visible on the lips/face, these hand cues assist the listener in separating "visemes" (sounds that look similar on the lips from one another, for example, /b/ and /p/). The hand cues are only related to specific speech sounds; they are not signs having linguistic intent and do not use gestural representations of the meanings of words in the way that a true sign system does.

Today's choices for parents who are hearing include learning strategies for listening and spoken language development, learning one or more visual communication approaches, or learning "all of the above." In some rare cases, it is possible that a child who is D/HH cannot access spoken language, such as a child who is born without an auditory nerve or cochlea, or a child with a severe auditory processing disorder. In these cases, the children may communicate

solely through visual communication, although children with severe auditory processing difficulties may benefit from wearing amplification. However, services are typically initiated before professionals know that a child who is D/HH cannot access spoken language effectively.

Parents who are D/HH and use ASL as their primary communication must consider whether or not they would like their child to also access spoken language through the use of amplification technology and educational services that focus on listening and spoken language.

When Families Choose a Goal for Spoken Language

Most parents who are hearing choose a goal of developing listening and spoken language skills for their child. There are two approaches toward this goal:

Auditory verbal communication uses a unisensory (auditory only) approach. To promote listening, therapists and families often use their hand or a screen to cover their mouths and eliminate visual cues while children focus on the input and their own speech production. In normal conversation, however, infants and toddlers tend to learn listening and spoken language naturally, with creative efforts to highlight sounds and auditory attention (e.g., "I Hear with My Little Ear").

With later-identified children, the auditory verbal approach usually involves a significant amount of drill and practice for the child to master auditory skills. With appropriate learning objectives, children who are D/HH have been able to rapidly develop skills, often with very few examples. When children become dominant auditory spoken language users, they do not rely on speechreading skills and, just as with children who have NH, they do not stare at their communication partner's face to access and understand the spoken language communication. However, this attention to the face may naturally occur in noisy environments, just as it would for children with NH.

In contrast, the **auditory oral** communication approach uses a dual-sensory access to spoken English through both auditory spoken language and speechreading cues. Historically, children and parents learned both auditory stimulation and speechreading skills. However, speechreading lessons are not common today in the instruction of children who are D/HH.

Assuring Optimal Amplification

When families chose spoken language, the audiologist becomes responsible for timely and appropriate fitting of amplification. Additionally, children must not only have technology capable of accessing spoken language, but must also have access to abundant verbal input in their environment. Children learn language by being exposed to language, and families can be shown how to increase that exposure.

The American Academy of Audiology (2013) has developed evidence-based guidelines with prescriptive formulae to fit children with hearing aids. The guidelines recommend using either National Acoustics Laboratory (NAL) or Desired Sensation Level (DSL) prescriptions for infants. Each of these formulae calculates the speech intelligibility index (SII) for varying input levels (soft, medium, and loud), taking into account the child's specific HL and stimulus chosen by the audiologist. The SII is a measure used to quantify audibility of the speech signal (ANSI, 2012) and is described in depth in Chapter 40.

Helping Parents and Teachers with Hearing Aid Management

Parents and teachers have much to learn about maintaining their child's hearing aids and cochlear implants. They need to know how to conduct a daily listening check of their child's hearing aids and be able to determine if the device is providing the desired input. Parents should be given a kit with materials such as a hearing aid air blower, listening stethoscope, battery tester, retention devices (clips and cords), dehumidifier kits, stickers, and information on loss prevention, moisture protection, and "child proofing." Hearing instrumentation orientation should include care of hearing aids and troubleshooting (see Chapter 39). There is much to remember about batteries, including battery life, storage, disposal, insertion, removal, and overnight storage. Information on coupling to assistive devices and telephones is needed. The website Equal Voice for Deaf Children Lesson 104 has a demonstration of troubleshooting instructions for hearing aids and cochlear implants (http://evdcweb.org/level100/lesson104/lesson104.html). For instructions related to specific implants, see Chapter 43.

All children using amplification technology should have their technology checked on a daily basis. The IFSP/IEP team should designate the professional most knowledgeable and available to conduct the daily amplification checks. Children should be taught as early as the preschool years to manage their own amplification and to report when their devices are not working. Without routine monitoring, up to 50% of the amplification used by children today in educational settings are likely to function inappropriately (Burkhalter et al., 2011).

Assessing the Child's Use of Technology

No matter how appropriately the child's amplification is fit, unless the child wears the amplification, the benefit of the amplification will be compromised. Two coordinated tools have been designed to capture parent and teacher input regarding use of amplification. The Parents' Evaluation of Aural/Oral Performance of Children (PEACH) and the Teachers' Evaluation of Aural/Oral Performance of Children (TEACH) are designed for children 3 to 7 years of age

(Ching et al., 2000). The PEACH and TEACH tools consist of an interview with parent or teacher with 15 questions targeting the child's everyday environment. The PEACH/TEACH include scoring for five subscales (Use, Quiet, Noise, Telephone, Environment).

Additionally, a data tracking feature is often available on pediatric hearing aids. Parents should be informed that the hearing aids have a data tracking system and that the audiologist can monitor amplification use. Parents can also be asked how many hours per day that the child wears his/her amplification. If the child is not using amplification often, the first question we should ask is if parents perceived little or no difference in the child when wearing or not wearing the amplification. Could the amplification be "underfit" and result in plugging the child's ears? Is there a way that the audiologist can demonstrate to the family what the child can do with amplification as compared to limitations when they do not wear the amplification?

ASSESSING CHILDREN'S PROGRESS

Auditory Skill Development

In addition to knowledge about the technical aspects of amplification, the audiologist is the expert about auditory skill development. The audiologist assesses auditory skills for several purposes: (1) To assure appropriateness of the amplification for detection and discrimination, (2) to monitor the child's progress in auditory skill development, and (3) to design appropriate habilitative techniques for the development of auditory skills sufficient for the acquisition of listening and spoken language. Auditory skills assessments that can determine the child's development in Detection, Discrimination, Identification, and Comprehension can provide valuable information for the audiologist (Erber, 1982):

Detection: Can the child detect that a stimulus is present?
Discrimination: Can the child detect differences or similarities between stimuli?
Identification: Can the child apply labels to the auditory stimuli that he/she discriminates?
Comprehension: Can the child understand the meaning of spoken stimuli?

A more complex hierarchy of auditory skills development is delineated in the Functional Auditory Performance Indicators (FAPI) discussed later in this chapter.

As soon as amplification is fit, parents should be taught how to be watchful observers of their child's responses to sound. Parents should be encouraged to observe how their child/student responds to the Ling speech sounds (ah, ee, oo, s, sh, m) (Yoshinaga-Itano and Sedey, 2000) and to report back to the audiologists if their child does not respond to these speech sounds (indicating specific frequencies). This informal listening check should be done frequently so if a change in response is noted, adjustments/repairs to amplification can be made quickly. If the amplification is still functional, the listening check might indicate a change in the child's hearing—an important consideration because the percentage of children with progressive HL increases with age (Berrittini et al., 1999). Teachers should also be taught how to do Ling sound checks on a daily basis whether or not the child is educated predominantly in special education services or in the regular classroom.

The measurement of auditory skill development in early childhood is a challenge. A number of observational checklists have been developed for the young child who is D/HH; unfortunately, almost all of these checklists lack any normative data on children with NH. However, since the goal of audiologic intervention is to keep children who are D/HH as comparable to their peers with NH as possible, it is very important to evaluate the children's auditory skills as compared to children with NH.

LittlEars

One parent questionnaire with normative data is the LittlEars Parent Interview (Kühn-Inacker et al., 2003), which is intended for children birth through 24 months. LittlEars consists of 35 questions. The first 18 items deal with auditory awareness, auditory attention, auditory localization, simple discrimination such as recognizing speech on the telephone, and recognizing emotion. The remaining 17 items address speech imitation and language comprehension, such as following commands, understanding simple and complex commands, and knowing family member names, such as Mommy and Daddy. The questionnaire was standardized on 218 NH children aged 0 to 24 months. LittlEars is designed for children with profound HL who are candidates for cochlear implants and for monitoring auditory skill development after implantation.

The Cincinnati Auditory Skills Checklist

This checklist (Meinzen-Derr et al., 2007) includes a number of items on a hierarchy of auditory skills. It provides a more comprehensive overview of the child's auditory skill development and can be used to document progress. The Cincinnati Auditory Skills Checklist (ASC) has 35 items divided into four auditory levels: (1) Detection, (2) Discrimination, (3) Identification, and (4) Comprehension. This checklist was designed for monitoring the auditory skill development of children with all degrees of HL.

Functional Auditory Performance Inventory (FAPI)

The FAPI is a criterion reference checklist and does not have norms for children with NH or children who are

D/HH. It is intended to monitor progress throughout intervention (Johnson and Brown, 2004). The FAPI can provide assistance to parents as they observe their child's auditory skill development on a regular basis. It addresses functional auditory skills in seven categories: (1) Awareness & Meaning of Sound, (2) Auditory Feedback and Integration, (3) Localizing Sound Source, (4) Auditory Discrimination, (5) Auditory Comprehension, (6) Short-Term Auditory Memory, and (7) Linguistic Auditory Processing. It provides a hierarchy of developmental skills and addresses differences in distance and listening conditions and uses a formula to help the therapist or educator define each skill as "not present", "emerging", "in process", or "acquired."

Early Listening Function (ELF)

The ELF (Anderson, 2000), for children age 5 months to 3 years, is not an assessment per se, but rather a validation and "discovery" tool, designed to help parents learn what their child can and cannot hear, with and without hearing aids, when speech and environmental sounds are affected by distance and noise. The ELF helps parents understand the impact of a disability they cannot see and helps them understand the conditions that yield consistent auditory responses from their child. The ELF has 12 listening situations in which the parent observes the child and records the distance the child responds to the auditory stimuli. The 12 activities are conducted in quiet and noisy situations, near the child and also farther away (6 inches, 3, 6, 10, and 15+ feet). Loudness calibration is not essential, but parent participation in typical environments is critical. Parents administer the ELF at home and then share their observations with the audiologist, who can monitor development over time. Anderson (2000) hypothesizes that the ELF may be beneficial to family engagement and empowerment. During ELF listening activities, parents are encouraged to envision the size of the child's "listening bubble," meaning the space around the child where audibility is optimal.

Beyond the early childhood period, audiologists may use standard pediatric speech discrimination tests to monitor a child's auditory discrimination ability in quiet and in noise, as described in Chapter 24.

Analyzing the Auditory Spoken Language Environment

Even if technology is fit correctly, children cannot learn to listen and use spoken language at age-appropriate levels unless they are exposed to spoken language. Research indicates this exposure should far exceed the usual input available to children with NH (Aragon and Yoshinaga-Itano, 2012). Technology is now available that is capable of characterizing the auditory/language environments of children who are D/HH. The LENA (Language Environment Analysis) is a small digital language processing recorder, about the size of a credit card carried in a pocket that can capture 10 to 16 hours of the daily auditory environment of the child (see www.lenababy.com). With this technology, parents/families are able to count the number of words spoken to their child in an average day, the number of conversational turns, and the number of child vocalizations. When the recordings are analyzed, the family and therapists receive specific information about the amount of adult spoken language available to the child, the frequency and quality of the child's vocalizations, the amount of silence in a day, and the percentage of the day spent in general noise, TV/radio, meaningful speech (within an audible range), and distant and overlapping speech. Audiologists and parents may be surprised to find that some children spend more than 60% of their day in silence, or 40% of their day amidst TV and radio noise, or that the home environment includes noise that is not under the control of the parent (appliances or outside noise). With this information, families become aware that their goal for their children to learn spoken language will be compromised unless the auditory characteristics of the learning and listening environment are modified.

The LENA instrument provides a mechanism for quantifying the daily spoken language environment or the daily "auditory diet" of the child. Aragon and Yoshinaga-Itano (2012) provided information about the language environment of children who had NH and children who were D/HH in both English- and Spanish-speaking homes. Results indicated that some children who are D/HH spend a high percentage of the day in silence (66% as compared to a mean of 28% for children with NH), in noise (20% as compared to a mean of 3% for children with NH), and with TV/radio playing (33% as compared to a mean of 10% for children with NH). These results prompted the early interventionists and audiologists to investigate the causes of these high percentages. In some cases, the noise levels were not under the control of the family; for example, noise from an old refrigerator or from a nearby river could not be reduced. In some cases, the amount of distant/overlapping language was high and the amount of meaningful language low. Parents were encouraged to read to their child several times a day as a natural way to increase their "word count."

As depicted in Figures 44.1, 44.2 and 44.3, the typical child with NH (in English- and Spanish-speaking homes) has access to an average of approximately 12,000 words a day, has approximately 474 conversational turns, and makes about 2,000 vocalizations. To elicit child vocalizations and conversational turns comparable to hearing children, the child who is D/HH needed to be exposed to an average of 17,000 to 18,000 words per day (Aragon and Yoshinaga-Itano, 2012). Many factors may contribute to this difference, as children who are D/HH are not always wearing their amplification, their amplification is not functioning properly, or the signal to noise ratio may adversely affect the quantity of their vocalizations and their conversational turn-taking.

To date, studies using the LENA system have primarily focused on infants, toddlers, and preschool children.

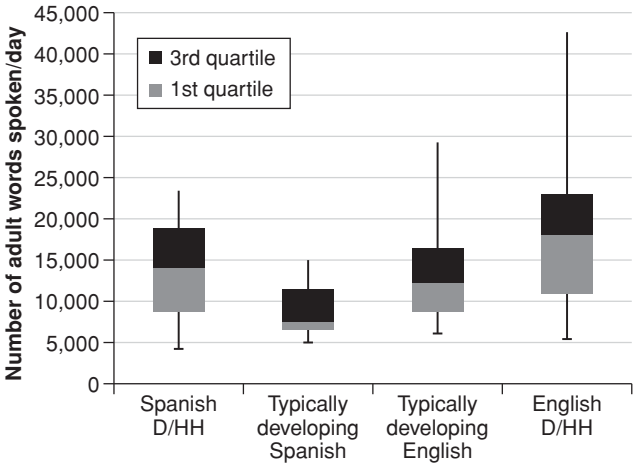


FIGURE 44.1 Adult word count for four groups: D/HH Spanish, typically developing Spanish, D/HH English, and typically developing English (Aragon and Yoshinago-Itano, 2012). [Reprinted with permission from Thieme Publishers.]

Additionally, older children's auditory spoken language environment can be measured with the Functional Listening Evaluation (FLE) (Johnson and Seaton, 2012), which assesses children's auditory abilities within the classroom environment. With the FLE, students are presented words and sentences at close and far distances, in quiet and in noise, to determine their abilities in each condition, as described in Chapter 26.

Vocal/Speech Development

Audiologists typically do not monitor vocal development, but they may be trained to monitor the relationship between auditory skill development and vocal production. Following are some tools used by other professionals for this purpose.

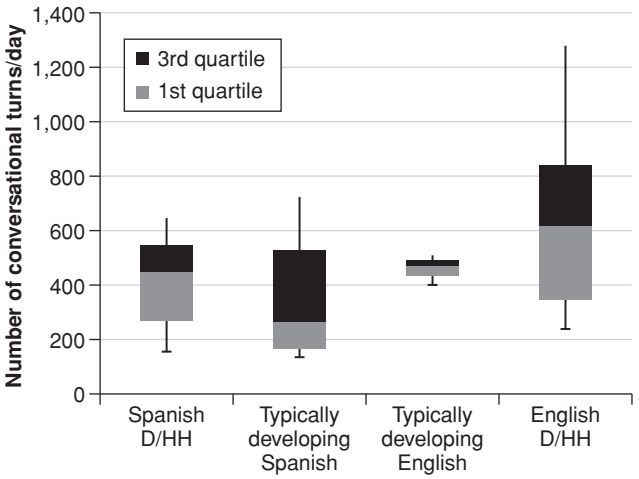


FIGURE 44.2 Conversational turns of four groups: Spanish D/HH, typically developing Spanish, typically developing English, and English D/HH (Aragon and Yoshinago-Itano, 2012). [Reprinted with permission from Thieme Publishers.]

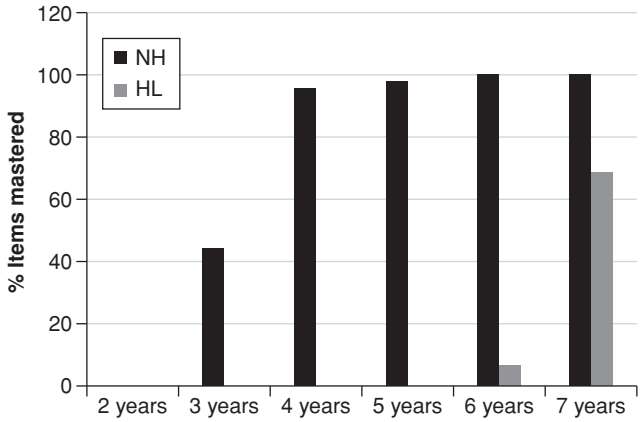


FIGURE 44.3 Percentage of items on Pragmatics Checklist mastered by age and hearing status (Goberis et al., 2012). [Reprinted with permission from Thieme Publishers.].

Assessing Speech Production

Speech-language pathologists often use the Conditioned Assessment of Speech Production (CASP) (Ertmer and Stoel-Gammon, 2008) to assess the speech production of a child who is D/HH. The CASP has three levels of assessment: (1) Pre-canonical Vocalizations (vowels with no consonants); (2) Basic Canonical Syllables (consonant–vowel productions); and (3) Advanced Forms (multisyllabic utterances with multiple consonants). The CASP is a clinically practical and developmentally appropriate tool for auditory-guided speech development in very young children who are D/HH. It provides a quick measure of progress over time of the vocal development for an infant population, an age group for whom vocal production assessment has been absent, and for assessments post-cochlear implantation from 6 to 24 months. The CASP is empirically tested and an objective measure of prelinguistic gains in speech perception and production.

Assessing Vocal Production

The Infant Monitor of Vocal Production (IMP) (Moore, 2010) is a parent interview designed for use in the first year of life. This assessment examines three important transitions in vocal production: (1) Reflexive vocal production that does not require an auditory feedback loop (4 to 6 months of age), (2) vocal production that mimics the suprasegmental and some segmental elements of the native spoken language at about 6 to 7 months of age, and (3) the transition from vocalizations to word production (from 10 to 12 months of age). The IMP has been especially useful

in cochlear implant candidacy considerations for children with auditory neuropathy/dyssynchrony. Collection of normative data is in process at the time of this writing.

Assessing Phoneme Development

Recently, Wiggin et al. (2013) described the emergence of correct production of English phonemes by degree of HL and age and found that, in general, the phoneme development of early-identified children who are D/HH followed the order of development of children with NH with some delays. Children with mild HL produce approximately 20 different consonant types by age 2.5 years, comparable to children with NH. However, as the degree of HL increases, so does the delay in development: Children with moderate HL reach this level by 3 years of age, and children with severe HL reach this level of consonant production by 5 years of age. Children with profound HL who do not have a cochlear implant do not typically develop intelligible speech by 5 years.

Measuring Speech Intelligibility

When children have NH, their parents and others report being able to understand about 25% to 50% of their speech at 15 months of age (Yoshinaga-Itano and Sedey, 2000). By 21 months, parents usually understand almost all of what children say with careful listening, and by 27 months almost all of children are rated as intelligible with little or no effort. Not surprisingly, HL impacts articulation development, and speech intelligibility is affected by the degree of HL. Even children with mild bilateral HL, identified early and receiving weekly EI services have delays in their speech intelligibility to 5 years of age.

A frequently used tool is the Speech Intelligibility Rating Scale (SIRS) (Semar and Metz, 1988), which rates intelligibility on a 6-point scale:

1 = I always or almost always understand the child's speech with little or no effort.
2 = I always or almost always understand the child's speech; however, I need to listen carefully.
3 = I typically understand about half of the child's speech.
4 = I typically understand 25% of the child's speech.
5 = The child's speech is very hard to understand. I typically understand only occasional, isolated words and/or phrases.
6 = I never or almost never understand the child's speech.

Yoshinaga-Itano and Sedey (2000) found that when parents, early interventionists, and independent coders used the SIRS to rate speech intelligibility, their ratings were quite consistent. They also reported learning from using the SIRS over time that children with mild and moderate HL had a more rapid growth rate in intelligibility than children with moderate, moderate-severe, severe, or profound HL.

Assessing Articulation

The *Goldman Fristoe Test of Articulation-2 (GFTA-2)* (Goldman and Fristoe, 2000) is a standardized test of articulation that uses picture cards to elicit words. It is commonly used to assess children who are D/HH. Early-identified children have made significant gains in their speech development, but still have some lingering articulation errors. Because many of these children have very intelligible speech, many of them do not receive intervention services in articulation. However, even at 7 years of age, children who are D/HH have articulation errors that may not interfere with intelligibility but are not a correct production of the phoneme in all positions in words. On the GFTA-2, 42% of children who are D/HH have demonstrated articulation scores below the 10th percentile (Sedey, 2009).

Tomblin et al. (2008) studied the longitudinal speech development of children with cochlear implants with a minimum of 8 years of cochlear implant experience. The development of speech sound production seems to stabilize after 6 years of cochlear implant use and typically approaches a plateau after 8 years of CI use. In this study, mean speech sound production accuracy was 15% pre-implantation, 63% after 4 years of CI use, and 81% after 8 years. The researchers concluded that a positive prognosis for speech production can be made at the 4-year point of cochlear implant use.

Children with HL greater than a puretone average of 50 dB HL have been found to have significant differences in their speech production in the first year of life even when early identified. McGowen et al. (2008) found that children with HL had fewer multisyllable utterances with consonants, fewer fricatives and fewer stops with alveolar-velar stop place, and more restricted front-back tongue positions for vowels than did the children with NH. When children's HL is not identified early, they tend to experience persistent difficulties in speech intelligibility in conversation and production of fricatives from 28 to 84 months of age (Moeller et al., 2010).

Language Development

The assessment of language development begins in infancy. Several assessments have been used, including the following.

MacArthur Bates Communicative Development Inventories

MacArthur Bates Communicative Development Inventories (CDI) (Fenson et al., 1993) is a parent questionnaire assessment of the child's lexicon size, gesture development, vocabulary comprehension, and syntax. The CDI has been used to monitor progress of children who are D/HH in the state of Colorado since the 1980s and is currently being used in 14 other states through the National Early Childhood Assessment Project (Sedey and Yoshinaga-Itano, 2012).

Using the CDI, Mayne et al. (2000) found that children who are D/HH in their region (Colorado) had expressive vocabulary delays even when they had been identified early and had normal cognitive development. By 2.5 years, the typically developing child has approximately 600 words in his/her expressive vocabulary, compared to 400 words among children who are D/HH, one more piece of evidence supporting aggressive intervention.

Expressive One Word Picture Vocabulary Test (EOWPVT-3)

The EOWPVT-3 is a standardized test of expressive vocabulary commonly used in the diagnostic evaluations of children with NH for language disorders and also for children who are D/HH (Brownell, 2000). The expressive vocabulary task requires a child to describe a picture with a single word. Children who are D/HH who are early-identified with appropriate early intervention follow-through have demonstrated vocabulary levels about 6 months delayed from the norms for children with NH from 4 to 7 years of age (Yoshinaga-Itano et al., 2009).

Test of Auditory Comprehension of Language (TACL-3)

The TACL-3 is a picture-pointing task that assesses the development of vocabulary, grammar, and sentence construction (Carrow-Woolfolk, 1999). It has been used to evaluate children who are D/HH and to compare their language development to children with NH. Research in this area indicates that children with and without HL develop similar receptive syntax skills (Yoshinaga-Itano et al., 2010). However, when examining languages samples of children interacting with their parents, delays in expressive syntax were found (Sedey, 2009). Children with NH tended to produce more utterances, words, and morphemes than children who were D/HH. In addition, children who are D/HH had significantly fewer negatives, conjunctions, and personal pronouns than children with NH.

Pragmatics

Pragmatics is the use of language in social situations, involving interpersonal skills such as taking conversational turns, adjusting one's language to the age or status of a communication partner, or rephrasing when one is misunderstood. Pragmatic language development of children who are D/HH in their early years has been neglected in the research literature. To address this concern, Goberis et al. (2012) developed the *Pragmatics Checklist* for parents and teachers to monitor the development of pragmatic language skills. The checklist includes 45 items across 7 pragmatic language categories: (1) Instrumental (request for action/object), (2) Regulatory-Command, (3) Interactional-Social rules/poise/politeness, (4) Personal Expression of Feelings, (5) Heuristic—Questions to obtain information, (6) Imaginative-Pretending, and (7) Informative—Cause and Effect.

Children with no HL usually master all the skills on this checklist by the age of 6. However, in a recent study, children with HL were found to have mastered only three of these skills at this age. This is an important finding relative to long-term child success. Establishing strong pragmatic language skills is important for the development of social skills and literacy. Focus should begin in early childhood but should continue throughout the education of the child. Partnerships between professionals and parents will assist in the development of age-appropriate pragmatic language skills.

Social-Emotional Development

Children who are early identified tend to have personal–social skills within the range of normal development, whereas children who are later identified have demonstrated significant delays in personal–social skill development commensurate with their expressive language skills (Yoshinaga-Itano and Abdala de Uzcategui, 2001). To evaluate these skills, professionals and parents often use the *Child Behavior Checklist* (Achenbach and Rescorla, 2000), designed for children aged 1.5 to 16. Age-appropriate checklists take about 10 minutes to complete and inquire about sleep problems, attention and anxiety, mood swings and different expressions of stress, as well as language development. Results can help determine the need for a referral to a professional counselor.

Another tool designed to help audiologists understand a child's social development is called *My World* (www.idainstitute.com). This counseling tool depicts classrooms and other spaces on a flat board and provides movable avatar figures that a child can use to represent him/her and other people. Children are asked about social situations at school, at home, on the playground, and other settings. The tool provides a vehicle for the child to convey information about his/her ease in listening, learning, communicating in real-world situations, and friendships and other social concerns. It has been successfully used on children who are D/HH as young as 3 years of age.

Barker et al. (2009) in a study of 116 children with severe to profound HL aged 1.5 to 5 years found significant relationships between language, attention, and behavior problems. Children with sensory/neural HL have been found to have higher rates of behavior problems (e.g., aggression, delinquency, and hyperactivity), rates being around 30% to 38%, compared to 3% to 18% for children with NH (van Eldik et al., 2004; Vostanis et al., 1997).

SUMMARY

This chapter examined the impact of HL on learning, from infancy through the school years. We examined the legal mandates, guidelines, and best practices available to families

and their children and the kinds of decisions families face, especially regarding communication. We briefly examined how children's learning challenges are measured and also reviewed some of the evidence-based practices currently being used to support learning success. Needless to say, the audiologist is crucial to this success.

FOOD FOR THOUGHT

1. Two documents (JCIH, 2013; Moeller et al., 2013) are currently being used to define early intervention practices for children who are D/HH and their families. How might these be applicable to the educational needs of older children?
2. Pediatric audiologists need to be careful observers of more than a child's hearing abilities. What other developmental issues should an audiologist monitor?
3. Audiologists need to be experts of the normal development of auditory skills. Observe young children at different age levels and try different auditory tasks with them to familiarize yourselves with what should be expected at each age level.

KEY REFERENCES

thePoint A full list of references for this chapter can be found at http://thePoint.lww.com. Below are the key references for this chapter.

Achenbach TM, Rescorla LA. (2000) *Manual for the ABESPA (Achenbach System of Empirically Based Assessment) Preschool Form and Profiles.* Burlington, VT: University of Vermont.

American Academy of Audiology. (2013) *Clinical Practice Guidelines: Pediatric Amplification.* Reston, VA: Author. Available online at: http://www.audiology.org/resources/documentlibrary/Documents/PediatricAmplificationGuidelines.pdf

American National Standard Institute. (2012) ANSI methods for the calculation of the speech intelligibility index (ANSI, S3.5, 1997, R2007). Available online at: http://asastore.aip.org/shop.do?pID=77

Anderson KL. (2000) Early Listening Function (ELF) checklist. Available online at: http://successforkidswithhearingloss.com/uploads/ELF_Questionnaire.pdf

Aragon M, Yoshinaga-Itano C. (2012) What we can learn from LENA: Language ENvironment Analysis of children to support children who are deaf or hard of hearing. *Semin Speech Lang.* 33 (4), 340–353.

Barker DH, Quittner AL, Fink NE, Eisenberg LS, Tobey EA, Niparko JK, the CDaCI Investigative Team. (2009) Predicting behavior problems in deaf and hearing children: the influences of language, attention, and parent–child communication. *Dev Psychopathol.* 21 (2), 373–392. doi:10.1017/S0954579409000212.

Berrittini S, Ravecca F, Franceschini SS, Matteucci F, Siciliano G, Ursino F. (1999) Progressive sensorineural hearing loss in childhood. *Pediatr Neurol.* 20, 130–136.

Brownell R. (2000) *Expressive One-Word Picture Vocabulary Test-3.* Novato, CA: Academic Therapy Publications.

Burkhalter CL, Blalock L, Herring H, Skaar D. (2011) Hearing aid functioning in the preschool setting: stepping back in time? *Int J Pediatr Otorhinolarygol,* 75 (6), 801–804.

Carrow-Woolfolk E. (1999) *Test of Auditory Comprehension of Language (TACL3).* Los Angeles, CA: Western Psychological Services.

Ching TC, Hill M, Psarros C. (2000) Strategies for evaluation of hearing aid fitting for children. *Paper presented at the International Hearing Aid Research Conference,* Lake Tahoe, CA.

English K, Naeve-Velguth S, Rall E, Uyehara-Isono J, Pittman A. (2007) Development of an instrument to evaluate audiologic counseling skills. *J Am Acad Audiol.* 18 (8), 675–687.

Erber N. (1982) *Auditory Training.* Washington, DC: Alexander Graham Bell Association.

Ertmer DJ, Stoel-Gammon C. (2008) The Conditioned Assessment of Speech Production (CASP): a tool for evaluating auditory-guided speech development in young children with hearing loss. *Volta Rev.* 108, 59–80.

Fenson L, Dale PS, Reznick JS, Thal D, Bates E, Hartung JP, et al. (1993) *MacArthur-Bates Communicative Development Inventories.* San Diego, CA: Singular Publishing Company.

Goberis D, Dalpes M, Abrisch A, Baca R, Yoshinaga-Itano C. (2012) The missing link in language development of deaf and hard of hearing children: pragmatic language development. *Semin Speech Lang.* 33 (4), 297–304.

Goldman R, Fristoe M. (2000) *Goldman-Fristoe Test of Articulation-2 (GFTA-2).*Circle Pines, MN: American Guidance Services.

Hersey P, Blanchard KB. (l993) *Management of organization behavior utilizing human resources (8th ed.).* Englewood Cliffs, NJ: Prentice-Hall.

Hintermair M. (2007) Parental resources, parental stress, and social emotional development of deaf and hard of hearing children. *J Deaf Stud Deaf Educ.* 11 (4), 494–513.

Hoffman Y, Drotar D. (1991) The impact of postpartum depressed mood on mother-infant interaction: like mother like baby? *Infant Ment Health J.* 12, 65–80.

Jezewski MA, Sotnik P. (2001) *Culture Brokering: Providing Culturally Competent Rehabilitation Services to Foreign-Born Persons.* Buffalo, NY: Center for International Rehabilitation Research Information and Exchange.

Johnson C, Brown A. (2004) Functional Auditory Performance Inventory (FAPI). Available online at: http://www.cde.state.co.us/cdesped/download/pdf/FAPI_3-1-04 g.pdf.

Johnson C, Seaver L, DesGeorges J. (2013) *Educational Advocacy for Students Who Are Deaf or Hard of Hearing: The Hands and Voices Guidebook.* Boulder, CO: Hands and Voices.

Johnson CD, Seaton J. (2012) *Educational Audiology Handbook.* 2nd ed. Clifton Park, NY: Delmar Cengage Learning.

Joint Committee on Infant Hearing. (2013) Supplement to the JCIH 2007 Position Statement: principles and guidelines for early intervention after confirmation that a child is deaf or hard of hearing. *Pediatrics.* 131 (4), e1324–e1349. Available online at: http://pediatricsaappublications.answerabc.com/content/131/4/e1324.extract

Kühn-Inacker H, Weichbold V, Tsiakpini L, Coninx S, D'Haese P. (2003) *Little Ears: Auditory Questionnaire.* Innsbruck: MED-EL.

Mayne A, Yoshinaga-Itano C, Sedey AL, Carey A. (2000) Expressive vocabulary development of infants and toddlers who are deaf or hard of hearing. *Volta Rev.* 100, 29–52.

McGowen RS, Nittrouer S, Chenausky K. (2008) Speech production in 12-month-old children with and without hearing loss. *J Speech Lang Hear Res.* 51, 879–888.

Meinzen-Derr J, Wiley S, Creighton J, Choo D. (2007) Auditory Skills Checklist: clinical tool for monitoring functional auditory skill development in young children with cochlear implants. *Ann Otol Rhinol Laryngol.* 116 (11), 812–818.

Moeller MP, McCleary E, Putman C, Tyler-Krings A, Hoover B, Stelmachowicz. (2010) Longitudinal development of phonology and morphology in children with late-identified mild-moderate sensorineural hearing loss. *Ear and Hearing.* 31 (5), 625–635.

Moeller MP, Carr G, Seaver L, Brown AS, Holzinger D. (2013) International consensus statement on best practices for working with families of children who are deaf and hard of hearing. *J Deaf Stud Deaf Educ.* 18 (4), 449–425.

Moore R. (2010) The Infant Monitor of Vocal Production. (IMP). Available online at: http://www.ridbcrenwickcentre.com/imp/

Pipp-Siegel S, Sedey AL, Yoshinaga-Itano C. (2001) Predictors of parental stress of mothers of young children with hearing loss. *J Deaf Stud Deaf Educ.* 7 (1), 1–17.

Pressman L, Pipp-Siegel S, Yoshinaga-Itano C, Kubicek L, Emde RN. (1999) A comparison of the links between emotional availability and language gain in young children with and without hearing loss. *The Volta Review.* 100 (5), 251–277.

Sedey A. (2009) Speech and language growth and predictors of successful outcomes. Paper presented at Penn State Low Incidence Conference, State College, PA.

Sedey A, Yoshinaga-Itano C. (2012) A national examination of language outcomes and development. Paper presented at National EHDI Conference, St. Louis, MO.

Semar V, Metz D. (1988) Criterion validity of Speech Intelligibility Rating-Scale procedures for the hearing-impaired population. *J Speech Hear Res.* 31, 307–316.

Tomblin JB, Peng S-C, Spencer LJ, Lu N. (2008) Long-term trajectories of the development of speech sound production in pediatric cochlear implant recipients. *JSLHR*. 51, 1353–1368.

US Department of Education. (2006) Assistance to states for the education of children with disabilities and preschool grants for children with disabilities; Final Rule, 34 CFR Parts 300 and 301. Available online at: http://idea.ed.gov/download/finalregulations.pdf

Van Eldik T, Treffers PD, Veeman JW, Verhulst FC. (2004) Mental health problems of deaf Dutch children as indicated by parents' responses to the child behavior checklist. *Am Ann Deaf.* 148 (5), 390–395.

Vostanis P, Hayes M, Du Feu M, Warren J. (1997) Detection of behavioral and emotional problems in deaf children and adolescents: comparison of two rating scales. *Child Care Health Dev.* 23, 233–246.

Wiggin M, Moushey JM, Nelson RS, Sedey AL, Yoshinaga-Itano C. (2013) Emergence of consonants in young children with hearing loss. *Volta Rev.* 113 (2), 127–148.

Yoshinaga-Itano C, Abdala de Uzcategui C. (2001) Early identification and social-emotional factors of children with hearing loss and children screened for hearing loss. In: Kurtzer-White E, Luterman D, eds. *Early Childhood Deafness.* Baltimore, MD: York Press Inc; pp 13–28.

Yoshinaga-Itano C, Baca R, Sedey A. (2010) Describing the trajectory of language development in the presence of severe to profound hearing loss: a closer look at children with cochlear implants versus hearing aids. *Otol Neurotol.* 31 (8), 1268–1274.

Yoshinaga-Itano C, Ruberry J. (1992). The colorado individual performance profile for hearing-impaired students: a data-driven approach to decision-making. *The Volta Review.* 94 (2), 159–187.

Yoshinaga-Itano C, Sedey A. (2000) Speech development of deaf and hard-of-hearing children in early childhood: interrelationships with language and hearing. *Volta Rev.* 100 (2), 181–212.

Yoshinaga-Itano C, Sedey A, Baca R. (2009) Longitudinal outcomes from birth through 7 years. XIIth Symposium on Cochlear Implants with Children, Seattle, WA.

Yoshinaga-Itano C, Sedey AL, Coulter DK, Mehl AL. (1998) The language of early- and later-identified children with hearing loss. *Pediatrics*. 102 (5), 1161–1171.

CHAPTER 45

Audiologic Rehabilitation

Joseph Montano

AUDIOLOGIC REHABILITATION

Many consider audiologic/aural rehabilitation (AR) to be the heart and soul of the profession of Audiology. Certainly it was the impetus for the military origins of the field and continues to be the way we define our profession. However, in practice, AR takes a back seat to diagnostic procedures and, in fact, may not be a reimbursed service through third-party insurers in the United States. Why the discrepancy then between practice and perception?

When audiologists are asked about their role in the AR process, opinions vary greatly. Some may feel it defines everything we do in our profession, whereas others consider it to be specialized treatments, such as speechreading and auditory training (AT). Audiologists even disagree on what to call this area of expertise: Aural, audiologic, or auditory rehabilitation. Few, however, would argue that audiologists play a pivotal role in the management of hearing loss in children and adults, regardless of what it is called or how it is defined.

Hearing health care continues to evolve and professional roles and boundaries have begun to blur. It is important that we have an understanding about the provision of audiology services, in this ever-changing healthcare landscape, and that we adapt the philosophies, skills, and knowledge that define our profession to the current state of service provision. With that in mind, this chapter will provide the reader with a foundation in AR that reflects the changes occurring in the provision of health services and infuse the process into a framework reflecting our practice environment.

The Changing Healthcare Arena

Audiology has, for the most part, been practiced within a medical/biomedical model of service delivery (Erdman, 2014; Erdman et al., 1994): A top-down system whereby patients are seen for assessment procedures resulting in traditional diagnoses and treatments. In this model, care is directed by the audiologist and the patients are, for the most part, passive in the process. They are expected to comply with the recommendations put forth by the audiologist that, more often than not, result in the acquisition of hearing aids for remediation of hearing loss. This method of service delivery can be particularly limiting when seen within the context of the ever-evolving world of patient-centered care.

Global healthcare initiatives are investing heavily in the move toward improved quality of health care through patient-centered practice. The Institute of Medicine (2001) emphasizes the importance of health provision that is patient driven and mindful of individuals' beliefs, wishes, and values. Medical decisions are guided by the inclusion of the person in the development of treatment goals and activities. The recent passage of the Patient Protection and Affordable Care Act (2010) is an example of legislation that encompasses the ideals of patient-centered care and the current shift in beliefs that influence health provision.

For the field of Audiology to embrace this patient-centered care philosophy, there needs to be a rethinking of the model used in practice, a shift from the biomedical to the biopsychosocial or rehabilitative methods of service delivery. This model would embrace the inclusion of the patient in the assessment and treatment decisions, a horizontal approach where the audiologist works together with the patient and family to agree on a method of treatment. The decisions are collaborative rather than prescriptive. For example, hearing aid recommendations should be made on the basis of the psychosocial and behavioral impact of the hearing loss rather than just the audiometric results. We need to listen to what the patient needs rather than tell them what they should have.

Consider for a moment the treatments historically associated with the provision of AR service: Hearing aid orientation, AT, speechreading, and communication groups. One can easily see how they can fit within a biomedical model of service provision. Once a hearing loss is identified, these options can be recommended as part of a treatment plan. Early definitions of AR tended to focus their attention on these procedural specific elements without consideration of the underlying characteristics of hearing loss and the impact it has on function.

Publications through the World Health Organization (WHO), most notably the International Classification of Impairment, Disability and Handicap (WHO, 1980) and the International Classification of Function, Disability and Health (2001), greatly influenced the development of more recent AR definitions and contributed to the move

toward patient-centered care-related content. ASHA (2001) described AR as an "ecological, interactive process that facilitates one's ability to minimize or prevent the limitations and restrictions that auditory dysfunctions can impose on well-being and communication, including interpersonal, psychosocial, educational and vocational functions" (p 2). Multiple factors are identified in this definition aligning themselves with the principles associated with patient-centered care. The emphasis on the "interactive process," function, and environmental influences highlight the importance of collaboration in the AR process.

Recently, Montano (2014) defined AR as "… a person-centered approach to assessment and management of hearing loss that encourages the creation of a therapeutic environment conducive to a shared decision process which is necessary to explore and reduce the impact of hearing loss on communication, activities, and participations …" (p 27). Key elements within this statement embrace the concepts closely aligned with changes occurring within the healthcare arena. Use of terminology such as "a patient-centered approach" and "shared decision process" reflects the paradigm shift currently underway. In addition, terms such as "activities" and "participations" are associated with the concepts presented in the WHO ICF (2001). Thus, I believe that the incorporation of AR into practice should include a biopsychosocial model of service delivery and not be restricted to the biomedical standard.

The provision of AR services is frequency focused on the results obtained from an audiogram with the emphasis largely on the dispensing of some kind of amplification device(s). This has been classified as a technocentric approach and follows the biomedical model of service delivery (Montano, 2011). AR practiced within the biopsychosocial model would stress the importance of counseling thereby considered a patient-centered approach, but not to the exclusion of amplification devices.

Table 45.1 compares and contrasts components of both the technocentric and patient-centered models. Although many of the services described may be similar, philosophically they differ. For example, although assessment of hearing is certainly important in both models, standard audiometric evaluation is more relevant in the technocentric

TABLE 45.1

Comparison of the Technocentric and Patient-Centered Models of Service Provision in Audiologic Rehabilitation

Technocentric Model	Patient-Centered Model
History—obtaining a detailed history contributes to differential diagnosis. Traditional methods typically involve the use of closed-set questionnaires	A patient narrative fosters a dialogue between clinician and patient through the use of open-set inquiry
Audiometry—precise evaluation of hearing is essential to the development of all specific AR goals	Self-assessment is a valuable tool to measure the impact of hearing loss on a person's function. This, along with the audiometric information, presents a more complete picture for the audiologist and can be insightful for the patient
Hearing assistive technology systems are critical to achieving improved communication in the presence of hearing loss. The use of hearing aids and assistive devices becomes the emphasis of treatment	Hearing assistive technology systems are certainly important but need not be the sole basis for intervention. They should be incorporated into a process of adjustment to hearing loss
Hearing aid orientation tends to become the emphasis of the therapeutic aspect of treatment	Interventions are decided through shared decision making and may include procedural therapies such as auditory/visual training and individual and group treatment
Verification is critical when amplification is part of the treatment plan	Verification is critical when amplification is part of the treatment plan and results need to be interpreted with regard to patient input and preference
Accessories are plentiful and add another layer of technology to an often complex adjustment patients are experiencing	Regardless of the technology devices or accessories, the emphasis is placed on person's needs and abilities
Follow-up consists primarily of acoustic modifications to amplification	Counseling-based follow-up, support, and encouragement are emphasized with referrals to outside sources, as necessary, such as consumer groups

TABLE 45.2

Examples of Auditory and Nonauditory Consequences of Hearing Loss

Auditory Consequences	Nonauditory Consequences
Patient is unable to follow conversations even in quiet environments	Increased social isolation, depression, may appear confused, anger
Patient is unable to hear effectively at office meetings	Nervous that job may be at risk, anxious that he/she may have missed information that was pertinent to his/her work responsibilities
Patient cannot hear whispers	Loss of intimacy, no longer enjoys quiet times at home with spouse, misses sounds of nature
Patient is unable to hear conversations in restaurants	Avoids going out with friends, is disengaged during conversations, feels left out
Patient is unable to hear the television unless the volume is very loud	Spouse watches same program in another room, arguments about the television volume
Patient struggles to hear conversation when there is even the slightest noise in the room	Works hard trying to focus on visual cues, exhaustion, irritability
Patient is unable to hear on the telephone or even the telephone ring	Anxious that he/she may miss an important call, refuses to answer the phone

example and self-assessment is a critical component of the patient-centered approach. Even basic procedures like obtaining patient history are fundamentally approached in different ways with one method using standard history intake forms and the other engaging in a dialogue or patient narrative. Therefore, combining these two approaches is likely to provide the best outcome.

The goal of AR is to help the person adjust to issues related to living with hearing loss, including both auditory and nonauditory aspects (Erdman, 2000). Thus, the audiologist should keep in mind that he/she is not only trying to obtain the best audiometric result, in general, but concerned about the specific psychosocial implications for the person. These may include issues related to depression, anxiety, increased isolation, and lack of intimacy, to name a few. Table 45.2 describes some examples of auditory and nonauditory consequences of hearing loss.

For every auditory characteristic of hearing loss, there are potential nonauditory ramifications. For example, a man who is unable to communicate effectively at an office meeting may feel anxious that his job could be in jeopardy or perhaps insecure that he will be able to understand important details presented. The impact of hearing loss is rarely just auditory and the audiologist must be perceptive to the possible related issues. As such, for him/her to build a relationship and help patients adjust to hearing loss, counseling needs to be a component of all aspects of AR, both assessment and treatment. Counseling then is the backdrop to the management of patients with hearing loss.

COUNSELING

Counseling is at the heart of the patient-centered approached described by Montano (2011). Although audiologists are aware of the importance of counseling their patients about hearing loss and hearing aids, many believe that it is a process that may be independent from their normal clinic responsibilities. I believe that counseling is critical for the person with a hearing loss. Clark and English (2014) reported that counseling is infused into all aspects of audiology practice and is the basis for developing a "common ground" with patients throughout each phase of assessment and treatment.

Counseling, within the audiologist's scope of practice, is considered by many to consist of two components: Information/education and personal adjustment/support (Clark and English, 2014; Luterman, 2001). That is, counseling tends to focus on providing patients and their families with education to help them self-manage their hearing loss and monitor their reaction to the information they are receiving. This is accomplished by a combination of careful observation and good listening skills. Telling a person that they need hearing aids may be upsetting to some resulting in an emotional response, thus the audiologist must be prepared for different reactions to such recommendations.

The audiologists' counseling role is not restricted to being purely educational, but rather is a critical partner in the journey toward patient acceptance of hearing loss. As such, counseling provides the support necessary for patients to better understand the impact of hearing loss in their lives. It requires the audiologist to be aware of the emotional adjustment one may undertake when dealing with a communication loss such as hearing impairment and be able to recognize the feelings expressed both verbally and nonverbally through their interactions.

A misconception is that the process is the same as therapeutic interventions performed by psychologists, social

workers, and other mental health professionals, when in reality it is the foundation for relationship building that will enhance the AR process. Although the audiologist is the professional who is most knowledgeable about hearing loss and the impact it may have on psychosocial functioning, should he/she encounter a patient presenting with clinical signs beyond those related to adjustment to hearing loss, an appropriate referral should be made. With the vast majority of patients, however, the audiologist is there to support them as they learn to live with hearing loss.

When counseling is included in a patient-centered care approach to AR, it can lead the audiologist in many directions. Counseling can provide the audiologist an understanding of the patient's journey and perspective on the hearing loss. Gregory (2012) described a possible patient journey tool developed by affiliates of the Ida Institute. The tool was designed to highlight the potential phases and directions one may go through on their way to seeking hearing health care. For example, early on in the journey, a patient may identify difficulty hearing in a certain situation such as a family gathering; or perhaps, a spouse might ask that the television volume be lowered. These early indications of hearing loss continue until the person acknowledges the possibility of hearing loss and begins to seek treatment. The use of patient journey narratives also illustrates the intricate nature and psychosocial manifestations of hearing loss (Manchaiah and Stephens, 2011).

Readiness for Change

An understanding of the person's journey can provide the audiologist with valuable information on the readiness of the patient to begin the AR process. This concept has been well researched in areas related to behavior change such as addictions, weight loss, and diabetes management. Similarly, Goldstein and Stephens (1981) identified four categories of "aural rehabilitation types" (see Table 45.3). Classifications range from those who are highly motivated to seek out AR services to those who are extremely negative about dealing with issues related to hearing loss. Judgments as to which category a patient may be classified can be made with knowledge obtained through a narrative history, responses to self-assessment questionnaires, or even the referral source for audiologic evaluation.

The stages of change model was developed with the notion that behavioral adjustment occurs in increments and that people move intentionally toward decision making (Prochaska and DiClemente, 1983). This model has recently been adapted by the Ida Institute (2011) to illustrate the stages of change faced by patients as they move toward AR treatment. As seen in Figure 45.1, a circle represents patient evolution from a stage of pre-contemplation/contemplation to preparation and action moving toward both successful maintenance and permanent exit or, in some cases, relapse, only to return to the circle of change at a later time. To progress through the cycle, patients seek out education, information, advice, encouragement, and support with the audiologist as the professional capable of meeting all these needs. By understanding the patient journey, knowing each story, and acknowledging his/her position within the stages of change, the audiologist has the foundation to provide a comprehensive AR program.

The audiologist and patient work together to determine which components of an AR program are necessary to help develop improved communication skills and ultimately establish successful management of hearing loss. Although amplification may be a vital element of this process, alone it will likely fall short from meeting this primary objective. The AR components, whether they consist of supportive counseling, information on communication strategies, or therapeutic intervention, are necessary to achieve favorable outcomes. For the audiologist to offer comprehensive solutions for patients with hearing loss, they must

TABLE 45.3

Audiologic Rehabilitation Candidacy Types

Type	Description	Example Characteristics
Type I	Very positive, motivated to improve	Self-referred, positive outlook, looking forward to improving communication skills
Type II	Positive with some complications	Concerned about stigma, acknowledges need for help, but nervous about hearing aids
Type III	Mostly negative, but agreeable to attempt trial with amplification	Referred by family member, significant concerns about hearing aids, may look to restrict use of amplification
Type IV	Negative, rejects possibility of hearing aids and AR	Forced by family member to seek treatment, agrees to hearing test but not AR consult

Adapted from Goldstein D, Stephens D. (1981) Audiologic rehabilitation: management model I. *Audiology*. 20, 432–452.

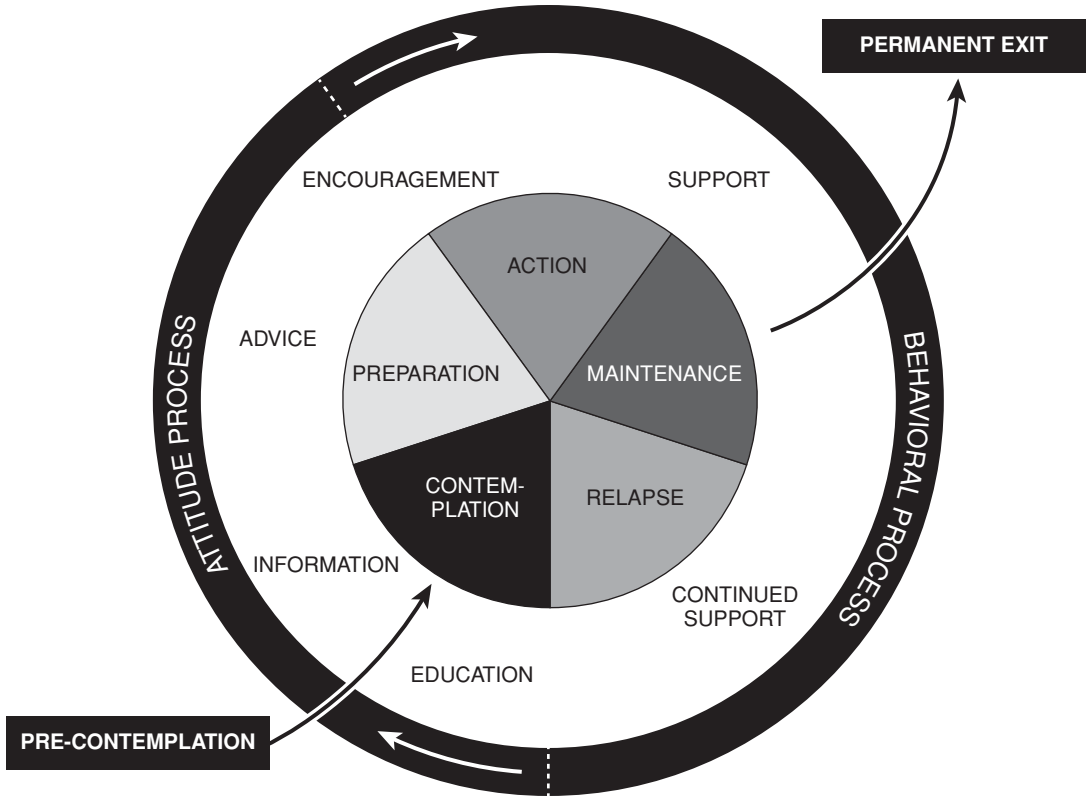


FIGURE 45.1 Ida Institute Circle Tool: Readiness for change.

be aware of not only the technology available to meet the amplification needs, but the existing procedural therapeutic interventions.

OPTIONS FOR THERAPEUTIC AR INTERVENTION

Although it would be wonderful if the use of hearing assistive technologies would resolve all the communication difficulties faced by individuals with hearing loss, this is not the case. Even the most skilled hearing aid user will report difficulties communicating in certain listening environments. Often, hearing aid dispensing takes place in optimal settings, quiet offices, and clinics and does not reflect the real-world conditions patients face on a daily basis. Although noise simulation programs are available from most hearing aid manufacturers, they only approximate the patient environments. Not only must they approach communicating with a sensory deficit, but this process must take place in locations where noise may be present, lighting may be insufficient, speakers may have accents or poor conversational skills, and discussion topics can change rapidly. To maximize communication performance, the person with hearing loss must learn to take advantage of all the sources of information available to assist with the interpretation of auditory cues. To best accomplish this, they must use visual and auditory information and communication strategies and be prepared for communication failures. As such, these concepts become the focus of communication therapy, most commonly referred to as auditory/visual training (AT and speechreading).

Auditory Training

In their historical perspective of AT, Kricos and McCarthy (2007) traced the roots of AT back to the 19th century. Although AT practice has been a component of AR since the evolution of Audiology after World War II (Ross, 1997), in recent years there has been a surge in popularity among audiologists because of the availability of interactive computer-based training programs. The ability to provide a therapeutic AT option that could be offered either in the clinical setting or in a home-based setting has spurred the growth of this intervention. Sweetow and Palmer (2005) performed a systematic review of research of individual AT in adults and although the results were mixed, they found no deleterious results of the training. As part of their discussion, they believed AT might be best accomplished through interactive computer-based programs as it was believed that individual adult intervention was time consuming and not cost efficient. More recently, Chisolm and Arnold (2012) performed an additional systematic review of research on individual AT and determined that evidence exists that AT results in at least short-term improvement of speech understanding.

What Is AT?

Interpretation of auditory input requires the use of multiple processes beyond hearing. Cognitive functions, pragmatics, language mastery, and visual perception are only a few of the components of auditory processing of information. Degradation of auditory input from hearing loss significantly impacts many of the processes used. With this in mind, AT is meant to help persons with hearing loss improve their ability to interpret, process, and assimilate auditory input. Sweetow and Sabes (2014) described AT as "a process designed to enhance the ability to interpret auditory experiences by maximally utilizing residual hearing" (p 277). Therefore, AT therapeutic intervention is intended to foster awareness of sound cues available to the listener (Carhart, 1960).

Intervention can be provided either individually or in group settings. Although group settings are valuable and desired to foster socialization and improved communication strategies and behaviors, it can sometimes be difficult to meet the individual AT needs of the group members. For this reason, AT intervention is best served on a one-to-one basis with programs designed with individual requirements considered. Speech perception testing (see Chapter 5 for information) can be performed to determine a baseline level of functioning and the training programs can be designed from the results obtained.

Training Considerations

Traditional approaches to AT are described as either analytic or synthetic. The use of analytic training requires the clinician to focus therapy on the fundamental aspects of speech, breaking it down to its component parts. This intervention focuses on vowel and consonant recognition through the use of phonemes or phoneme clusters.

Synthetic training, on the other hand, emphasizes listening skills through the use of sentence materials that incorporate the inclusion of auditory and linguistic skills for interpretation of the message (Sweetow and Sabes, 2014).

Erber (1982) described a framework for AT that included four levels of speech skills that are essential elements of communication: Detection, discrimination, identification, and comprehension. Speech detection or awareness simply refers to the ability to recognize that sound is either present or absent and is the basis for more advanced levels of speech understanding. Discrimination is the ability to distinguish one sound or groups of sounds from another. It implies that the person is able to detect differences in acoustic characteristics such as intensity, duration, and rate. The next level of speech understanding is identification. Here, the listener is capable of applying labels to the sounds heard. That is, the ability to correctly identify the acoustic input. Finally, once the speech has been identified, the final level is comprehension. Here, the listener understands and assigns meaning to the perceived speech signal. Comprehension is, of course, the necessary requirement for communication. It is the result of detection, discrimination, and identification.

AT traditionally progresses from easy speech perception tasks to more complex stimuli. Treatment follows a hierarchy of the levels of speech perception, progressing from gross to fine sound discrimination, and both analytic and synthetic approaches are used to achieve this progression. In analytic training, differences between voicing characteristics, place, and manner of articulation may be the focus. These same concepts can then be reinforced through the use of sentence material.

Technology and AT

Computer-based AT programs have flourished in recent years. As a result, audiologists have increasingly begun recommending AT to their patients. Sweetow and Sabes (2007) likened the need for AT to physical or occupational therapy when an individual suffers a leg or arm injury. These therapies are widely accepted and expected as part of the treatment for these conditions. The availability of home-based training programs makes it more accessible for many.

There are a number of computer-based programs available. Sweetow and Sabes (2014) describe these programs as typically automated and adaptive. They are able to monitor use and skill level, and the programs tend to increase in difficulty as the user progresses. Scores are maintained and in some cases can be monitored from the audiologist's office. The exercises contained in many of these programs consist of auditory/visual and cognitive training, as well as repair and communication strategies.

Two programs in particular have generated a great deal of interest, Listening and Communication Enhancement (LACE) (Sweetow and Sabes, 2006) and Read My Quips (Levitt et al., 2011). LACE, in particular, is responsible for stimulating a resurging interest in AT and is the most widely used computer-based program.

LACE is designed to offer adults with hearing loss exercises meant to enhance listening skills and improve communication. The program provides activities to address listening in adverse conditions and some of the cognitive changes that impact aging adults. Training is offered in 20, half-hour sessions and a record of skill is recorded and plotted for easy interpretation. LACE is divided into three main sections: Degraded speech, cognitive training, and communication strategies.

Degraded speech exercises consist of situations where an adult with hearing loss would experience listening difficulties such as speech in noise, rapid speech, and competing speakers. Cognitive training includes processing speed and auditory memory. Finally, communication strategies are provided in areas such as environmental modifications, expectations, and effective communication behaviors.

Read My Quips (Levitt et al., 2011; Sensesynergy, 2011) is designed as an entertaining adaptive speech in noise program. The authors believed that for training to be effective it should be fun. With that in mind, the program presents a series of crossword puzzles consisting of amusing quips (i.e., "Can an atheist get insurance against acts of god?"). It uses an auditory/visual format with cues offered under varying signal to noise ratios.

Many of the available AT computer-based programs combine both auditory and visual stimuli in their exercises. The use of both modalities in communication enhances the perception of auditory input and is consistent with the natural methods used to process speech.

Visual Speech Perception/ Speechreading

There are numerous influences that impact our ability to perceive spoken language. Most communication interactions include both auditory and nonauditory information used concomitantly to receive and decipher verbal input. For a message to be perceived, the listener uses expertise garnered from audition and vision, environmental perception, contextual and facial cues, nonverbal communication, gestures, and language competency. Often, interpretation of the spoken signal is degraded because of factors such as environmental conditions (noise, competing speech, reverberation, lighting, and distance from the source), speaker characteristics (foreign accent, facial hair, rate of speech, loudness of speech, and vocal characteristics), and hearing loss. To maximize the ability to perceive spoken language, the listener uses information received from a variety of input channels with speechreading becoming a compliment to hearing.

The terms speechreading and lipreading are frequently used interchangeably to describe visual speech perception. Although similar, they imply different aspects of this process with lipreading referring to the perception of more specific articulatory movements and speechreading being more inclusive accounting for information provided through lipreading along with gestures, facial expression, and environmental influences (Lansing, 2014).

Visual speech perception, although an important component of the multisensory process we call communication, is threatened by various factors when it is the sole source of spoken language. See Table 45.4 for some of the limitations associated with visual speech perception and the understanding of spoken language including issues related to the visual signal itself, language, environmental factors, and communication styles.

Visual signal refers to the specific characteristics of speech including visibility, place, and manner of articulation. Much of what is spoken cannot be interpreted by vision alone. Many sounds are produced within the mouth with no external visual characteristics. In addition, many sounds that are visible appear identical to the receiver. For instance, place of articulation produces confusions when presented in the visual condition alone. The allophones /p, b, m/ appear the same on the lips and would only be distinguishable through sentence or word content. Consider the sentence "He will hit the ball with the bat." Although the word "bat" may have the same appearance on the lips as "mat" and "pat," the context of the sentence provides the necessary information for interpretation. Sounds and words that look alike are referred to as homophenes and said to be homophenous (Jeffers and Barley, 1971). The use of /p, b, m/ is an example of homophenous sounds and "pat, mat, and bat" are homophenous words. The category when viewed together as a group of sounds is a viseme referring to the units of speech that are indistinguishable from each other (Fisher, 1968).

Visemes are classified by the visibility of speech characteristics. Binnie et al. (1974) classified visemes into five distinct homophenous categories: Bilabials (/p, b, m/),

TABLE 45.4

Limitations of Visual Speech Perception on Understanding Spoken Language

The visual signal	Much of speech cannot be visually observed, many phonemes have identical physical appearances, and perception is dependent on voicing, place, and manner of articulation
Language competency	Spoken language is impacted by pragmatics, syntax, semantics, and phonological processes. Interpretation of the input signal is related to language potential
Environmental conditions	The setting for communication impacts perception: Lighting, distance from the speaker, room decor, auditory and visual distractions
Communication styles	Characteristics of the speaker and proficiency of the listener influence perception: Patterns of articulation, presence of facial hair, cultural influences, speechreading ability

Adapted from Lansing C. (2014) Visual speech perception in spoken language understanding. In: Montano J, Spitzer J, eds. *Adult Audiologic Rehabilitation*. 2nd ed. San Diego, CA: Plural Publishing; pp 253–276.

labiodentals (/f, v/), interdentals (/θ, ð/), rounded labials (/ʃ, ʒ/), and linguals (/t, d, n, s, z, k, g/). Given that the high percentage of sounds do not possess externally visible characteristics along with the redundancy of those that do have visibility, it is understandable why visual information alone may not suffice to provide adequate communication.

In addition to limitations imposed by the speech stimuli themselves, visual perception is also impacted by linguistic or language processes. As shown in Table 45.4, the listener's ability to process and interpret language has an impact on their ability to perceive the spoken message. There are many linguistic constraints that can occur in the communication of a message that have their bases in the listener's language proficiency. Areas such as pragmatics, lexicon, syntax, and phonologic processes are relevant to speech perception and will influence a listener's ability to interpret auditory input.

Environmental influences impact visual speech perception in a number of ways. Conditions such as lighting, distance from the speaker, angles, room clutter, and auditory distractions all can impact the transmission and reception of a speech signal. Sanders (1993) reported that a 5-foot distance between the speaker and listener is optimal for speech perception and Johnson and Snell (1986) identified a minimum visual acuity of 20/30 in at least one eye necessary for successful speechreading ability.

Visual Perception Training

Speechreading assessment and training has been the focus of much research throughout the decades with very little consensus on standardized procedures. There are a limited number of available speechreading tests that have gone through the appropriate standardization procedures, many of which were developed decades ago. There is a need in AR, to develop normative data on speechreading abilities and create standardized tests to allow accurate skill measurement. Although systematic reviews for AR have been performed with specific areas covered such as AT and group intervention, there is no current publication available to look specifically at speechreading. Binnie (1977) measured attitudes about communication following speechreading training. He reported that although pre- and postmeasurements of speechreading ability did not yield any statistically significant improvements, there was a measurable positive improvement with subjects' confidence in communication.

A consideration faced by clinicians when they are deciding on speechreading activities is whether materials used should be presented in the vision alone or vision and audition condition. Certainly, vision alone would be most challenging and may actually be a source of frustration for many patients. Alpiner and Schow (2000) recommended a bisensory approach to speechreading assessment and training. The combination of both vision and audition more accurately represents the patient's true mode of communication and would be useful for planning treatment and monitoring progress. The use of a bisensory approach implies that materials can be presented using both modalities in quiet and in simulated conditions of background noise.

As was the case with AT, speechreading therapy can be provided using either an analytic, synthetic, or a combination of approaches. Analytic speechreading would focus on identifying the visual components of viseme speech groups starting with very visible phonemes like the bilabials and progressing to the more difficult to identify such as the linguals. The training would focus exclusively on the visual characteristics of the phonemes. Synthetic training would use this same strategy, more visible to less visible, but present material in sentences or continuous discourse looking to encourage an understanding of meaning and not perhaps every sound segment.

DeFilippo and Scott (1978) first introduced the idea of continuous discourse tracking as a method to evaluate and train speech perception. The method rapidly gained wide acceptance largely as it appeared at a time when cochlear implant technology was being introduced for clinical use. Tracking became the preferred method of training for cochlear implant recipients and was quickly assimilated into AR programs throughout the world. According to the authors, "A talker and receiver engage in a dialogue for a designated period of time in which the receiver reports his perception of successive segments of read text and is corrected by the talker until the text is verbatim. Performance is measured in number of words or text repeated correctly per unit of time" (p 1186).

The method is particularly useful as it can be used in a variety of presentation conditions: Visual alone, vision and audition, amplification alone, cochlear implant alone, and so on. Typically, the presenter chooses the discourse material based on the interests and language skill levels of the patient. A series of strategies are reviewed and the discourse is read in short segments frequently for a period of 10 minutes. At the end of the time period, the total number of words, correctly identified, is divided by the number of minutes of the exercise yielding a word per minute score. This score is then plotted on a graph providing the patient with a visual representation of the progress he/she is making in treatment with a comparison between performance with and without amplification. Figure 45.2 represents the outcome of tracking for a patient wearing binaural amplification. Note the steady increase in the word per minute scores across treatment sessions with performance being the best in the aided visual condition.

Computer-Based Speechreading Training

The use of computer-based training programs has become increasingly popular in recent years. As was the case with the programs described in the AT section of this chapter, most available programs combine audition and vision in their

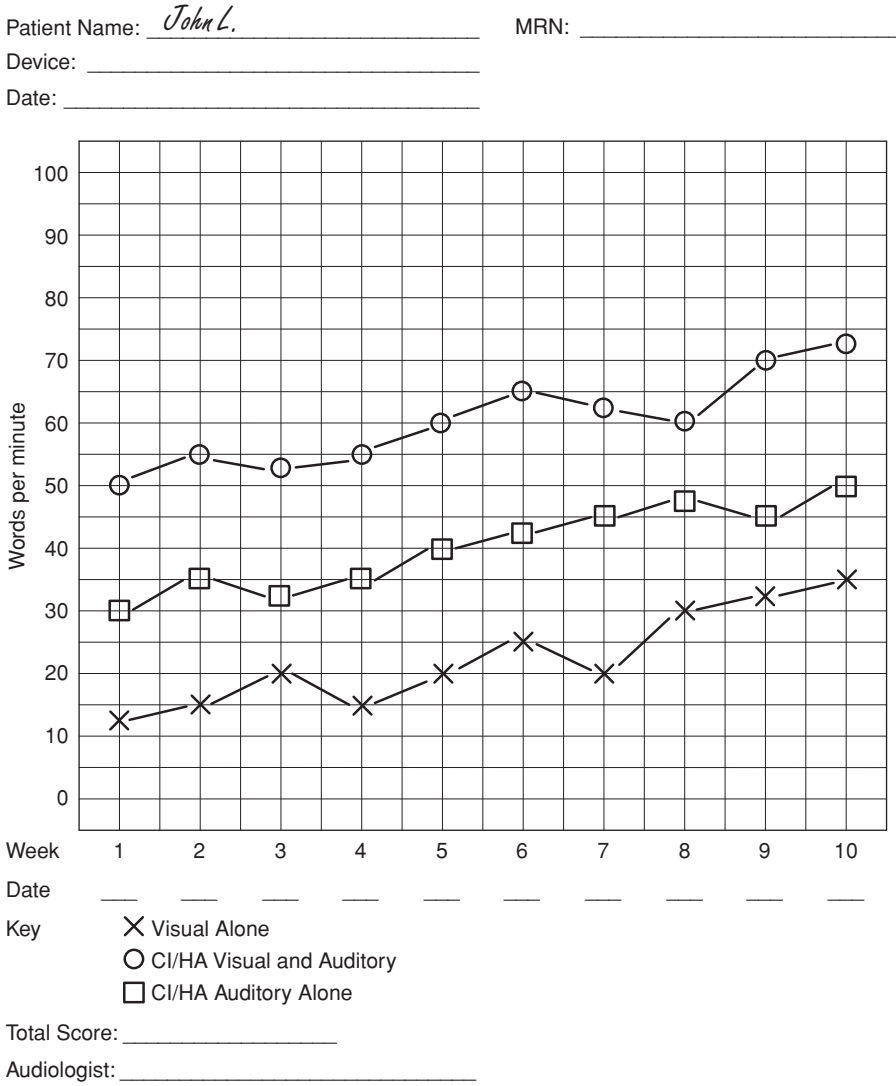


FIGURE 45.2 Speech tracking progress chart.

training. A popular program, Seeing and Hearing Speech (Sensimetrics Corporation, 2008) is an interactive program that allows users to proceed at their own pace with exercises focused on areas such as word stress, hearing in noise, and consonant confusions. Users have the option of practice in either visual alone or vision/audition conditions. Materials can be presented at varying rates and results are plotted along a continuum for easy interpretation. Included in the program are tips for successful communication.

Conversation Made Easy (Tye-Murray, 2002) is another interactive speechreading program available through the Central Institute for the Deaf. It consists of three modules for training adults and teenagers focusing on communication repair strategies. As with Seeing and Hearing Speech users can decide on the modality for presentation: Vision, audition, or vision/audition. These programs along with the ones described in the AT section provide opportunities for self-study of speechreading and AT. Although these programs can be effective, they are somewhat isolating as they are performed alone in one's home environment. To foster improved communication, group intervention may allow for increased socialization and reinforcement of the skills that were developed through either individual therapy or at-home computer-based intervention.

Group Treatment

There is an abundance of research on the value of group aural/audiologic rehabilitation (GAR), with results suggesting positive outcomes. Certainly, the idea that treatment be provided for people with hearing loss in a group setting makes sense. GAR, for individuals with communication challenges and their communication partners, can be valuable for exchanging information and ideas about living with hearing loss. One of the benefits of GAR is the ability for the audiologist to actively address the psychosocial effects of hearing loss (Preminger and Nesbitt, 2014). Trychin (2009) refers to hearing loss as a communication loss that

has an impact not only on the patients but also on the people around them. The group setting creates a safe haven for communication with partners who possess similar traits. It provides the participants an opportunity to not only share experiences, and learn from others, but also provide support and counsel, and know they are not alone in their experiences.

Systematic reviews of the literature seem to support the inclusion of group treatment in AR programs. Findings reported by Hawkins (2005) supported GAR highlighting achieved benefits that included reduction in participation restrictions, that is interactions on the social level, improvement in the use of amplification, improved communication strategies, and improvements in personal adjustment. Chisolm and Arnold (2012) identified a series of studies that demonstrated consistent short-term and some long-term benefits of GAR interventions (Chisolm et al., 2004; Hickson et al., 2007; Preminger and Yoo, 2010).

Although evidence exists that GAR can be an effective component of AR, few audiologists offer such services. This may be because they are unsure of how to proceed or uncomfortable with the notion of running a group. To address this deficiency, faculty and staff of the Ida Institute (2012) developed a web-based tool designed to encourage the inclusion of GAR in hearing healthcare settings including hospitals, speech and hearing centers, community centers, and private practice (Montano et al., 2013). The tool, referred to as the Group Rehabilitation on-Line Utility Pack (GROUP), is available at their website (www.idainstitute.com) following the link to the "Tool Room." Once accessed, the audiologist is provided with an online guide that can assist him/her in developing, marketing, and implementing GAR programs in their work facility. The GROUP is broken into several specific areas, including a thorough bibliography and resource materials for the participant. The resource library provides information on marketing, establishing groups, activities that can be used in the session, and information on reimbursement for services. Perhaps one of the most informative aspects in this section is the ability for the participant to view actual videos with the authors demonstrating GAR in action. Trychin (2014) provides specific suggestions for developing and implementing GAR programs with suggestions on specific activities that can be used in treatment, and they are demonstrated and available in the resource library of the program.

GAR programs can be developed and run in many different ways. They might be therapeutic and provide the opportunity for the audiologist to provide information on treatment options, hearing aid troubleshooting, speechreading, and so on. The groups can be primarily for socialization, giving people the opportunity to practice the skills they have developed to enhance communication with hearing loss. Referral to consumer support organizations, such as the Hearing Loss Association of America, can meet some of the socialization and information needs of patients. This is especially true when GAR programs are not offered in the community. Regardless, the opportunity to meet and socialize with others with similar conditions is rationale enough for referral to local support groups.

SUMMARY AND FUTURE CONSIDERATIONS

The purpose of this chapter was to provide the reader with a basic foundation on services available for the provision of treatment to help patients manage their hearing loss. It was presented in the context of patient-centered care, that is, the recommendations and treatment plans are developed using a shared decision process that includes the cooperative efforts of both the patient and the audiologist. In this way the AR programs can be unique to the people they are established for. The audiologist acts as a guide and jointly helps patients explore the options available to them. In some cases it might be the need for and use of hearing aids and assistive listening devices, and for others perhaps instruction on communications strategy techniques. Other patients might need individualized treatment including AT and speechreading.

Regardless of the direction the AR program takes, the audiologist accompanies the patient on his/her continuing journey of learning to live with hearing loss and developing the skills necessary to maximize abilities. The foundation of such a program is counseling. The audiologists need to embrace the concept that they are indeed the most qualified to assist patients with hearing loss and explore the options available to them. The provision of information and support is the basis for this counseling relationship. The partnership that develops between the audiologist and the patient with hearing loss will be at the core of successful AR programming. Embracing the concept of the biopsychosocial model of service delivery will ensure that AR remains a vital part of the audiologist's identity in the new and ever-changing healthcare arena.

Understanding the need and importance of providing AR does not ensure the availability of these services for people with hearing loss. There have been obstacles to AR implementation that impede its inclusion in many programs. One in particular in the United States is the lack of reimbursement for these services. At the time of this writing, ASHA has spearheaded efforts to introduce legislation that would support audiologist reimbursement for comprehensive audiology care. This would change its position from a specific diagnostic professional classification to one that is inclusive, with rehabilitative services.

Future trends in health care will demand a more patient-centered provision of service. The sale of hearing aids is quickly being engulfed by outside entities such as insurers, big box stores, and physicians. If audiology is to thrive it should return to its roots and embrace the rehabilitative foundations of our profession. There is nothing I

can think of that would align itself better with a patient-centered philosophy and the future trend in health care than the inclusion of rehabilitation services by audiologists. AR should be the centerpiece of services provided for adults with hearing loss.

REFERENCES

Alpiner J, Schow R. (2000) Rehabilitative evaluation of hearing impaired adults. In: Alpiner J, McCarthy P, eds. *Rehabilitative Audiology Children and Adults.* Baltimore: Lippincott Williams and Wilkins; pp 305–331.

American Speech-Language Hearing Association (ASHA). (2001) Knowledge and skills required for the practice of audiologic/aural rehabilitation. In: *ASHA Desk Reference.* Vol. 4. Rockville, MD: ASHA.

Binnie C. (1977) Attitude changes following speechreading training. *Scand Audiol.* 6 (1), 13–19.

Binnie C, Montogomery A, Jackson P. (1974) Auditory and visual contributions to the perception of consonants. *J Speech Hear Res.* 17, 619–630.

Carhart R. (1960) Auditory training. In: Davis H, Silverman R, eds. *Hearing and Deafness.* 2nd ed. New York: Holt Rinehart and Winston; pp 346–359.

Chisolm T, Arnold M. (2012) Evidence about the effectiveness of aural rehabilitation programs for adults. In: Wong L, Hickson L, eds. *Evidence-Based Practice in Audiology.* San Diego, CA: Plural Publishing; pp 235–266.

Chisolm TH, Abrams HB, McArdle R. (2004) Short- and long-term outcomes of adult audiological rehabilitation. *Ear Hear.* 25 (5), 464–477.

Clark JG, English K. (2014) *Counseling-Infused Audiologic Care.* Boston: Pearson.

DeFilippo CL, Scott BL. (1978) A method for training and evaluating the reception of ongoing speech. *J Acoust Soc Am.* 63, 1186–1192.

Erber NP. (1982) *Auditory Training.* Washington, DC: Alexander Graham Bell Association for the Deaf.

Erdman SA. (2000) Counseling adults with hearing impairment. In: Alpiner J, McCarthy P, eds. *Rehabilitative Audiology: Children & Adults.* 3rd ed. Baltimore: Lippincott Williams and Wilkins.

Erdman SA. (2014) The biopsychosocial approach in patient and relationship-centered care: implications for audiologic counseling. In: Montano J, Spitzer J, eds. *Adult Audiologic Rehabilitation.* 2nd ed. San Diego, CA: Plural Publishing; pp 159–207.

Erdman SA, Wark D, Montano JJ. (1994) Implications of service delivery models in audiology. *J Acad Rehabil Audiol.* 27, 45–60.

Fisher CG. (1968) Confusions among visually perceived consonants. *J Speech Hear Res.* 11, 796–804.

Goldstein D, Stephens D. (1981) Audiologic rehabilitation: management model I. *Audiology.* 20, 432–452.

Gregory M. (2012) A possible patient journey: a tool to facilitate patient centered care. *Semin Hear.* 33, 9–15.

Hawkins DB. (2005) Effectiveness of counseling-based adult group aural rehabilitation programs: a systematic review of the evidence. *J Am Acad Audiol.* 16, 485–493.

Hickson L, Worrall L, Scarinci N. (2007) A randomized controlled trial evaluating the active communication education program for older people with hearing impairment. *Ear Hear.* 28, 212–230.

Ida Institute. (2011) Patient motivation. Available online at: www.idainstitute.com/tool_room/ (accessed September 24, 2013).

Ida Institute. (2012) Group Rehabilitation On-line Utility Pack (G.R.O.U.P.). Available online at: www.idainstitute.com/group (accessed September 18, 2013).

Institute of Medicine. (2001) *Crossing the Quality Chasm: A New Health System for the 21st Century.* Washington, DC: National Academy Press.

Jeffers J, Barley M. (1971) *Speechreading (Lipreading).* Springfield, IL: Thomas.

Johnson D, Snell KB. (1986) Effects of distance visual acuity problems on the speechreading performance of hearing-impaired adults. *J Acad Rehabil Audiol.* 19, 42–55.

Jorgensen SV, Hansen HV, Hessov IB, Lauritsen JB, Madelung S, Tonnesen H. (2003) *Operation-Complications Are Preventable.* Copenhagen: Copenhagen International Health Promoting Hospitals and Health Services, Bispebjerg Hospital.

Kricos P, McCarthy P. (2007) From ear to there: a historical perspective on auditory training. *Semin Hear.* 28, 89–98.

Lansing C. (2014) Visual speech perception in spoken language understanding. In: Montano J, Spitzer J, eds. *Adult Audiologic Rehabilitation.* 2nd ed. San Diego, CA: Plural Publishing; pp 253–276.

Levitt H, Oden C, Simon H, Noack C, Lotze A. (2011) Entertainment overcomes barriers of auditory training. *Hear J.* 64, 40–42.

Luterman D. (2001) *Counseling Persons with Communication Disorders and Their Families.* 4th ed. Austin, TX: ProEd.

Manchaiah VK, Stephens D. (2011) The patient journey: living with hearing impairment. *J Acad Rehabil Audiol.* 44, 29–40.

Montano JJ. (2011) Building relationships: an important component to the aural rehabilitation process. *ENT Audiol News.* 20 (4), 91–92.

Montano JJ. (2014) Defining audiologic rehabilitation. In: Montano J, Spitzer J, eds. *Adult Audiologic Rehabilitation.* 2nd ed. San Diego, CA: Plural Publishing; pp 23–34.

Montano J, Preminger J, Hickson L, Gregory M. (2013) A new web-based tool for group audiologic rehabilitation. *Am J Audiol.* [Epub ahead of print].

Montgomery AA, Jackson PL. (1983) Physical characteristics of the lips underlying vowel lipreading performance. *J Acoust Soc Am.* 73 (6), 2134–2144.

Patient Protection and Affordable Care Act. (2010) Pub. L. No. 111-148, 124 Stat. 119-1025.

Preminger J, Nesbitt L. (2014) Group audiologic rehabilitation for adults: justification and implementation. In: Montano J, Spitzer J. eds. *Adult Audiologic Rehabilitation.* 2nd ed. San Diego, CA: Plural Publishing; pp 307–328.

Preminger J, Yoo J. (2010) Do group audiological rehabilitation activities influence psychosocial outcomes? *Am J Audiol.* 19, 109–125.

Prochaska J, DiClemente C. (1983) Stages and processes of self-change of smoking: towards an integrated model of change. *J Consult Clin Psychol.* 51, 390–395.

Ross M. (1997) A retrospective look at the future of aural rehabilitation. *J Acad Rehabil Audiol.* 30, 11–28.

Sanders DA. (1993) *Aural Rehabilitation.* 3rd ed. Englewood Cliffs, NJ: Prentice-Hall.

Sensesynergy. (2011) Read my quips. Available online at: www.sensesynergy.com (accessed September 24, 2013).

Sensimetrics Corporation. (2008) *Seeing and Hearing Speech: Lessons in Lipreading and Listening.* Somerville, MA: Sensimetrics.

Sweetow R, Palmer CV. (2005) Efficacy of individual auditory training in adults: a systematic review of the evidence. *J Am Acad Audiol.* 16, 494–504.

Sweetow RW, Sabes JH. (2006) The need for and development of an adaptive Listening and Communication Enhancement (LACE™) Program. *J Am Acad Audiol.* 17 (8), 538–558.

Sweetow RW, Sabes JH. (2007) Listening and communication enhancement (LACE). *Semin Hear.* 28, 133–141.

Sweetow RW, Sabes JH. (2014) Auditory training. In: Montano J Spitzer J, eds. *Adult Audiologic Rehabilitation.* 2nd ed. San Diego, CA: Plural Publishing; pp 277–290.

Trychin S. (2009) *Enabling communication partnerships.* Paper presented at Ida Institute Seminar 3A, Skodsborg, Denmark.

Trychin S. (2014) Peer support/consumer perspective. In: Montano J, Spitzer J, eds. *Adult Audiologic Rehabilitation.* 2nd ed. San Diego, CA: Plural Publishing; pp 378–398.

Tye-Murray N. (2002) *Conversation Made Easy: Speechreading and Conservation Training for Individuals Who Have Hearing Loss (Adults and Teenagers).* St. Louis, MO: Central Institute of the Deaf.

World Health Organization. (1980) *International Classification of Impairment Disability and Handicap.* Geneva: World Health Organization.

World Health Organization. (2001) *International Classification of Functioning.* Geneva: World Health Organization.

CHAPTER 46

Infection Control

A.U. Bankaitis

INTRODUCTION

Acquired immunodeficiency syndrome (AIDS) and subsequent discovery of the human immunodeficiency virus (HIV) during the 1980s moved infection control to the forefront in the healthcare community. The concern for cross-contamination associated with HIV resulted in the issuance of federally mandated infection control requirements, providing the healthcare industry with specific guidelines on how to minimize the risk of exposure to potentially infectious agents. Since that time, the scope of infection control has expanded well beyond blood-borne diseases to include all microorganisms associated with potential degree of disease transmission. Although implementing an infection control plan is a relatively straightforward endeavor, appreciating its relevance to audiology along with understanding general principles and required components is necessary to ensure proper application and execution of such principles.

INFECTION CONTROL

Infection control refers to the conscious management of the environment for the purposes of minimizing or eliminating the potential spread of disease (Bankaitis and Kemp, 2003, 2005). Although the discovery of HIV impacted infection control in all areas of health care, it is not limited to minimizing the spread of HIV; rather, infection control programs are designed to minimize the spread of disease from any number of ubiquitous microorganisms readily found throughout the clinical environment (e.g., *Staphylococcus, Pseudomonas*) regardless of how remote the possibility of disease transmission may be perceived. The mindset of an effective infection control program is based on the universal assumption that every patient, bodily fluid, substance, or agent is potentially infectious. As such, diagnostic and rehabilitative services provided by audiologists must be delivered in a manner consistent with infection control guideline requirements and applied uniformly across each and every patient.

RELEVANCE OF INFECTION CONTROL TO AUDIOLOGY

As primary healthcare providers for hearing and vestibular disorders, audiologists have always been expected to practice basic hygiene procedures in the form of hand washing and general housekeeping; however, infection control must extend well beyond these general practices for several reasons. Table 46.1 provides a list of reasons why infection control is relevant to audiology. First, a number of governing bodies have issued federal mandates related to infection control making it a legal responsibility for audiologists to implement required infection control elements as specifically outlined throughout established guidelines within the clinical environment. The Occupational Safety and Health Administration (OSHA) is the federal agency governed by the United States Department of Labor that is responsible for regulating the workplace to ensure safety and healthful working environments. OSHA oversees and enforces infection control programs throughout environments where patient care services are provided to ensure compliance with federal standards as well as those guidelines outlined in the Universal (Standard) Precautions issued by the Centers for Disease Control and Prevention (CDC). In other words,

TABLE 46.1

Relevance of Infection Control to Audiology

Federal mandates issued by OSHA applicable to and required of all healthcare providers
Nature of audiologic practice inherently increases potential for disease transmission
Scope of audiology practices increases chances of exposure to bodily fluids including cerumen, ear drainage, pus, mucous, and/or blood
Audiologic services sought by wide range of immunocompromised individuals susceptible to opportunistic infections
Microbial contamination of hearing instruments

infection control is the law; failure of compliance results in citations and fines.

Beyond legal requirements, managing patients with hearing and/or vestibular disorders involves a notable degree of patient contact, including the use of various reusable objects that come in direct and indirect contact with multiple patients. From this perspective, the very nature in which audiologic services are delivered inherently increases the potential for disease transmission, and appropriate measures must be taken to eliminate cross-contamination. For example, contact transmission represents the most common means of disease transmission in clinical environments where these types of services are provided (Bankaitis and Kemp, 2003, 2005, 2007; Bankaitis et al., 2005). Directly touching a patient's draining ear without the use of gloves and reusing an object such as an immittance probe tip, otoscope specula, or the bell portion of a hearing aid listening stethoscope whereby patients are indirectly exposed to contamination are examples of how contact transmission may occur in the audiology clinic. Furthermore, audiologists often touch and manipulate the ear and/or make direct or indirect contact with a patient's skin, natural orifices of the body that serve as portals of entry for microorganisms. To eliminate or minimize the potential spread of disease in the clinical environment, it is paramount for audiologist to deliver clinical procedures in a manner specifically designed to eliminate potential microbial transmission via direct and indirect contact as well as prevent the very same microorganisms to gain access to the human body via natural body orifices.

Audiology's scope of clinical practice is vast and diverse, involving many types of noninvasive and invasive patient contacts that potentially expose the clinician to bodily fluids to which appropriate barriers and infection control procedures must be used. For example, intraoperative monitoring, cerumen management, vestibular testing procedures that may result in patients getting sick, postoperative audiologic assessment of cochlear implant and middle ear implant recipients, and patients with healing surgical wounds can easily expose the clinician to cerumen, ear drainage, mucous, blood, and the like. Over the past several decades, as the scope of audiology practice has expanded, the incidence of exposure to blood and other bodily fluids and the subsequent risk of exposure to blood-borne pathogens substantially increase (Kemp and Bankaitis, 2000). The relevance of infection control in the clinic where audiology services are provided cannot be overstated.

Furthermore, audiology services are sought by a wide range of patients who vary across factors known to potentially comprise the integrity of the immune system. Although not an exhaustive list, a patient's age, underlying disease state (i.e., diabetes, cancer), nutritional status, socioeconomic background, and/or exposure to past and current pharmacologic interventions will influence how well a patient's body can fight off disease (Bankaitis and Kemp, 2003, 2005). Varying degrees of immunocompromise manifest in unpredictable ways that are not necessarily evident or identifiable; although individuals with underlying disease may appear healthy, because their immune system is compromised in some way, they are inherently at greater risk for infection. The hallmark of immunocompromise is the susceptibility of individuals to opportunistic infections and diseased states caused by ubiquitous organisms residing in abundance throughout the environment that rarely cause disease or infection in healthy individuals; rather, these microbes take the opportunity to infect those exhibiting some degree of immunocompromise (Bankaitis and Kemp, 2003, 2005). Given the right conditions, these infections can be life threatening in some cases. For example, the bacterium S*taphylococcus* resides on skin surfaces. Given the universal nature of the bacterium, the assumption by clinicians may be that *Staphylococcus* is an innocuous bacterium for which infection control procedures are not necessary. On the contrary, although this bacterium is ever-present throughout the environment, it accounts for a high percentage of nosocomial or hospital-acquired infections (Murray et al., 1994). Since most hospital patients are sick and exhibit varying degrees of immunocompromise, despite the universal nature of *Staphylococcus,* these patient populations remain extremely susceptible to such microorganisms. From this perspective, audiologists must adhere to a proactive strategy to minimize the possibility of the inadvertent spread of disease in the clinical environment.

Finally, objects coming in direct or indirect contact with patients may be contaminated with potentially infectious microorganisms. Bankaitis (2002) documented the presence of light to heavy amounts of bacterial and/or fungal growth on the surface of hearing instruments removed from the ears of adult patients. The predominant organism recovered was *Staphylococcus;* however, each hearing aid was contaminated by a unique combination of bacterial and/or fungal microbial growth. A follow-up study conducted by Sturgulewski et al. (2006) revealed similar findings. Based on the findings of these studies, it is plausible to assume that other reusable objects used by audiologists with multiple patients may be contaminated with varying degrees of microbial growth. The relevance of infection control from this perspective cannot be overstated.

IMPLEMENTATION OF INFECTION CONTROL PRINCIPLES

In the United States, the CDC issued a number of similar recommendations and guidelines for minimizing cross-infection of blood-borne diseases to healthcare workers. These pronouncements were officially formalized into the Universal Blood and Blood borne Pathogen Precautions (CDC, 1987). The pronouncements were originally intended to protect healthcare workers from blood and blood-borne pathogens; however, these precautions have since been expanded to include all potentially infectious body substances.

TABLE 46.2

Standard Precautions as Issued by the Centers for Disease Control and Prevention

Appropriate personal barriers (gloves, masks, eye protection, and gowns) must be worn when performing procedures that may expose personnel to infectious agents
Hands must be washed before and after every patient contact and after glove removal
Touch and splash surfaces must be precleaned and disinfected
Critical instruments must be sterilized
Infectious waste must be disposed of appropriately

Source: CDC. (1987) Recommendations for prevention of HIV transmission in healthcare settings. *Morb Mortal Wkly Rep*. 36 (suppl 2), 1S–18S.

The five general pronouncements are outlined in Table 46.2. The pronouncements are relatively straightforward; however, each is reviewed in more detail with specific attention placed on its application to clinical management of patients with communication, hearing, and swallowing disorders.

Appropriate Personal Barriers

Appropriate personal barriers refer to gloves, masks, eye protection, and/or gowns which must be worn during the provision of services and/or procedures that may expose audiologists to potentially infectious agents or substances.

Gloves

Appropriately fit gloves are indicated during invasive procedures or those procedures where open wounds and/or visible blood are present. Wearing gloves is indicated when hands are likely to become contaminated with potentially infective material such as blood, body fluids, secretions, excretions, or mucous membranes, as well as in those situations to prevent gross microbial contamination of hands (CDC, 2002; WHO, 2004). Outside of the operating room environment, clean, nonsterile gloves may be used when touching potentially infective material (WHO, 2004). Table 46.3 provides a general guide as to when gloves should be worn by clinicians providing diagnostic or rehabilitative services to individuals with communication, hearing, and swallowing disorders.

The size of the glove is important and should be determined for each clinician separately to ensure an appropriate fit. As shown in Figure 46.1, gloves are considered to fit appropriately when the glove fits the hand tightly, adhering very close to the skin without being too tight. This will allow for effective manipulation of objects, items, or instruments during the provision of services without compromising manual dexterity. Gloves that fit too loosely (Figure 46.2) will hinder

TABLE 46.3

Glove Use Guidelines for Audiologists

In the presence of an open wound and/or visible blood (at the level of patient's ears and/or clinician's fingers, palms, wrists)
When handling hearing instruments or earmolds that have not been cleaned and disinfected first
When removing cured earmold impressions from the ear canal
When cleaning instruments contaminated with cerumen, mucus, or other bodily substances
When submerging or removing reusable instruments into or from a cold sterilant
When hands are likely to become contaminated with potentially infectious material including cerumen, saliva, mucous membranes
In the operating room environment during patient preparation or any other procedures during or after the surgical procedure where hands could potentially come in contact with blood, bodily fluids, or other contaminated materials or contaminated objects

Source: Adapted from Bankaitis AU, Kemp RJ, Krival K, Bandaranayake DW. (2005) *Infection Control for Speech-Language Pathology.* St. Louis, MO: Auban, Inc. and Bankaitis AU, Kemp RJ. (2005) *Infection Control in the Audiology Clinic*. St. Louis, MO: Auban, Inc. Reprinted with permission by Auban, Inc.

the clinician's manual dexterity, creating potential frustration during the execution of procedures and/or increasing the likelihood for an accident to occur that could otherwise be avoided. One-size-fits-all gloves should be avoided since they are not designed to fit most hands appropriately.

Gloves are considered one-time use items and should not be reused. Furthermore, the same pair of gloves should not be used on different patients. After use, gloves should be properly removed and disposed. Unless grossly contaminated

FIGURE 46.1 Appropriately sized glove that fits tightly, adhering closely to the skin. Reprinted with permission by Auban, Inc.

FIGURE 46.2 Loosely fitted gloves inappropriate for audiologic procedures, resulting in loss of manual dexterity and difficulty handling and/or manipulating objects. Reprinted with permission by Auban, Inc.

with blood or other bodily fluids, gloves may be disposed of in the regular trash or according to the protocol dictated by the hearing care facility. It is highly unlikely for standard clinical procedures related to communication, hearing, and swallowing disorders to result in copious amounts of blood or bodily fluid contamination to require arrangements for hazardous waste removal.

Masks, Eye Protection, and Gowns

Disposable masks, safety glasses, and gowns must be worn when there is a risk of splash or splatter of blood, bodily fluids, secretions, or excretions or when the clinician may be at risk of airborne contamination. The protective barriers, masks and eye protection, should be worn when using buffing wheels or drills during hearing aid or earmold modification procedures. Clinicians providing services to hospital patients with tuberculosis (TB) must wear special TB masks when the diagnosed patient has not been on an antibiotic regimen for 10 days.

As with gloves, disposable masks are not reusable and should be disposed of properly. Eye protection may or may not be reusable as dictated by the specific manufacturer's intended design. Disposable eye protection must be disposed of according to the healthcare facility's established protocol. Conversely, reusable eye protection should be cleaned and properly decontaminated according to the manufacturers' instructions (WHO, 2004). Finally, contaminated or soiled gowns should be removed as soon as possible with disposable gowns being discarded appropriately; reusable gowns necessitating laundering must be routed to the appropriate laundering facility.

Hand Hygiene

Hand hygiene represents the single most important procedure for effectively limiting the spread of infectious disease (Bankaitis and Kemp, 2003, 2005). It is one of the most critical components of a basic infection control program. Hand hygiene involves the process of washing hands with soap and water or using antimicrobial "no-rinse" hand degermers. Traditional hand washing involves the use of hospital-grade, liquid soap. Hospital-grade soap is gentler than household soaps and contains special emollients that moisturize the skin and are effective in reducing or minimizing chapping, chafing, or drying of the skin from excessive hand washing (Bankaitis and Kemp, 2003, 2005). Antimicrobial "no-rinse" products refer to the alcohol-based hand rubs that do not require the use of or access to running water. The availability and accepted use of alcohol-based, no-rinse hand degermers have led to a substantial increase in hand hygiene compliance among healthcare workers. The CDC specifies that hand hygiene procedures must occur prior to the initiation of invasive procedures, before providing services to patient, and after glove removal, and any time that the professional feels it is warranted (CDC, 2002). Table 46.4 provides a guideline as to when hand hygiene should be performed.

TABLE 46.4

Hand Hygiene Guidelines for Audiologists

Prior to initial contact with patient, at the beginning of the patient appointment
At the end of patient contact
After glove use, immediately after removing gloves
Before eating, drinking, smoking, applying lotion or makeup
After eating, drinking, smoking, applying lotion or makeup
After using bathroom facilities
Any time it is felt necessary and appropriate

Source: Adapted from Bankaitis AU, Kemp RJ. (2005) *Infection Control in the Audiology Clinic.* St. Louis, MO: Auban, Inc. and Bankaitis AU, Kemp RJ, Krival K, Bandaranayake DW. (2005) *Infection Control for Speech-Language Pathology*. St. Louis, MO: Auban, Inc. Reprinted with permission from Auban, Inc.

Cleaning and Disinfecting

As outlined in Table 46.5, cleaning refers to procedures in which gross contamination is removed from surfaces or objects without killing germs (Bankaitis and Kemp, 2003, 2005). It does not necessarily involve any degree of germ killing; rather, it serves as an important precursor to disinfecting. Cleaning must occur prior to disinfection; the absence of precleaning a surface will diminish the effectiveness of disinfecting techniques (Kemp and Bankaitis, 2000). In contrast, disinfection refers to a process in which germs are killed. The degree of disinfection that can occur expands across a fairly wide continuum and depends on the specific type and number of microorganisms a product kills.

TABLE 46.5

Definition of Infection Control Terms

Cleaning	Removal of gross contamination without necessarily killing germs
Disinfecting	Process in which germs are killed
Sterilization	Process in which 100% of germs are killed, including associated endospores

Both touch and splash surfaces must be cleaned and then disinfected between patient appointments. Surfaces that come in regular direct or indirect contact with patients and/or clinicians are referred to as touch surfaces. Following each patient appointment and before commencing with the next appointment, countertops, tables, service areas, and the armrest of chairs must be cleaned and disinfected. Splash surfaces are essentially the same thing but involve surfaces that have been contaminated by particles expelled by a patient or clinician, such as when a patient coughs, sneezes, or drools on a surface. As with touch surfaces, splash surfaces must also be cleaned and disinfected after each patient appointment.

Critical Instruments and Sterilization

Critical instruments refer to instruments or objects that meet at least one of the following three criteria: (1) Reusable item introduced directly into the bloodstream (e.g., needles); (2) reusable, noninvasive instrument that comes in contact with intact mucous membranes or bodily substances (e.g., blood, saliva, cerumen, mucous discharge, pus); or (3) a reusable, noninvasive instrument that can potentially penetrate the skin from use or misuse (instruments used for cerumen removal, instruments inserted in the nose, mouth, etc.). Within the context of audiology, reusable items that make contact with mucous membranes, saliva, or cerumen and are intended to be used with multiple patients should be cleaned first and then sterilized. Although not necessarily an exhaustive list, examples of instruments reusable include immittance probe tips contaminated with copious amounts of cerumen and/or drainage and reusable cerumen removal instruments (curettes, hooks, suction tubes).

The term sterilization refers to killing 100% of vegetative microorganisms, including associated endospores (Table 46.5). When microbes are challenged, they revert to the more resistant life form called a spore (Kemp and Bankaitis, 2000). Sterilants, by definition, must neutralize and destroy spores because if the spore is not killed, it may become vegetative again and cause disease. Whereas disinfection involves killing germs, sterilization involves killing all germs and associated endospores each and every time (Bankaitis and Kemp, 2007).

There are several different sterilization techniques including the use of an autoclave or the application of cold sterilization techniques. Since the autoclave involves pressurized heat, most audiologists will be limited to utilizing cold sterilization techniques since reusable rubber, silicone, plastic, or acrylic objects will not withstand traditional heat pressurization sterilization techniques. Cold sterilization involves soaking instruments in liquid chemicals approved by the Environmental Protective Agency (EPA) for a specified number of hours. Only two ingredients have been approved by the EPA as sterilants: (1) Glutaraldehyde and (2) hydrogen peroxide. Products containing the active ingredient glutaraldehyde in concentrations of 2% or higher or those containing the active ingredient hydrogen peroxide (H_2O_2) in concentrations of 7.5% or higher may be used to sterilize instruments.

Reusable items to be sterilized must be cleaned first because organic material (e.g., blood and proteins) may contain high concentrations of microorganisms with chemical germicide properties that can negatively impact the sterilization process. In addition, it is imperative for cold sterilization procedures to be followed according to instructions provided by the product manufacturer. Soaking times necessary to achieve sterilization will differ from solution to solution. Whereas most glutaraldehyde-based products require 10 hours of soaking time to achieve sterilization, hydrogen peroxide products typically require 6 hours of soaking time. Removing instruments or objects prior to the necessary soaking time will result in high-level disinfection and not sterilization. Reviewing product information for instruction of use is critical.

Disposal of Infection Waste

Within the context of audiology, disposable items contaminated with saliva, mucous, discharge, cerumen, blood, or blood by-products may be disposed of in regular waste receptacles; however, in the event the item is contaminated with copious amounts, it should first be placed in a separate, impermeable bas (i.e., bio-hazard bag) and only then discarded in the regular trash (Bankaitis and Kemp, 2003, 2005). This practice will separate the contaminated waste from the rest of the trash and minimize the chance of maintenance or cleaning personnel coming in casual contact with it. Disposing of sharp objects such as razors or needles requires special consideration and must be disposed of in a puncture-resistant, disposable container (sharps container).

WRITTEN INFECTION CONTROL PLAN

Whereas standard precautions serve as the guideline as to how audiologists must modify diagnostic and/or rehabilitative procedures for purposes of minimizing the spread

TABLE 46.6

Required Sections of Written Infection Control Plan as Outlined by OSHA

Employee exposure classification
Hepatitis B (HBV) vaccination plan and records of vaccination
Plan for annual training and records of training
Plan for accidents and accidental exposure follow-up
Implementation protocols
Postexposure plans and records

of disease, the written infection control plan serves as the guiding cornerstone of the specific clinic's global infection control plan, outlining exactly how infection control goals are to be achieved. OSHA requires each facility in the United States to have a written infection control plan and for that plan to be available to all workers. As listed in Table 46.6, the written plan must include specific requirements mandated by OSHA. The following sections review each required element in further detail.

Employee Exposure Classification

Employees must be assigned into one of three different categories according to the potential degree in which a specific employee may be exposed to blood and other infectious substances based on primary work responsibilities. Category one employees include personnel whose primary job assignment exposes them to potential cross-infection with blood-borne diseases or other potentially infectious microbes. This category typically includes physicians, nurses, paramedics, and dentists. Audiologists whose primary job responsibilities include intraoperative monitoring procedures may be categorized as category one employees. Category two employees include personnel whose secondary job assignment potentially exposes them to cross-infection. Most practicing audiologists will fall in this category, including AuD students in training. Finally, employees classified in category three include personnel whose job requirements in the office never expose them to blood or other bodily fluids including administrators and receptionists who do not provide clinical services.

Hepatitis B (HBV) Vaccination Plan and Records of Vaccination

Employers must offer employees in healthcare settings the opportunity to receive a HBV vaccination. The HBV vaccination must be offered to all category one and category two employees free of charge. The employee is not required to accept the offer of vaccination; in the event the employee refuses vaccination, a waiver must be signed noting the refusal of the offered vaccine and filed in the employee records. OSHA requires that this record be retained for length of employment plus 30 years (Kemp et al., 1996).

Plan for Annual Training and Records of Training

OSHA requires a plan for annual training and maintenance of records documenting that such training occurred. Specifically, OSHA has outlined that infection control training must include explanations of symptoms and modes of transmission of blood-borne diseases, location and handling of personal protective equipment, information on the HBV vaccine, and follow-up procedures to be taken in the event of an exposure incident. Although the standard does not specify length of training, infection control training must be provided to new employees at the time of initial assignment and then minimally every year thereafter. Each facility is to conduct and document completion of annual infection control training for each employee. During the course of the year, if an update or new procedure is to be implemented, appropriate training must be conducted in a timely fashion to ensure that the new or updated procedures are understood and implemented. Established employees changing exposure classification categories must undergo infection control retraining within 90 days of the change in classification category. Records of these training sessions are to be filed with the infection control plan in a designated location.

Plan for Accidents and Accidental Exposure Follow-up

The fourth requirement of the written infection control plan involves outlining specific steps to be taken in the event an accident occurs within the clinical environment, which can expose individuals to blood-borne pathogens or other potentially infectious agents, and steps to be taken in the event an employee is accidentally exposed to blood-borne pathogens or other potentially infectious agents. For example, in the event a patient falls and suffers a nosebleed, or becomes sick, every staff member should know what steps to take to address the accident. In addition, accidental exposures to blood-borne pathogens require follow-up. Although these encounters may be relatively rare in audiology environments, an emergency plan must be created and put in place.

Implementation of Protocols

This section of the written infection control plan specifically outlines protocols that dictate how specific procedures will be executed in the clinical environment for purposes of minimizing exposure to potentially infectious

agents. Written procedures that outline how an audiologist is to execute a diagnostic or rehabilitative procedure in a manner designed to minimize or eliminate the likelihood of cross-contamination or exposure to a potentially infectious agent are referred to as a work practice control. Since the extent of services provided by a specific employer will differ from clinic to clinic, the types of work practice controls outlined will differ from clinic to clinic depending on what diagnostic and or rehabilitative services are being provided. For example, a clinic employing audiologists who only provide intraoperative monitoring will maintain different work practice controls within the organization's infection control plan than a public school employing an audiologist.

Furthermore, there exists a certain amount of flexibility as to how a diagnostic procedure can be appropriately executed. Whereas work practice controls are to be developed with the five standard precautions in mind, the degree to which a specific clinic chooses to be more conservative in infection control procedures remains an individual decision made by a particular clinic. For example, one clinic may determine that otoscopy can be performed with ungloved hands following hand hygiene procedures and only in those instances where ear drainage and/or abrasions at the level of the patient's ear and/or clinicians hands are not evident. If ear drainage and/or any abrasions are present, the use of gloves will be mandated. In contrast, another clinic may decide that any procedure requiring direct patient contact, such as otoscopy, will require audiologists to use gloves without exception. Either approach is acceptable and consistent with the goal of a written infection control plan. An inappropriate approach that would involve the development of a work practice control specifically designed, for example, to disallow the use of gloves from the perspective of saving money and reducing overhead costs is not appropriate. Developing audiology-specific work practice controls are beyond the scope of this chapter. For more detailed information on infection control work practice controls for audiology and access to infection control templates, the reader is referred to the book *Infection Control for the Audiology Clinic* by Bankaitis and Kemp (2005).

Postexposure Plans and Records

Finally, the last requirement of an infection control plan as dictated by OSHA involves record keeping of documents related to treatment and subsequent outcomes associated with exposure to potentially infectious pathogens, including HIV.

SUMMARY

Audiologists conduct a variety of diagnostic and rehabilitative procedures that pose a potential risk of exposure to saliva, mucosal secretions, bodily fluids, cerumen, blood, and blood by-products. It is important to recognize the risks associated with exposure to such substances as well as the consequences of cross-contamination to the potential health of both the clinician and the patient. As reiterated throughout this chapter, these risks can be significantly minimized with the implementation and execution of appropriate infection control protocols. The goal of an infection control plan is to consciously manage the clinical environment for the specific purposes of eliminating or minimizing the spread of disease. Producing a written infection control plan is a requirement and associated work practice controls designed to outline how audiologic procedures will be executed must appropriately apply standard precautions.

FOOD FOR THOUGHT

1. I wear gloves whenever I take earmold impressions and remove any wax. Do I really need to use them only once? Isn't this more to protect me than my client? Gloves can be expensive.
2. I wash my hands so often in the clinic that they become cracked and dried out after several clinical days in a row. Do I really need to wash my hands after every client?
3. Instead of washing my hands, can't I just use some form of sanitizing agent? Can I also clean my audiological equipment such as probe tips in this same agent?

REFERENCES

Bankaitis AU. (2002) What's growing on your patients' hearing aids? *Hear J.* 55 (6), 48–56.

Bankaitis AU, Kemp RJ. (2003) *Infection Control in the Hearing Aid Clinic.* St. Louis, MO: Auban, Inc.

Bankaitis AU, Kemp RJ. (2005) *Infection Control in the Audiology Clinic.* St. Louis, MO: Auban, Inc.

Bankaitis AU, Kemp RJ. (2007) Infection control in the audiology clinic. In Campbell K, ed. *Pharmacology and Ototoxicity for Audiologists.* Clifton Park, NY: Thompson Delmar Learning; pp 124–137.

Bankaitis AU, Kemp RJ, Krival K, Bandaranayake DW. (2005) *Infection Control for Speech-Language Pathology.* St. Louis, MO: Auban, Inc.

CDC. (1987) Recommendations for prevention of HIV transmission in healthcare settings. *Morb Mortal Wkly Rep.* 36 (suppl 2), 1S–18S.

CDC. (2002) Guideline for hand hygiene. *Morb Mortal Wkly Rep.* 51 (RR16), 1–44.

Kemp RJ, Bankaitis AU. (2000) Infection control. In: Hosford-Dunn H, Roeser RJ, Valente M, eds. *Audiology: Practice Management.* New York: Thieme Medical Publishers; pp 257–272.

Kemp RJ, Roeser RJ, Pearson DW, Ballachanda BB. (1996) *Infection Control for the Professions of Audiology and Speech Language Pathology.* Olathe, KS: Iles Publications.

Murray PR, Kobayashi GS, Pfaller MA, Rosenthal KS (eds). (1994) Staphylococcus. In: *Medical Microbiology.* 2nd ed. St. Louis, MO: Mosby-Year Book, Inc; pp 166–179.

Sturgulewski S, Bankaitis AU, Klodd D, Haberkamp T. (2006) What's still growing on your patient's hearing aids? *Hear J.* 59 (9), 45–48.

World Health Organization. (2004) Practical guidelines for infection control in health care facilities. Available online at: http://74.125.95.132/search?q=cache:IJTpaPrtK0YJ:www.searo.who.int/LinkFiles/Publications_PracticalguidelinSEAROpub41.pdf+practical+guidelines+for+infection+control+in+health+care+facilities&hl=en&ct=clnk&cd=1&gl=us.

SECTION V

Appendices

Genetics Glossary (Terminology)

Here we provide definitions of terms used in Chapter 25 that may not be familiar to audiologists. While this serves as a supplement to the chapter on hereditary hearing loss, it is not all-inclusive. Curious readers are referred to several excellent online glossaries of genetics terminology. These include the glossary in Genetics Home Reference (http://ghr.nlm.nih.gov/glossary), the illustrated glossary found in GeneReviews (http://www.ncbi.nlm.nih.gov/books/NBK5191/) and the Talking Glossary of Genetics Terms produced by the National Genome Research Institute (http://www.genome.gov/glossary/).

Adenine: One of the four chemical bases in *DNA* and *RNA*. The others are *cytosine, guanine* and *thymine* (*uracil* in RNA). It pairs with thymine on the complimentary DNA strand. Adenine is represented by the letter A.

Allele: One version of a *gene* or a specified location (*locus*) on a *chromosome* (from GeneReviews, http://www.ncbi.nlm.nih.gov/books/NBK5191/).

Amino acid: Molecules that are the building blocks of *proteins*. There are a total of 20 amino acids encoded by *nucleotides* in *DNA*. A series of three nucleotides (or one *codon*) encodes a specific amino acid. Many amino acids are strung together in long chains to form proteins.

Aneuploidy: One or more extra or missing *chromosomes*. For example, *trisomy* or *monosomy*.

Anterior lenticonus: Eye disorder in which the lens bulges in an anterior direction (toward the front of the body). Diagnosis can be made by microscopic examination of the eye. This finding is a characteristic feature of Alport syndrome.

Autosomal/autosomes: Refers to any of the *chromosomes* other than the sex-determining chromosomes (i.e., the X and Y); can also refer to the *genes* on the nonsex chromosomes (from GeneReviews, http://www.ncbi.nlm.nih.gov/books/NBK5191/).

Autosomal dominant: Type of inheritance pattern in which a single mutant copy of a *gene* on any one of the nonsex *chromosomes* is required for expression of a condition or characteristic. That is, one *wild-type* allele and one mutant allele are sufficient to confer disease.

Autosomal recessive: Type of inheritance pattern in which two mutant copies of a *gene* (i.e., two mutant *alleles*) on a *homologous* pair of any of the nonsex *chromosomes* are required for expression of a condition or characteristic.

Base pairs: Pairs of chemical bases that bond to each other in the double strands of *DNA*. *Adenine* always pairs with *thymine,* and *cytosine* always pairs with *guanine*.

Biallelic: Involving both *alleles* of a *gene*.

Biotinidase deficiency: *Heritable* condition in which the body is unable to process the vitamin biotin appropriately. Treatment with biotin supplements helps to reduce or prevent symptoms, including hearing loss.

Centromere: Constricted region that separates the long arm (*q arm*) and short arm (*p arm*) of the *chromosome*.

Choana (pl. choanae): Either one of the paired openings of the nasal cavity into the nasopharynx.

Chromosome: Physical structure consisting of *DNA* and supporting *proteins* called chromatin. Human cells normally contain 46 chromosomes identified as 23 pairs; 22 pairs are *autosomes* and 1 pair are the sex chromosomes (from GeneReviews, http://www.ncbi.nlm.nih.gov/books/NBK5191/).

Codon: Set of three adjacent *nucleotides* that collectively code for an *amino acid,* or the initiation (start codon) or end (stop codon) of *translation* of coding *DNA* into amino acids.

Coloboma: Congenital malformation, often described as a hole, in a structure of the eye (e.g., iris, retina, lid, optic nerve, etc.).

Compound heterozygote: Individual who has two different mutant *alleles* at a particular *locus,* one on each *chromosome* of a pair; usually refers to individuals affected with an *autosomal recessive* disorder (from GeneReviews, http://www.ncbi.nlm.nih.gov/books/NBK5191/).

Consanguineous: Term used to describe individuals with common ancestry; related by blood

Consanguinity: State of being *consanguineous*.

Cytogenetics: Branch of biology concerned primarily with studying *chromosomes*.

Cytosine: One of the four chemical bases in *DNA* and *RNA*. The others are *adenine, guanine,* and *thymine* (*uracil* in RNA). It pairs with guanine on the complimentary DNA strand. Cytosine is represented by the letter C.

Deletion: Loss of genetic material, ranging from a single *nucleotide* to an entire piece of a *chromosome*. An analogy of one type of deletion at the molecular level is shown in Table 25.1.

***De novo* mutation:** Genetic *mutation* that occurs in the germ cell (i.e., sperm or egg) or fertilized embryo; a new mutation. The mutation is not carried by either parent.

Digenic inheritance: Additive disease-causing interaction of *mutations* of two different *genes* at different *loci* (vs. recessive conditions where mutations occur at corresponding *alleles* at the same locus).

DNA: Deoxyribonucleic acid; the molecule that encodes the *genes* responsible for the structure and function of an organism and allows for transmission of genetic information from one generation to the next (from GeneReviews, http://www.ncbi.nlm.nih.gov/books/NBK5191/).

Duplication: Type of *mutation* in which there is an extra copy of a *gene* or segment of contiguous *DNA* resulting in extra genetic material on the *chromosome.*

Dysmorphic: Structural abnormality of the anatomy, often congenital.

Dystopia canthorum: Lateral displacement of the inner canthus of the eyes that gives the appearance of a wide nasal bridge.

Encephalomyopathy: Disorder or disease of the brain or spinal cord; often referring to global dysfunction in these structures.

Euploidy: Normal number of *chromosomes.* In humans there are 23 pairs of chromosomes, 46 in total; 44 of these chromosomes are *autosomes,* and 2 are sex chromosomes (X or Y). This is typically expressed as 46, XX in females and 46, XY in males.

Exome: Collectively, all of the *exons* in the *genome* of a given organism or cell.

Exon: Sequence of *DNA* that remains present (i.e., is not removed) in the mature messenger *RNA* and that codes for the amino acids of the protein product (modified from GeneReviews, http://www.ncbi.nlm.nih.gov/books/NBK5191/).

Expansion: Type of genetic *mutation* in which a segment of *DNA* is aberrantly repeated within a sequence. An analogy of expansion at the molecular level is shown in Table 25.1.

Expression: In genetics (1) the detectable outcome(s) of a *gene;* the observable effect of a gene in traits. For example, if a person has a mutation in a gene that is critical for hearing and as a result, that person has a hearing loss. The *genotype* has caused a hearing loss *phenotype* (i.e., the hearing loss trait is *expressed*). Also (2), expression of a gene to produce a protein product.

Frameshift mutation: Type of genetic *mutation* when an *insertion* or *deletion* of *nucleotides* occurs by some multiple other than three so that all subsequent *codons* will be affected, thus shifting the entire reading frame of the remaining sequence. An analogy of a frameshift mutation is provided in Table 25.1.

Gene: The basic unit of heredity, consisting of a segment of *DNA* arranged in a linear manner along a *chromosome.* A gene codes for a specific *protein* (from GeneReviews, http://www.ncbi.nlm.nih.gov/books/NBK5191/).

Gene therapy: Altering the *expression* of abnormal *genes* with genes that function normally in an attempt to treat a genetic condition.

Genetic markers: Identifiable segment of *DNA* with enough variation among individuals that its inheritance and coinheritance with *alleles* of a given *gene* can be traced (from GeneReviews, http://www.ncbi.nlm.nih.gov/books/NBK5191/).

Genetic modifiers: See *modifier genes.*

Genome: Entirety of an organism's genetic material. This includes *DNA* sequences that code for *genes* and DNA sequences that are noncoding.

Genotype: An organism's specific genetic composition; the combination of *alleles* specific to an individual. Also can refer to a specific pair of alleles at a given *locus.*

Guanine: One of the four chemical bases found in *DNA* and *RNA.* The others are *adenine, cytosine,* and *thymine* (*uracil* in RNA). It pairs with cytosine on the complimentary DNA strand. Guanine is represented by the letter G.

Hemizygous: Refers to an individual with only one copy of a *gene* pair or one member of a *chromosome* pair. For example, males are hemizygous for many genes on both the X and Y chromosomes because they have only one copy of each of these genes.

Heritability: Ability of a trait to be inherited (i.e., passed from parent to offspring); the proportion of observable traits related to genetic factors.

Heterogeneity: In genetics, when mutations of different *genes* independently result in a single *phenotype.* For example, hereditary hearing loss can result from mutations in many different genes (i.e., it is a *heterogeneous* condition).

Heteroplasmy: The situation in which, within a single cell, there is a mixture of mitochondria, some containing mutant *DNA* and some containing normal DNA (from GeneReviews: http://www.ncbi.nlm.nih.gov/books/NBK5191/).

Heterozygous: Refers to an individual with two different variations of the paired *alleles* of a *gene;* in such cases the person is heterozygous for that gene.

Homologous *(chromosomes):* A pair of chromosomes—one maternally derived and one paternally derived. The organization of *loci* on each chromosome should be the same.

Homozygous: Refers to an individual with identical copies of the paired *alleles* of a *gene;* in such cases the person is homozygous for that gene.

Insertion: Type of genetic *mutation* when segments of *DNA* ranging from one to several *nucleotides* in length are incorrectly inserted within a sequence. An analogy of insertion at the molecular level is shown in Table 25.1.

Intron: Noncoding segment of *DNA* interspersed among *exons;* a portion of a *gene* that does not function in coding for *amino acids.* The functions of introns are not entirely understood, but they are removed during the process of *RNA* editing to produce the mature RNA that is *translated* into a protein product.

Inversion: When a segment of a *chromosome* breaks off and is reinserted in a reverse order within the same chromosome.

Iris heterochromia: Differently colored areas of the same eye, or each eye being a different color.

Karyotype: Collective number, structure, and organization of *chromosomes;* can refer to the chromosomal character of either an individual or a species.

Locus (pl. loci): Specific physical location of a *gene* on a *chromosome.*

Matrilineal inheritance: Traits passed from parent to offspring, only through the female (maternal) line. This is typically via *mtDNA.*

Meiosis: A phase of nuclear cell division that results in four daughter cells that each contain half the number of *chromosomes* of the parent cell. Meiosis results in the production of sperm and oocytes containing exactly one of each chromosome.

Mendelian inheritance: Process by which traits are passed from parent to offspring via a single *gene locus* following a set of principles proposed by Gregor Mendel: The Law of Segregation (one copy of each gene pair is inherited separately from each parent) and the Law of Independent Assortment (separate genes for different traits are inherited independently of each other).

Messenger RNA (mRNA): One of the three types of ribonucleic acid (RNA), mRNA is the spliced, or processed, transcript that is exported from the cell nucleus to the ribosomes in the cytoplasm where transcription occurs. *Amino acids* encoded by the mRNA are assembled to form *proteins.*

Missense mutation: Type of genetic *mutation* in which a single *nucleotide* is substituted for another, the outcome of which causes the substitution of a different amino acid. An analogy of a missense mutation is shown in Table 25.1.

Mitochondria: Subunit of a cell (organelle) with a primary role of supplying the chemical energy within the cell.

Mitochondrial DNA (mtDNA): The unique *DNA* of mitochondria, which is separate from nuclear DNA. Because sperm cells lose their mitochondria during fertilization, mtDNA is only inherited from the female parent (*matrilineal inheritance*) and is passed on to all of her offspring.

Mitosis: A phase of cell division where the replication of cells results in two daughter cells that carry the exact *chromosomes* and nuclear *DNA* as the parent cell.

Modifier genes: *Genes* that alter, or modify, the *expression* of a different gene. These can influence the onset, progression, and severity of disease.

Monogenic inheritance: When *heritable* conditions or traits are caused by a mutation in a single *gene*.

Monosomy: Total or partial loss of one copy of a *chromosome* pair.

Mosaicism: When a single organism has some cells that carry a different *genome* than other cells in the body.

Multifactorial inheritance: Inheritance related to multiple factors, at least one of which is genetic; can describe the direct and indirect interaction between *genes* and environmental exposures that, collectively, results in disease.

Mutation: A mutation is an alteration in the *nucleotide* sequence of a *gene.* A mutation may be benign with no functional consequence (*polymorphism*), it may cause disease or dysfunction, or it may be beneficial to the organism.

Nonsense mutation: Type of genetic *mutation* in which a single *nucleotide* is substituted for another, resulting in the production of a premature *stop codon.* An analogy of a nonsense mutation is shown in Table 25.1.

Nonsyndromic: Not part of a *syndrome;* a disorder that occurs in isolation. For example, hearing loss in the absence of other clinical signs.

Nucleotide: Molecules composed of a nitrogenous base, a five-carbon sugar (ribose or deoxyribose) and at least one phosphate group. Nucleotides are the basic building blocks of *DNA* and *RNA.*

Obligate Carriers: Family members who have one copy of a genetic *mutation* in question based on the pattern of inheritance and genotype of their offspring.

p arm: Short arm of a *chromosome.*

Pathognomonic: Indicative of disease; a finding or findings characteristic of a particular disease and not observed with other conditions; a diagnostic marker.

Patrilineal inheritance: Traits passed from parent to offspring, only through the male (paternal) line. This is typically via the Y *chromosome.*

Pedigree: Visual representation of a family's health history through a common set of symbols; charting tool used to catalog the occurrence and presentation of *phenotypes,* with genetic relationships traced through connecting lines and across generations.

Penetrance: Percentage of individuals with a genetic *mutation* that express the associated trait; often related to *dominant inheritance,* the percentage of individuals who carry a dominant *mutation* who actually *express* the trait. *Modifier genes* also affect penetrance.

Phenocopy: Condition in which an environmentally caused trait mimics an inherited trait. For example, hearing loss from chemotherapy versus hereditary hearing loss.

Phenotype: Manifestation of *genes* into observable traits; the observable properties of an organism (e.g., eye color, height, hearing loss).

Pleiotropy: Multiple, seemingly unrelated *phenotypes* that result from a single genetic *mutation*(s).

Point mutation: Type of genetic *mutation* in which a single *nucleotide* is substituted for another, but the total number of nucleotides in the sequence remains unchanged.

Polygenic inheritance: Cumulative effect of many *genes* on a *phenotype;* in contrast to effects from a single gene or pair of genes.

Polymorphism: Variations in a *gene,* or *DNA* sequence that occur with high frequency in a population. They can range from a single nucleotide polymorphism (SNP) to changes in large segments of DNA, but they are nondisease causing.

Polyploidy: Extra copy of an entire set of *chromosomes;* common in some species, but lethal in humans.

Proband: First affected family member to come to medical attention.

Protein: Class of molecules made up of long chains of *amino acids,* encoded by *DNA* or *RNA*. Proteins have wide-ranging functions including structure, function, and regulation of organs and tissues in the body.

q arm: Long arm of a *chromosome.*

Retinitis pigmentosa: Degenerative disease of the retina in the eye; characterized by decreased vision at night and loss of peripheral vision.

Ribonucleic acid (RNA): Biological molecule with vital roles in coding, decoding, regulation, and expression of *genes.* It is composed of a single strand of bases (A, C, G and U) and a ribose–phosphate backbone.

Ribosomal RNA (rRNA): RNA component of the *ribosome.* rRNA directs the translation of mRNA into *proteins.*

Ribosome: Cellular complex that helps bind *amino acids* to form *proteins* during the process of *translation.*

Segregation: Separation of *phenotypic* or *genotypic* elements within a population.

Sex-linked inheritance: Type of inheritance pattern in which there is a *mutation* in a *gene* on one of the sex *chromosomes.* When the mutated gene is on the X chromosome, this pattern is termed X-linked inheritance. When the mutated gene is on the Y chromosome this pattern is termed Y-linked inheritance.

Start Codon: Specific group of three *nucleotides* in a coding region of a *gene* that encode the beginning of the chemical *translation* into a *protein.*

Stop Codon: Specific group of three *nucleotides* in a coding region of a *gene* that encode the end of the chemical *translation* into a *protein.*

Syndromic: The co-occurrence of related symptoms or signs associated with disease. For example, individuals with Usher syndrome have hearing loss and retinitis pigmentosa.

Telomere: Region of repetitive *nucleotide* sequences at each end of the *chromosome.*

Thymine: One of four chemical bases in *DNA*. The others are *adenine, cytosine,* and *guanine.* It pairs with guanine on the complimentary DNA strand. Thymine is represented by the letter T. Thymine is replaced by *uracil* (U) in *RNA.*

Transcription: The process of synthesizing *RNA* from a *DNA* template.

Transfer RNA (tRNA): Located in the cytoplasm of a cell, transfer RNA delivers an *amino acid* to the ribosome that corresponds to each three-*nucleotide codon* of *mRNA.* The amino acid is transferred to the growing polypeptide chain on the ribosome to make *proteins.*

Translation: The process of synthesizing an *amino acid* sequence (*protein* product) from the *messenger RNA* (mRNA) code (from GeneReviews, http://www.ncbi.nlm.nih.gov/books/NBK5191/).

Translocation: Change of location. In genetics this typically refers to transfer of a segment of a *chromosome* to a new position, most often on another chromosome.

Trisomy: Total or partial gain of one copy of a *chromosome* pair.

Uracil: One of the four chemical bases in *RNA*. The others are *adenine, cytosine,* and *guanine.* Uracil is represented by the letter U. *Thymine* on *DNA* is replaced by uracil on RNA.

Variable expressivity: Variation in the *phenotypic* expression among individuals who carry the same genetic *mutation* (e.g., varying degrees of hearing loss).

Wild type: The *genotype* or *phenotype* most commonly observed in nature.

IDEA 2004 Key Regulations Pertaining to Deaf Education and Audiology

PART B: RELATED SERVICES 34 CFR 300.34(b)

Exception; services that apply to children with surgically implanted devices, including cochlear implants.

[1] Related services do not include a medical device that is surgically implanted, the optimization of that device's functioning (e.g., mapping), maintenance of that device, or the replacement of that device.

[2] Nothing in paragraph (b)(1) of this section:

(i) Limits the right of a child with a surgically implanted device (e.g., cochlear implant) to receive related services (as listed in paragraph (a) of this section) that are determined by the IEP Team to be necessary for the child to receive FAPE.

(ii) Limits the responsibility of a public agency to appropriately monitor and maintain medical devices that are needed to maintain the health and safety of the child, including breathing, nutrition, or operation of other bodily functions, while the child is transported to and from school or is at school; or

(iii) Prevents the routine checking of an external component of a surgically implanted device to make sure it is functioning properly, as required in §300.113(b).

PART B: DEFINITION OF AUDIOLOGY 34 CFR 300.34(c)(1)

Audiology includes:

(i) Identification of children with hearing loss;

(ii) Determination of the range, nature, and degree of hearing loss, including referral for medical or other professional attention for the habilitation of hearing;

(iii) Provision of habilitation activities, such as language habilitation, auditory training, speech reading (lip-reading), hearing evaluation, and speech conservation;

(iv) Creation and administration of programs for prevention of hearing loss;

(v) Counseling and guidance of children, parents, and teachers regarding hearing loss; and

(vi) Determination of children's needs for group and individual amplification, selecting and fitting an appropriate aid, and evaluating the effectiveness of amplification.

PART C: DEFINITION OF AUDIOLOGY 34 CFR 303.12(d)

Audiology includes:

(i) Identification of children with impairments, using at risk criteria and appropriate audiological screening techniques;

(ii) Determination of the range, nature, and degree of hearing loss and communication functions, by use of audiologic evaluation procedures;

(iii) Referral for medical and other services necessary for the habilitation or rehabilitation of children with auditory impairment;

(iv) Provision of auditory training, aural rehabilitation, speech reading and listening device orientation and training, and other services;

(v) Provision of services for the prevention of hearing loss; and

(vi) Determination of the child's need for individual amplification, including selecting, fitting, and dispensing of appropriate listening and vibrotactile devices, and evaluating the effectiveness of those devices.

PART B: INTERPRETING SERVICES 34 CFR 300.34(c)(4)

Interpreting services includes:

(i) The following when used with respect to children who are deaf or hard of hearing: Oral transliteration services, cued language transliteration services, and sign language transliteration and interpreting services, and transcription services, such as communication access real-time translation (CART), C-Print, and TypeWell; and

(ii) Special interpreting services for children who are deaf–blind.

PART B: ASSISTIVE TECHNOLOGY 34 CFR 300.105(a)(2)

On a case-by-case basis, the use of school-purchased assistive technology devices in a child's home or in other settings is required if the child's IEP Team determines that the child needs access to those devices in order to receive FAPE.

PART B: ROUTINE CHECKING OF HEARING AIDS AND EXTERNAL COMPONENTS OF SURGICALLY IMPLANTED MEDICAL DEVICES 34 CFR 300.113

(a) *Hearing aids.* Each public agency must ensure that hearing aids worn in school by children with hearing impairments, including deafness, are functioning properly.
(b) *External components of surgically implanted medical devices.*
  (1) Subject to paragraph (b)(2) of this section, each public agency must ensure that the external components of surgically implanted medical devices are functioning properly.
  (2) For a child with a surgically implanted medical device who is receiving special education and related services under this part, a public agency is not responsible for the postsurgical maintenance, programing, or replacement of the medical device that has been surgically implanted (or of an external component of the surgically implanted medical device).

PART B: DEVELOPMENT, REVIEW, AND REVISION OF IEP, CONSIDERATION OF SPECIAL FACTORS 34 CFR 300.324(2)(iv)

The IEP Team **must:**

(iv) Consider the communication needs of the child, and in the case of a child who is deaf or hard of hearing, consider the child's language and communication needs, opportunities for direct communications with peers and professional personnel in the child's language and communication mode, academic level, and full range of needs, including opportunities for direct instruction in the child's language and communication mode; and
(v) Consider whether the child needs assistive technology devices and assistive technology services.

ASSISTIVE TECHNOLOGY; PART B: 34 CFR 300.5-.6 AND C: 34 CFR 303.12

Assistive technology device means any item, piece of equipment, or product system, whether acquired commercially off the shelf, modified, or customized, that is used to increase, maintain, or improve the functional capabilities of children with disabilities. The term does not include a medical device that is surgically implanted, or the replacement of such device.

Assistive technology service means any service that directly assists a child with a disability in the selection, acquisition, or use of an assistive technology device. The term includes:

(a) The evaluation of the needs of a child with a disability, including a functional evaluation of the child in the child's customary environment;
(b) Purchasing, leasing, or otherwise providing for the acquisition of assistive technology devices by children with disabilities;
(c) Selecting, designing, fitting, customizing, adapting, applying, maintaining, repairing, or replacing assistive technology devices;
(d) Coordinating and using other therapies, interventions, or services with assistive technology devices, such as those associated with existing education and rehabilitation plans and programs;
(e) Training or technical assistance for a child with a disability or, if appropriate, that child's family; and
(f) Training or technical assistance for professionals (including individuals providing education or rehabilitation services), employers, or other individuals who provide services to, employ, or are otherwise substantially involved in the major life functions of children with disabilities.

PART B: DEFINITIONS 34 CFR 300.8(b)

[1] *Deaf-blindness* means concomitant hearing and visual impairments, the combination of which causes such severe communication and other developmental and educational needs that they cannot be accommodated in special education programs solely for children with deafness or children with blindness.
[2] *Deafness* means a hearing impairment that is so severe that the child is impaired in processing linguistic information through hearing, with or without amplification that adversely affects a child's educational performance.
[3] *Hearing impairment* means an impairment in hearing, whether permanent or fluctuating, that adversely affects a child's educational performance but that is not included under the definition of deafness in this section.

Functional Skills Screening for Children with Hearing Loss[1]

This functional skill screening is designed to gain a profile of a child's general development as well as skills that are specifically impacted by hearing impairment. It may also be used to monitor a child's development over time. Information may be obtained through observation, review of records, interview with family members, or the child directly. Tools such as Ida's *My World* may also be helpful in gaining an understanding of the child's perceptions. Any concerns raised in this process should be discussed with the child's parents, early intervention provider, teacher of the deaf, speech–language pathologist, classroom teacher, or other appropriate personnel so that they are addressed in the child's developmental or educational program.

Name_____ **Date**_____ **AGE**_____ **Person completing form**_____

PROFILE

Domain	Skill Area	❶	❷	❸	❹	❺
Cognitive/ Behavioral/ Social	Thinking/Reasoning					
	Learning Style/Attention					
	Social/Classroom Behavior					
	Life Skills					
Self-Advocacy	Knowledge					
	Application					
Communication	Expressive					
	Receptive					
Physical	Vision					
	Use of Limbs					
	Balance					
	Overall Physical Health					
Language	Receptive					
	Expressive					
Speech Intelligibility	Skills					
Auditory & Listening Skills	Use of Amplification					
	Audition & Listening					

Directions: Rate each domain using the scale that follows. Then complete the profile by checking the box that corresponds with the score for each skill area of each domain.

❶ Age Appropriate

❷ Mild Limitation

❸ Moderate Limitation

❹ Moderately Severe Limitation

❺ Severe Limitation

[1] Cheryl DeConde Johnson (2013). Adapted from the Colorado Individual Performance Profile (CIPP) (2004), Colorado Department of Education; Karchmer & Allen (1999), The Functional Assessment of Deaf/Hard of Hearing Students 144(2), 68–77; and from Liebermann & Gott (1984), Hearing Impaired Performance Profile – Revised Edition (HIPP-R): An Assessment Integration Document, The School Board of Broward County, Florida.

DIRECTIONS: Rate the child's skills using the following scale as defined by each domain description below:

❶ Age Appropriate ❷ Mild Limitation ❸ Moderate Limitation ❹ Moderately Severe Limitation ❺ Severe Limitation

Domain: Cognitive/Behavioral/Social						Score
Thinking/ Reasoning	❶ Child thinks and reasons normally, plays games, solves puzzles, and problems comparably to other students of the same age.	❷ Between 1 & 3	❸ Child is slow to solve age-appropriate puzzles and problems or learn new things, but may acquire these intellectual skills with instructional supports.	❹ Between 3 & 5	❺ Child has considerable difficulty solving age-appropriate puzzles and problems, lags far behind peers and may require individualized instruction to master even simple tasks.	
Learning Style/ Maintaining Attention to Classroom Tasks	❶ Child has skills needed to independently engage in learning tasks; usually attends to classroom instruction sufficiently to learn material; requires minimal teacher support to complete class work.	❷ Between 1 & 3	❸ Child has some skills needed to independently engage in learning; attention in class is frequently off-task, sufficient to impede learning; child can master classroom tasks with close monitoring and instructional support.	❹ Between 3 & 5	❺ Child is unable to independently engage in learning; exhibits extreme difficulty attending to classroom material, even for short periods of time; may act impulsively or withdraw frequently from classroom activities.	
Social Interaction/ Classroom Behavior	❶ Child exhibits social skills and behavior that are appropriate for his/her age.	❷ Between 1 & 3	❸ Child exhibits some inappropriate behavior that may include fighting, biting, hitting, screaming; however, behavior is not disruptive enough to require frequent separation of the child from the classroom.	❹ Between 3 & 5	❺ Child frequently exhibits inappropriate social behavior and is often disruptive of classroom activities; often needs to be separated from the class.	
Life Skills	❶ Child is able to take care of personal possessions, dress, toilet, follow rules, travel, and use money independently similar to other students of the same age.	❷ Between 1 & 3	❸ Child exhibits some inappropriate behavior that may include fighting, biting, hitting, screaming; however, behavior is not disruptive enough to require frequent separation of the child from the classroom.	❹ Between 3 & 5	❺ Child frequently exhibits inappropriate social behavior and is often disruptive of classroom activities; often needs to be separated from the class.	
Domain: Self-Advocacy						**SCORE**
Knowledge	❶ Child understands personal medical status and health needs including concepts of hearing and hearing loss and interpretation of audiogram; hearing and other assistive technologies; accommodations (e.g., strategies to address learning and communication needs) and consumer rights (e.g., disability and access rights).	❷ Between 1 & 3	❸ Child understands basic aspects of personal medical status and health needs (e.g., can describe basic components of hearing loss and basic communication implications), hearing and other assistance technologies (e.g., describes basic aspects of personal hearing instruments and other assistive technologies used), and basic accommodations (e.g., priority seating, assistive technology).	❹ Between 3 & 5	❺ Child has no understanding of personal medical status and health needs, use of hearing and other assistance technologies, or accommodations.	

Application	❶ Child consistently assumes responsibility for personal health and medical needs including management and appropriate use of hearing and other assistive technologies, applying accommodations (e.g., strategies to address compromised learning and communication needs) and advocating for consumer rights (e.g., disability and access rights).	❷ Between 1 & 3	❸ Child assumes some responsibility for some personal health and medical needs including basic use and troubleshooting of hearing and other assistance technologies and applying some basic accommodations (e.g., priority seating, assistive technology, closed captioning).	❹ Between 3 & 5	❺ Child does not assume any responsibility for personal health and medical needs, management of hearing and other assistive technologies or accommodations.	

Domain: Communication	Note: If the child uses a sign language interpreter, evaluate functioning in reference to communication through that interpreter.					**SCORE**
Expressive Communication	❶ Child expressively communicates with his/her teacher and peers fluently and easily using appropriate pragmatic skills (e.g., listening, turn-taking, stays on topic).	❷ Between 1 & 3	❸ Child has some difficulty expressing him/herself and using pragmatic skills (e.g., interrupts inappropriately, switches topics without informing others) with the mode of communication generally used in the classroom; these difficulties can be overcome by prompting, repetition, and explanation.	❹ Between 3 & 5	❺ Child has considerable difficulty expressing him/herself and using appropriate pragmatic skills in the mode of communication generally used in the classroom.	
Receptive Communication	❶ Child comprehends the communication of others in the classroom accurately and easily.	❷ Between 1 & 3	❸ Child has some difficulty comprehending communication from others in the classroom using the mode of communication generally used for classroom interaction. Difficulties can be remediated by repetition and explanation.	❹ Between 3 & 5	❺ Child has considerable difficulty comprehending communication from others in the classroom, even when accommodations such as interpreters, assistive listening devices, etc., are used.	

Domain: Physical						**SCORE**
Vision	❶ Child sees with normal acuity, using corrective lenses if necessary.	❷ Between 1 & 3	❸ Even with corrective lenses, student has some problems seeing the blackboard or objects in visual periphery, or reading small print, and requires minimal additional accommodations, e.g., preferential seating, magnification of reading materials.	❹ Between 3 & 5	❺ Even with corrective lenses or other accommodations, student cannot see and comprehend visual communication (such as sign language) from across a room; requires significant accommodations, e.g., large print, Braille, mobility training, deaf-blind interpreter.	
Use of hands, arms, legs	❶ Child uses hands, arms, and legs normally in daily activities, e.g., walking up and down stairs, using a pencil to write, participating in physical activities.	❷ Between 1 & 3	❸ Child has some limitations in the use of hands, arms, and/or legs, but is ambulatory and can use hands and arms for simple daily activities.	❹ Between 3 & 5	❺ Child is nonambulatory or is severely limited in his/her use of hands and arms.	

Balance (dizziness, motion sickness, coordination in the dark)	❶ Child participates normally in all physical activities without loosing balance, falling down, or experiencing dizziness.	❷ Between 1 & 3	❸ Child reports dizziness, nausea, falling down, or shows some mild lack of coordination when participating in physical activities.	❹ Between 3 & 5	❺ Child often stumbles or falls due to lack of balance, and/or frequently reports feeling dizzy or sick while in motion.	
Overall physical health	❶ Child has the usual health problems and illnesses typical for children the same age. Absences from school due to illness typical for children the same age.	❷ Between 1 & 3	❸ Child has frequent or ongoing health problems, but they are either mild or medically controllable and do not significantly impair educational progress.	❹ Between 3 & 5	❺ Child has frequent or ongoing health problems that are either not well controlled or result in near-total restriction of activities.	

Domain: Language

SCORE

Receptive Language	❶ Child understands all 8 functions of language appropriate for age: Directions, Explanation, Narration, Description, Negotiation/ Persuasion, Conversation, Questions, Writing (written language).	❷ Child understands 6/8 functions of language appropriate for age and needs support to maintain them.	❸ Child understands language within 1 year of age level (e.g., understands a variety of sentence patterns, assimilates new language with ease, follows and completes multi-step tasks- oral & written).	❹ Child understands language between 1 and 2 years below age level (e.g., limited variety of sentence patterns, assimilates new language after repeated instruction, follows and completes a two to three part task [oral & written], has limited comprehension of idioms).	❺ Child understands language at level more than 2 years below age (e.g., understands concrete language).	
Expressive Language	❶ Child understands all 8 functions of language appropriate for age (see receptive language).	❷ Student understands 6/8 functions of language appropriate for age and needs support to maintain them.	❸ Child uses language within 1 year of age level (e.g., varies sentence patterns, assimilates new language, uses some idioms, uses some multiple word meanings).	❹ Child uses language between 1 and 2 years below age level (e.g., limited variety of sentence patterns, assimilates new language after repeated instructions, conveys meaning despite numerous structural errors).	❺ Child uses language at level more than 2 years below age (e.g., conveys meaning by using a variety of communication systems).	

Domain: Speech Intelligibility

SCORE

	❶ Child's speech is completely intelligible to a variety of listeners.	❷ Child's speech is generally intelligible to a variety of listeners (e.g., vocal quality appropriate for age and sex, uses intonation and rhythm patterns to convey meaning, has few articulation errors).	❸ Child's speech is fairly intelligible to a variety of listeners (e.g., intelligible to listener when topic is unknown, minimal problems in voicing and pitch and varying intonation and/or rhythm patterns).	❹ Child's speech is very difficult to understand by a variety of listeners (e.g., vocalizes spontaneously but only isolated words and/or phrases are understood, voicing and pitch are irregular with significant difficulty with intonation and/or rhythm patterns).	❺ Child's speech is unintelligible to an untrained listener or child does not use speech (e.g., only vocalizes on demand, imitates just a few speech sounds, poor voice quality [nasal, guttural, raspy], pitch is irregular).	

Domain: Auditory and Listening Skills						SCORE
Use of Amplification	❶ Child consistently uses and manages amplification (personal and assistive technology) as prescribed (e.g., independent use and care, reports problems when they occur).	❷ Child successfully uses prescribed amplification about 75% of the time, requires some prompting; can independently care for instruments and report problems.	❸ Child requires prompting and assistance to use prescribed amplification; may decline use of assistive technology; needs assistance with use and care, does not consistently report problems.	❹ Child refuses to use prescribed amplification or only uses upon demand; needs regular assistance with use and care; does not report problems when they occur.	❺ Child does not benefit from amplification or prefers not to use amplification.	
Audition & Listening	❶ Child uses audition as the primary modality for receptive communication (e.g., listens attentively, understands spoken language and idioms including stress patterns for meaning and inferring discrete emotional information such as sarcasm).	❷ Child uses audition, supplemented by visual supports when needed for receptive communication (e.g., listens attentively, understands speech auditorially) has some difficulty relating stress patterns to meaning, has difficulty inferring fine emotional information (such as sarcasm) based on intonation.	❸ Child uses a combination of auditory and visual modalities for receptive communication (e.g., understands speech when "face-to-face" with speaker, interprets an auditory speech signal when given structural cues, has difficulty inferring gross emotional information such as anger based on intonation).	❹ Child realizes some benefit from auditory modality but primarily relies on visual or tactile input for receptive communication (e.g., uses audition only for awareness of sound, recognizes common environmental sounds).	❺ Child does not use the auditory modality for listening or communication.	

Self-Advocacy Skills Checklist

Self-Advocacy Competencies: Elementary School	
Health/Medical Access	Basic concepts of hearing ❑ Describes how we hear ❑ Describes basic problems that cause hearing loss Basic parameters of the audiogram ❑ Describes degrees of hearing loss ❑ Describes basic implications of hearing loss
Hearing Technology and Usage	Responsibility for equipment ❑ Understands and reports when amplification devices are functioning (i.e., ON/OFF) ❑ Reports other malfunctions such as static, interference, etc. ❑ Learns to manage daily maintenance of equipment-charging Hearing Assistance Technology (HAT), changing batteries, basic earmold cleaning ❑ Uses a calendar to report daily use and device functioning Use of individual amplification devices ❑ Knows the basic parts of the personal amplification used (i.e., earmold, microphone, battery door) ❑ Knows the basic parts of HAT used (transmitter vs. receiver, attachment of audio shoes)
Educational Services	Communication challenges and needs ❑ Describes basic characteristics of successful communication ❑ Identifies basic accommodations to address personal communication needs (e.g., priority seating)

Self-Advocacy Competencies: Middle School	
Health/Medical Access	Concepts of hearing and hearing loss ❑ Describes own hearing loss (degree and configuration) ❑ Describes cause of HL (if known) ❑ Describes basic communication implications of hearing loss ❑ Describes basic hearing loss prevention strategies
Hearing Technology and Usage	Responsibility for equipment ❑ Transports equipment to and from various school environments ❑ Understands and is able to notify teacher or speaker when devices are not working properly ❑ Understands the flexibility of the devices (i.e., ability to couple to audio devices–computers, TV, PA system) Use of individual amplification devices ❑ Understands basic functioning of personal and HAT devices ❑ program options in HA/CI/BAHA ❑ limitations of technology ❑ Utilizes the devices in different environments (i.e., lectures, small groups, pass around) ❑ Actively participates in training of staff on equipment Use of assistive technologies ❑ Identifies and demonstrates basic understanding of other assistive technologies to accommodate hearing loss (e.g., telephone, captioning, alerting devices)

Educational Services	Strategies to address communication challenges ❑ Describes communication challenges and strategies that work ❑ Identifies needed accommodations and presents them at IEP meeting ❑ Describes needed accommodations to instructors Legal rights ❑ Understands basic legal rights under IDEA

Self-Advocacy Competencies: High School

Health/Medical Access	Concepts of hearing and hearing loss ❑ Provides detailed description of own hearing loss (type, degree, configuration, cause, implications for communication) ❑ Develops and rehearses a script for disclosing hearing loss information and required accommodations ❑ Explains communication implications to others Access to hearing health professionals ❑ Identifies pertinent medical and health specialists, their supporting roles, and how to locate them (audiology, otology, genetics, mental health/counseling) ❑ Identifies own medical/health support persons
Hearing Technology and Usage	Responsibility for equipment ❑ Demonstrates ability to troubleshoot all hearing and HAT and follows predetermined procedures for getting equipment serviced Use of individual amplification devices ❑ Understands how to manipulate technology in more difficult listening situations ❑ Understands how to connect equipment into other audio devices independently ❑ Demonstrates knowledge of HAT use beyond the classroom Use of resources ❑ Demonstrates use of web to locate information and resources about hearing and HAT ❑ Describes funding options for hearing, HAT, and other assistive technologies Use of other assistive technologies to accommodate hearing loss ❑ Describes characteristics of other assistive technologies such as telephone, captioning, alerting devices, text messaging
Educational Services	Educational history and current status ❑ Explains educational strengths and challenges ❑ Identifies academic support needs ❑ Formulates present levels of functioning for IEP and IEP goals ❑ Describes achievements and performance levels for ***Transition Plan Summary of Performance*** Personal Profile and Accommodations Letter (PPAL) ❑ Develops a PPAL that identifies needed accommodations and presents profile at IEP meeting ❑ Describes needed accommodations to instructors ❑ Develops alternative strategies/solutions when accommodations not provided/available Transition ❑ Describes and differentiates IDEA, 504, ADA as it relates to hearing loss including eligibility criteria ❑ Provides evidence of successfully submitted scholarship applications when pursing higher education or employment applications if pursing employment ❑ Provides evidence of meeting with office of disabilities services to identify available services for higher education or human resource office for employment

Self-Advocacy Competencies: Adult	
Health/Medical Access	❑ Utilizes health and medical support when needed
Hearing Technology and Usage	❑ Utilizes HAT and personal amplification devices in occupational, social, professional contexts ❑ Demonstrates knowledge of where to access information about new technology and its related benefits
Educational and Consumer Awareness	❑ Describes educational history and current performance levels (educational test scores, learning styles, communication abilities) ❑ Describes PPAL to instructors, employers, disability coordinators, VR counselor, community settings ❑ Use 504 and ADA to obtain accommodations ❑ Develops plan to access disability support services when pursing higher education or accommodations for employment

From Guide to Access Planning, www.phonakPhonak US, http://www.phonakpro.com/us/b2b/en/pediatric/GAP.html. Used with permission.

Nonauditory Effects of Noise Exposure

Nonauditory effects of noise exposure are those effects that do not cause hearing loss. Some of these are seen by changes in body functions, such as heart rate, and in learning/cognition in children. Nonauditory effects of noise exposure have been noted as far back as 1930 (Smith and Laird, 1930). In that specific study, nonauditory effects pertain to stomach contractions in healthy human beings when exposed to noise.

There are both laboratory and field studies of nonauditory effects. Laboratory studies set up well-controlled conditions and, therefore, are more suited to examine specific changes but are typically unsuited for examining long-term effects that may result in disease or cognitive/educational problems (Bronzaft, 1991). While laboratory studies can be more precise than field studies, they may or may not have any bearing on reality. In contrast, field studies are inherently less well designed in order to control for unwanted variables, but their conclusions may be more applicable to reality. Field studies are well suited to look at the long-term effects of disease and/or educational effects. For example, Stansfeld et al. (2000) demonstrate that, although transportation (truck) noise can disturb sleep patterns in a well-controlled laboratory setting, this is generally not the case in field studies because people tend to adapt over time to environmental noise. A major difficulty with all research into nonauditory factors is that subjective responses not based on intensity or duration may be quite significant. There are three classic studies from the early 1980s that provide an excellent overview for the interested reader: Cohen and Weinstein (1981) and DeJoy (1984) and Thompson (1981). Although there are more recent studies, these have found similar results. High variability and questionable applicability continue to plague research in this very difficult area.

CARDIOVASCULAR EFFECTS

In well-defined laboratory studies, the "noise/stress hypothesis is well understood: Noise activates the pituitary–adrenal–cortical axis and the sympathetic-adrenal-medullary axis. Changes in stress hormones including epinephrine, norepinephrine, and cortisol are frequently found in acute and chronic noise experiments. The catecholamines and steroid hormones affect the organism's metabolism" (Babisch, 2002, p. 1). However, few measurable biologic changes are directly or indirectly related to clinical changes in a population.

Most of the studies on cardiovascular effects have been performed in the laboratory on animals (mostly on rats). However, DeJoy (1984) commented that the rat may not be an appropriate model and that a primate species may be more appropriate. When primates were used in the laboratory, it was also found that blood pressure increased as the noise levels increased, but there was a large degree of variability in the studies.

In the few field studies on humans, blood pressure has been measured, but again, the level of variability is great. Sloan (1991, p. 23), reviewing available data, notes that when taken as a whole "although there are inconsistencies in the findings…they generally support the assertion that exposure to noise is associated with higher levels of blood pressure." Data are still limited, however, and the results may depend on many uncontrolled factors, such as subjective response, the exact nature of physiologic assessment, and the animal model. In addition, it is still not known whether increased blood pressure in a noisy environment will lead to cardiovascular disease. Stansfeld et al. (2000) echo this concern and demonstrate that although laboratory studies show an association between noise and cardiovascular disease, field studies show only a weak relationship.

The physiologic rationales of the effects on body chemistry as a result of increased exposure to noise are beyond the scope of this chapter, but the interested reader is referred to Babisch (2002) and Raymond (1991).

Effects of Noise on Sleep

Pollak (1991, p. 41) noted that "(1) noises are more annoying when they occur at times when people expect to rest or sleep, (2) noise can interrupt sleep, and (3) noise can also have subtle effects on sleep…that are detectable only with specialized instruments." Most laboratory studies use truck and aircraft noise as stimuli and measure the effect on a range of sleep study parameters. Noise can delay sleep and shift the sleep stages upward (i.e., more shallow sleeping). Upward sleep stage shifts have been observed even in relative quiet with 25- to 30-dB sound pressure level (SPL). Cardiovascular changes are usually not noted until the stimulus level is just below the arousal level for that individual. Thiessen (1978, 1983) found that as peak noise intensity increased, there was a linear increase in the probability of a change in sleep stage. Similar results have been found by Matheson et al. (2003) and Ouis (2002) .

Effects of Noise on Fetal Development

There are some data suggesting an increased risk of noise-induced damage in fetuses, but this is still a very controversial issue. The interested reader is referred to Ryals (1990) and Stansfeld et al. (2000) for more information.

Nakamura (1977) noted low birth weights when the pregnant mother was exposed to high levels of occupational noise. Schell (1981) found that noise may in fact decrease birth weight. However, Edmonds et al. (1979) found that aircraft noise exposure had no significant effect on fetal development in pregnant women. Stansfeld et al. (2000), in laboratory studies, found no evidence that noise exposure contributes to congenital birth defects or low birth weight.

Effects of Noise on Learning

When speech is masked by background noise (e.g., at a noisy party), this is similar to having a hearing loss (with equivalent masked hearing thresholds). Children with even slight hearing losses have been shown to have decreased educational and cognitive performance. Davis (1985) found that children with a minimal (25 dB) hearing loss scored almost two full grade levels lower in reading comprehension by grade 4 (despite having minimal differences in the first grade).

Specifically with respect to normal-hearing children in a noisy school environment, Cohen et al. (1973) found that children whose classrooms were on the street level (nearer to truck and car noise) performed poorer in reading ability than children whose classrooms were in quieter locations. Bronzaft and McCarthy (1975) studied the reading ability of children in one school near elevated train tracks. Half the classrooms faced the train track, and the other half were on the quieter back part of the school. Students in the quieter classrooms did better on reading achievement tests, and by grade 6, those in the quieter classrooms were a full grade point ahead of those in the noisier classrooms. Green et al. (1982), in studying children near a New York airport, found that as noise level increased, the percentage of those children falling below grade reading level also increased. Wachs (1982) noted that children were slower to develop language skills in noisier homes. Matheson et al. (2003) noted similar results with low but statistically significant correlations between neuroendocrine tests, blood pressure measurements, and educational success.

Again, it should be stressed that presence of biologic measures, such as heightened hormone or blood pressure levels, does not necessarily relate to long-term clinical changes in a population. While these changes may have long-term effects, there is no current evidence to support this extrapolation.

NOISE STANDARDS AND THEIR HISTORY

The earliest regulations designed to protect workers' hearing from NIHL were adopted by the US armed forces as a result of the tremendous amount of NIHL suffered by US service members in World War II (Gasaway, 1985). The first recommended exposure limit was issued by the US Air Force (USAF) in 1948, followed by the first enforceable hearing conservation regulation (also by the USAF) in 1956 (Suter, 1988). The 1956 USAF regulation identified five aspects of hearing conservation which still form the basis of modern standards, that is:

- Noise reduction efforts
- Measurement of noise exposure
- Education of workers
- Use of hearing protection
- Audiometric surveillance

These requirements evolved from research and recommendations made by CHABA (Suter, 1988). After initial development by the armed forces, several groups, most notably the American Conference of Governmental Industrial Hygienists (ACGIH), established recommended exposure limits for the civilian workforce. In 1969, ACGIH issued a voluntary Threshold Limit Value (TLV) for noise that represented a greatly simplified version of the CHABA recommendations (Suter, 1988). In 1969, the TLV was adopted by the Occupational Safety and Health Administration (OSHA) under the Walsh-Healey Public Contracts Act, which applied to large federal contracts, and separately under the Federal Coal Mine Health and Safety Act (Suter, 1988). Then, in 1971, following the establishment of the OSHA, the Walsh-Healey exposure requirements were promulgated as a Permissible Exposure Limit (PEL) for noise in general industry and construction (Suter, 1988). This PEL remains in force today, and specifies a Time-Weighted Average (TWA) exposure limit (referred to as a Criterion Level, or L_C) of 90 dBA over an 8-hour workshift, with a 5-dB exchange rate (OSHA, 1981). The PEL requires that employers attempt to reduce noise exposures above 90-dBA TWA through noise controls, though subsequent OSHA policy interpretation effectively raised this level to 100 dBA. Workers exposed above the 90-dBA TWA limit must use hearing protection devices, and hearing protectors are further required for exposures that exceed 115 dBA for 1 second or more. OSHA also recommends hearing protectors for exposures above 140 dB SPL regardless of duration. To provide further protection to noise-exposed workers, OSHA promulgated the Hearing Conservation Amendment in 1983, which requires employers to provide baseline and annual hearing conservation training to workers exposed above an Action Level 85-dBA TWA, requires baseline and annual audiometric surveillance, and requires that workers exposed between 85 and 90 dBA

be offered hearing protectors. OSHA's Hearing Conservation Amendment does not apply to workers in a number of industries, including agriculture, construction, oil and gas extraction, and offshore marine work. Miners are covered by an essentially equivalent PEL administered by the Mine Safety and Health Administration (MSHA), railroad workers fall under a similar regulation administered by the Federal Railroad Administration, and offshore workers fall under the jurisdiction of the US Coast Guard, which administers, though rarely enforces, a similar regulation.

The National Institute for Occupational Safety and Health (NIOSH), established in 1971, is tasked with conducting occupational health and safety research and recommending best practice exposure limits (as compared to OSHA, which uses public rulemaking to set mandatory exposure limits, and therefore must include factors such as economic feasibility in their rulemaking efforts). In 1972, NIOSH established a Recommended Exposure Limit (REL) of 85-dBA TWA L_C with a 5-dB exchange rate (Suter, 1988). However, in 1998, NIOSH revised its REL to incorporate a 3-dB exchange rate, while retaining the 85-dBA exposure limit. This is consistent with the TLV for noise, which was updated to these same specifications in 1994. Both of these voluntary limits recommend that audiometry, noise controls, and use of hearing protection begin at TWA exposures of 85 dBA, and may therefore be considered more protective than the OSHA regulation. The US Department of Defense, as well as the USAF, US Army, and US Navy, have all moved to exposure limits that are consistent with the current NIOSH REL and TLV. Individual states in the United States can opt to have state OSHA programs that administer regulations at least as protective as those promulgated by federal OSHA. None of the state OSHA programs have PELs or hearing conservation requirements that differ considerably from the federal OSHA programs, though several states, including Washington and Oregon, extend coverage to industries such as construction and agriculture.

Noise regulations around the globe are much simpler to describe. Virtually every high-income country in the world, and many medium- and low-income countries as well, has adopted exposure regulations that specify an 85-dBA TWA L_C and 3-dB exchange rate. For example, these limits are required in countries within the European Union. Outside of the United States, only a handful of countries—including Brazil and Israel—use regulations consistent with the OSHA PEL, or a mix of the OSHA PEL and NIOSH REL (e.g., an 85-dBA TWA exposure limit combined with a 5-dB exchange rate). A summary of US and worldwide noise standards and regulations can be found at http://sitemaker.umich.edu/neitzel/files/hearing_loss_references.pdf.

In addition to these regulatory and voluntary occupational exposure limits, limits have been recommended for the protection of public health. Specifically, both the US Environmental Protection Agency (EPA, 1974), and World Health Organization (WHO, 1999) have recommended a 24-hour exposure limit of 70 dBA with a 3-dB exchange rate. This is equivalent to an 8-hour exposure at 75 dBA, with no noise exposure for the other 16 hours per day; note that this represents a strong and highly debatable assumption in modern societies. This 24-hour exposure limit is intended to protect against any hearing loss at 4000 Hz among any exposed individual, and can be considered truly "safe"—whereas many occupational exposure limits accept some level of excess risk of hearing loss (e.g., as many as one-third of workers with sound exposures at the OSHA PEL of 90-dBA TWA daily over a 40-year period are expected to sustain a material hearing impairment).

All noise regulations and standards specify—either implicitly or explicitly—methods to determine individual workers' noise exposures. Such determinations can be simple, as is the case when comparing a measured TWA exposure level for a worker to the relevant exposure limit. However, when noise measurements are made with a sound level meter and involve exposures to different noise levels for varying periods of time, it becomes necessary to convert these noise levels and durations into an accumulated personal noise dose. This is done by comparing the ratio of exposure time (C) at each given level to the allowable time (T) at that level, as shown in the equation below:

$$Dose\% = 100\ (C_1/T_1 + C_2/T_2 + \cdots + C_n/T_n)$$

Allowable times can be determined by referencing the relevant exposure standard: For compliance purposes, these times are located in Appendix A in the OSHA Noise Regulation (29 CFR 1910.95), whereas the NIOSH best practices recommendation can be found in Chapter 1 of the 1998 Criteria Document for Noise Exposure (DHHS/NIOSH report number 98–126, NIOSH, 1998). Example T values from each standard are based on the data in Table 32.2 presented earlier.

If a worker had an exposure of 4 hours at 95 dBA, 2 hours at 90 dBA, and 2 hours at 85 dBA over the course of a workshift, their OSHA dose would be computed as follows:

$$\begin{aligned} Dose\% &= 100\ (4/4 + 2/8 + 2/16) \\ &= 100\ (1.0 + 0.25 + 0.125) \\ &= 100\ (1.375) \\ &= 137.5\% \end{aligned}$$

For comparison purposes, the NIOSH dose for the same exposure would be 605%.

Allowable times that are not specifically listed in the relevant standard can be computed directly using the equation below:

$$T = 480 \text{ minutes}/2^{((Lp - LC)/ER)}$$

Where L_C is the criterion level, L_P is the measured SPL in dBA, and ER is the exchange rate (in dB).

The dose value resulting from a dosimeter measurement or computed using the equation above can be computed into a TWA value using the equation below:

$$TWA = (ER/\log 2) \times \log 10\ (D/100) + L_C$$

Where *ER* is the exchange rate, *D* is the dose, and L_C is the criterion level. In the case of the worker described earlier, the OSHA TWA (using a 5-dB exchange rate and 90-dBA L_C) would be 92.3 dBA, whereas the NIOSH TWA (using a 3-dB exchange rate and 85-dBA L_C) would be 92.8 dBA.

REFERENCES

Babisch W. (2002) The noise/stress concept, risk assessment and research needs. *Noise Health.* 4, 1–11.

Bronzaft AL. (1991) The effects of noise on learning, cognitive development, and social behavior. In: Fay TH, ed. *Noise and Health.* New York, NY: The New York Academy of Medicine; pp 87–92.

Bronzaft AL, McCarthy DP. (1975) The effect of elevated train noise on reading ability. *Environ Behav.* 7, 517–528.

Cohen S, Glass D, Singer J. (1973) Apartment noise, auditory discrimination and reading ability in children. *J Exp Soc Psychol.* 9, 422–437.

Cohen S, Weinstein N. (1981) Non-auditory effects of noise on behavior and health. *J Soc Issues.* 37, 36–70.

Davis J. (1985) Hard of hearing children in the schools. Seminar presentation in St. Cloud, MN.

DeJoy DM. (1984) The nonauditory effects of noise: Review and perspectives for research. *J Aud Res.* 24, 123–150.

Edmonds LD, Layde PM, Erikson JD. (1979) Airport noise and teratogenesis. *Arch Environ Health.* 34, 243–247.

Environmental Protection Agency. (1974) *Information on the Levels of Environmental Noise Requisite to Protect Public Health and Welfare with Adequate Margin of Safety: A report of the Environmental Protection Agency (EPA).* Washington, DC: US Environmental Protection Agency.

Gasaway DC. (1985) *Hearing Conservation: A Practical Manual and Guide.* Englewood Cliffs, NJ: Prentice-Hall.

Green KB, Pasternak BS, Shore RE. (1982) Effects of aircraft noise on reading ability of school-age children. *Arch Environ Health.* 37, 24–31.

Matheson MP, Stansfeld SA, Haines MM. (2003) The effects of chronic aircraft noise exposure on children's cognition and health: 3 field studies. *Noise Health.* 5, 31–40.

Nakamura R. (1977) Gestation in noise (Abstract). In: Toongsuwan S, Suvonnakoto T, eds. *Congress Handbook.* Bangkok: Seventh Asian Congress of Obstetrics and Gynecology.

Occupational Safety and Health Administration. (1981) Occupational noise exposure: Hearing conservation amendment. *Fed Reg.* 46, 4078–4179.

Ouis D. (2002) Annoyance caused by exposure to road traffic noise: An update. *Noise Health.* 4,69–79.

Pollak C. (1991) The effects of noise on sleep. In: Fay TH, ed. *Noise and Health.* New York, NY: The New York Academy of Medicine; pp 41–60.

Raymond LW. (1991) Neuroendocrine, immunologic, and gastrointestinal effects of noise. In: Fay TH, ed. *Noise and Health.* New York, NY: The New York Academy of Medicine; pp 27–40.

Ryals BM. (1990) Critical periods and acoustic trauma. In: *National Institutes of Health (NIH) Consensus Development Conference on Noise and Hearing Loss, Program and Abstracts.* Washington, DC: National Institutes of Health.

Schell LM. (1981) Environmental noise and human prenatal growth. *Am J Phys Anthropol.* 56, 156–163.

Sloan RP. (1991) Cardiovascular effects of noise. In: Fay TH, ed. *Noise and Health.* New York, NY: The New York Academy of Medicine; pp 15–26.

Smith EL, Laird DA. (1930) The loudness of auditory stimuli which affect stomach contractions in healthy human beings. *J Acoust Soc Am.* 15, 94–98.

Stansfeld SA, Haines MM, Brown B. (2000) Noise and health in the urban environment. *Rev Environ Health.* 15, 43–82.

Suter AH. (1988) The development of federal standards and damage risk criteria. In: Lipscomb DM, ed. *Hearing Conservation in Industry, Schools and the Military.* Boston, MA: College-Hill Press; pp 45–66.

Thiessen GJ. (1978) Disturbance of sleep by noise. *J Acoust Soc Am.* 64, 216–222.

Thiessen GJ. (1983) Effect of intermittent and continuous traffic noise on various sleep characteristics and their adaptation. In: Rossi G, ed. *Proceedings of the Fourth International Congress on Noise as a Public Health Problem (Turin).* Vol. 2. Milan, Italy: Edizioni Techniche a cura del Centro Ricerche e Studi Amplifon.

Thompson SJ. (1981) *Epidemiology Feasibility Study: Effects of Noise on the Cardiovascular System.* Washington, DC: United States Environmental Protection Agency.

Wachs TD. (1982) Relation of home noise-confusion to infant cognitive development. Paper presented at the Annual Meeting of the American Psychological Association. Washington, DC.

WHO. (1999) Guidelines for community noise. In: Berglund B, Lindvall T, Schwela DH, eds. Geneva: World Health Organization.

Recommendations and Counseling

The rehabilitative process begins at the end of the evaluation when the audiologist shares results, describes treatment options (using decision aids) and discusses patient preferences, so that an informed decision can be reached. Insuring that the patient and communication partner are involved in decisions regarding treatment options will promote compliance with recommendations. The patient's stage of readiness in combination with considerations regarding physical, sociologic, cognitive, and psychosocial status must inform the discussion. Integral to patient-centered care is that treatment options are concordant with the patient's values and motivations. The patient has come in with questions and should walk away with concrete answers, an action plan and a roadmap regarding next steps. The audiologist should attempt to determine stage of readiness (e.g., precontemplation, action) using simple questions such as "*I know I have a hearing problem, and I intend to take action to solve it soon*" as the response will inform the counseling sessions and decisions regarding targeted treatment interventions. Hence, an individual with a mild hearing loss with significant participation restrictions who experiences difficulty in a variety of listening situations and who is in the action stage of readiness might be a candidate for hearing aids; whereas someone with a mild hearing impairment with difficulty understanding the television may benefit form a conversation about devices for the television. It is very important that before the patient leaves, most questions are answered and they understand that the interventions available can help promote ease of listening, can improve enjoyment of activities important to them, and that it is beneficial to act early to insure that the consequences of hearing loss become an intolerable burden. When appropriate, and depending on auditory processing abilities it is wise to recommend an interactive computerized auditory-cognitive training program such as the Listening and Communication Enhancement (LACE) software to improve skills lost due to normal age-related change in such cognitive functions as executive control, speed of processing, and working memory. This "physical therapy for the ears" may be beneficial for many persons with ARHL including those not yet motivated or ready to purchase hearing aids, or people in whom hearing aids are not effectively compensating for their communication breakdowns (Sabes & Sweetow, 2007). To reiterate, basic to client-centered care is that the audiologist present intervention options and the person with hearing loss and their communication partner share in the decision-making process.

CASE PRESENTATION

Mr. R., an active 75-year-old executive, recently returned to work part-time after 1 year of full-time retirement from a family business. The case history revealed that he is in excellent health and enjoys being productive. He indicated that his work entails small group meetings with colleagues and that he has considerable difficulty when noise is present in the room and when he is sitting around large conference room tables. He also finds that he has more difficulty understanding female than male voices. Because of these problems, he scanned the internet for information on hearing loss and came to several websites that conducted hearing screenings. Mr. R. completed an online screening version of the HHIE. His hearing profile revealed a score of 20, suggesting a mild-to-moderate self-perceived hearing handicap indicative of necessity for a referral to a local audiologist. The site directed him to a list of audiologists in his geographic area. Mr. R. scheduled an appointment with the audiologist armed with considerable information about hearing loss and hearing aids and with questions regarding the virtues of hearing aid use.

The audiometric evaluation revealed that, indeed, Mr. R. had mild bilateral sensory/neural hearing loss in both ears with excellent word recognition ability, which belied his experiences in the real world. The audiologist administered the QuickSIN and scores revealed a moderate SNR loss. Responses to the HHIE-S revealed a score of 22, suggesting a mild handicap. Questioning regarding Mr. R.'s social engagement revealed that recently, he was reluctant to engage in previously enjoyed activities given his struggle understanding others. The PHQ-9 was administered to screen for depressive symptoms and a score of 9 emerged which is consistent with mild depression. The MMSE score was 24, consistent with mild cognitive impairment. In light of the latter test results, the patient was referred back to his primary care physician and it was noted that the untreated hearing impairment may be a variable contributing to his scores on the MMSE and PHQ-9.

The audiologist suggested to his physician that Mr. R. might benefit from bilateral digital hearing aids given his desire to remain socially connected and his frustrations communicating with friends, colleagues, and family members. Mr. R.'s physician administered the PHQ-9, the MMSE, and the deJong Giervald short scales of emotional and social loneliness and responses suggested that Mr. R.

is feeling lonely and socially disconnected. The possibility of an antidepressant was discussed as was possible auditory interventions such as amplification with hearing aids. Mr. R. was reluctant to take medication and decided to try hearing aids to see if in fact his recent feelings of social disconnectedness were linked to his difficulty understanding others. Mr. R. returned to the audiologist with his wife. Mr. R. decided to purchase hearing aids for a 1-month trial and agreed to use the online version of the Listening and Communication Enhancement (LACE) software to improve listening skills and to foster improved auditory processing (Sabes & Sweetow, 2007).

At the 3-week postfitting appointment, verification and validation studies were conducted including perceived ease of listening studies in different noise backgrounds. Audibility of speech was high according to score on the Speech Intelligibility Index (SII) verifying that speech was audible and usable with his hearing aids (Hornsby, 2004). HHIE scores revealed a significant reduction in self-perceived handicap with aided scores improving to 6, suggesting with 95% confidence that this was a true change in the HHIE score from the unaided condition. Score on the MMSE and PHQ-9 improved slightly which was of interest and responses to the loneliness scale improved, as well.

Mr. R. completed the HHIE 3 months after the fitting and continued to be deriving considerable benefit from the hearing aids. Tracking of scores on the LACE at the 3-month postfitting revealed improved scores on each of the subscales relative to baseline. This case was of interest because he presented with a mild hearing impairment yet expressed communication difficulties and frustrations when communicating; this suggested challenges beyond what could have been predicted from basic pure-tone testing. The more thorough examination confirmed the speech understanding difficulties in adverse listening situations and some psychosocial sequelae. His physician noted a definite improvement in Mr. R.'s affect and as an aside he commented that it was much easier to communicate with him when he was wearing his hearing aids. The comprehensive client-centered examination and partnering with the primary care physician enabled Mr. R. to obtain the assistance he needed and, more importantly, to satisfy the communicative needs presented by his particular lifestyle. He is now a strong advocate for hearing aids and has been instrumental in helping some of his friends purchase hearing aids. He also is an advocate for use of LACE in combination with hearing aids as he attributes his improved ease of listening in noisy situations to the exercises he did with the LACE online.

REFERENCE

Sabes J, Sweetow R. (2007) Variables predicting outcomes on listening and communication enhancement (LACE). *Int J Audiol*. 46, 374–383.

Iowa Tinnitus Activities Questionnaire

IOWA TINNITUS ACTIVITIES QUESTIONNAIRE MAY 05

Name: **Date:**

Please indicate your agreement with each statement on a scale from 0 (completely disagree) to 100 (completely agree).

#	Statement	0–100
1.	My tinnitus is annoying.	
2.	My tinnitus masks some speech sounds.	
3.	When there are lots of things happening at once, my tinnitus interferes with my ability to attend to the most important thing.	
4.	My emotional peace is one of the worst effects of my tinnitus.	
5.	I have difficulty getting to sleep at night because of my tinnitus.	
6.	The effects of tinnitus on my hearing are worse than the effects of my hearing loss.	
7.	I feel like my tinnitus makes it difficult for me to concentrate on some tasks.	
8.	I am depressed because of my tinnitus.	
9.	My tinnitus, not my hearing loss, interferes with my appreciation of music and songs.	
10.	I am anxious because of my tinnitus.	
11.	I have difficulty focusing my attention on some important tasks because of tinnitus.	
12.	I just wish my tinnitus would go away. It is so frustrating.	
13.	The difficulty I have sleeping is one of the worst effects of my tinnitus.	
14.	In addition to my hearing loss, my tinnitus interferes with my understanding of speech.	
15.	My inability to think about something undisturbed is one of the worst effects of my tinnitus.	
16.	I am tired during the day because my tinnitus has disrupted my sleep.	
17.	One of the worst things about my tinnitus is its effect on my speech understanding, over and above any effect of my hearing loss.	
18.	I lie awake at night because of my tinnitus.	
19.	I have trouble concentrating while I am reading in a quiet room because of tinnitus.	
20.	When I wake up in the night, my tinnitus makes it difficult to get back to sleep.	

Scoring

Area	Questions	Score
Emotions and Thoughts	1, 4, 10, 12	%
Hearing and Communication	2, 6, 14, 17	%
Sleep	13, 16, 18, 20	%
Concentration	3, 7, 11, 15, 19	%
Total		%

Tyler RS, Gehringer AK, Noble W, Dunn CC, Witt SA, Bardia A. (2006) Tinnitus activities treatment. In: Tyler RS, ed. Tinnitus treatment: Clinical Protocols. New York: Thieme Medical Publishers.

Loudness and Annoyance of Everyday Sounds

Some everyday sounds are loud and some are soft. Some everyday sounds are annoying and some are not. Please rate the **loudness** and the **annoyance** of the following sounds. Do not consider the annoyance when rating the loudness and do not consider the loudness when rating the annoyance. For example, a sound may be very loud, but not annoy you. Likewise, a sound may be very soft, yet be very annoying. Rate the sounds using a scale from 0 (not loud/annoying) to 100 (unbearably loud/annoying).

	Sound	Loudness [0–100]	Annoyance [0–100]
1.	Standing next to a dog barking		
2.	Someone stacking dishes in the same room		
3.	Hearing music on the radio in a car when the volume is adjusted for normal-hearing listeners		
4.	Hearing music on the radio in a quiet room when the volume is adjusted for normal-hearing listeners		
5.	Telephone ringing in the same room		
6.	Television in the same room when the volume is adjusted for normal-hearing listeners		
7.	Standing next to a lawnmower		
8.	Standing next to a car door closing		
9.	Talking with someone in a noisy restaurant		
10.	Baby crying in the same room		

Tyler RS, Bergan C, Preece J, Nagase S. (2003). Audiologische Messmethoden de Hyperakusis. In: Nelting M, ed. *Hyperakusis* (39–46). Stuttgart: Georg Thieme Verlag.

The Relative Handicap of Hearing Loss, Tinnitus, and Hyperacusis

The following questions relate to hearing loss, tinnitus, and hyperacusis. Hyperacusis is either when sounds that are moderately loud for other people are *too* loud for you or when you find sounds annoying. Please rate your agreement/disagreement with the following statements, using a scale from 0 (completely disagree) to 100 (completely agree).

		Because of Your Hearing Loss (0–100)	Because of Your Tinnitus (0–100)	Because Some Sounds are too Loud or Annoying (0–100)
1.	You avoid shopping			
2.	You do not go out with your friends			
3.	You have given up some hobbies			
4.	You do not go to restaurants			
5.	You avoid being in crowds			
6.	You feel depressed			
7.	You feel anxious			
8.	You are not able to concentrate			
9.	Your quality of life is poor			
10.	You are not able to perform tasks or jobs as well			

Tyler RS, Bergan C, Preece J, Nagase S. (2003). Audiologische Messmethoden de Hyperakusis. In: Nelting M, ed. *Hyperakusis* (39–46). Stuttgart: Georg Thieme Verlag.

AUTHOR INDEX

Aaron, R., 801
Abaya, H., 585
Abbas, P.J., 197, 829
Abdala, C., 363, 364, 372
Abdala de Uzcategui, C., 846
Abrahams, Y., 670
Abrams, D.A., 539–540
Abrams, H., 803
Abrams, H.B., 68, 649, 858
Abrisch, A., 844, 846
Academy of Doctors of Audiology, 808, 814
Achenbach, T.M., 846
Achor, J., 231, 239
Achor, L.J., 253
Acoustical Society of America (ASA), 10, 78, 79, 81, 90, 91, 92, 104
Adamiec, L.C., 42
Adams, J.C., 357, 367
Adappa, V., 752
Adelman, C., 52
Adler, L.E., 329
Adukna, O., 823
Adunka, O.F., 56
Agnew, J., 762
Ahad, P., 534, 541, 586
Ahissar, E., 540
Ahissar, M., 535, 540
Ahlfors, S.P., 317
Ahmed, Z.M., 484, 488–489, 495
Ahn, B.H., 385
Aiken, S.J., 167, 518
Ainsworth, J., 104
Aithal, S., 159
Aitkin, L.M., 516
Aizawa, N., 520
Akdas, F., 587
Akin, F.W., 418
Alaerts, J., 286
Alain, C., 517
Alberta Health Services, 772
Alberti, P., 625
Albright, K., 443
Alexander, G., 669
Alexander, G.C., 69, 783
Alexander, J.M., 764
Alexander, N.B., 422
Alford, B., 172, 177, 183
Alford, B.R., 175
Alho, K., 330
Alku, P., 532
Allen, A.R., 253
Allen, J.B., 155
Allen, L., 505
Allen, M.L., 618
Allen, P., 634
Allison-Levick, J., 148
Alpiner, J., 856
Alpini, D., 418
Alsalaheen, B.A., 417
Althen, H., 326
Amatuzzi, M., 220, 227
Amenedo, E., 324
American Academy of Audiology (AAA), 3, 6, 9, 147, 148, 312, 443, 447, 450, 576, 682, 684, 686, 759, 761–765, 767, 771, 772, 805, 841
American Academy of Neurology, 422
American Academy of Otolaryngology-Head and Neck Surgery (AAO-HNS), 310
American Academy of Pediatrics (AAP), 120, 131, 443, 444, 448, 450
American Chart Company, 32
American College of Medical Genetics, 443
American College of Nurses-Midwives, 444–445
American Geriatrics Society, 643
American Medical Association, 18
American National Standards Institute (ANSI), 9, 11, 19, 32, 33, 34, 36, 38, 45, 50, 57, 61, 62, 78, 79, 81, 90–92, 104, 146, 167, 168, 604, 612, 678, 714, 730–732, 735, 736, 755, 768, 770, 780, 784, 785, 811, 841
American Psychiatric Association, 585, 588
American Speech-Language Hearing Association (ASHA), 20, 35–37, 40, 45, 50, 61–65, 71, 81–83, 85, 86, 98, 100, 101, 107, 120, 146, 147, 169, 312, 437, 443, 664, 802, 803, 805, 814, 850
American Telemedicine Association, 659
Amlie, J.P., 492
Amlie, R.N., 257
Ammana, H.R., 489
Amsel, L., 636
Andaz, C., 627
Anderson, G., 650
Anderson, H., 53, 92–94, 99, 103, 183, 306
Anderson, J.S., 249
Anderson, K., 503
Anderson, K.L., 688, 843
Anderson, L.G., 71
Anderson, S., 325, 531, 537
Anderson, T.J., 427
Andersson, G., 669, 670
Andrews, P.C., 752
Andrus, J.N., 167, 521
Angelaki, D.E., 386
Angel, C.R., 429
Anson, B.J., 160
Anson, S., 583
Anstey, K., 635
Anthony, L., 176, 177
Antia, S.D., 503
Antonelli, A., 362
Antonelli, A.R., 234
Aoyagi, M., 274, 281, 282
Aragon, M., 843, 844
Arakawa, E., 386
Araki, S., 243
Aran, J.M., 207, 215
Arbogast, J., 752
Arcaroli, J., 828
Arehart, K.H., 159, 160, 605, 606
Arehole, S., 329
Arlinger, S., 678, 719
Arnce, K.D., 44
Arnedt, C., 832
Arnett, J., 489
Arnold, C.L., 452
Arnold, M., 853, 858
Arnos, K.S., 477, 496
Arnott, S.R., 517
Arriaga, M.A., 172, 183, 232, 417
Arroyo, C., 325
Arslan, E., 207–228, 245
Arts, H.A., 306, 832
Asai, H., 218
Asan, 489
Ashbaugh, C., 832
Asher, W.J., 62
Ashmead, D.H., 589, 592
Ashmore, J.F., 357
Askew, J.W., 488
Aso, S., 209
Assaf, Y., 520
Assmann, P.F., 535
Astuto, L.M., 488
Atlas, M., 700
Atlas, M.D., 182, 662, 664, 669
Attias, J., 56
Aung, M.H., 235
Auriemmo, J., 764
Aussilloux, C., 585
Austen, S., 618
Australian Government Department of Health and Aging, 664
Avraham, K.B., 485
Aw, S.T., 403

Babbidge, H., 442
Babcock, M., 750
Babin, R.W., 647, 651, 652
Baca, R., 844, 846
Bachmann, K.R., 120
Bachman, R., 143
Badie, B., 306
Bagatto, M., 715, 761, 764–766, 768–770, 796, 797
Bagatto, M.P., 260, 767, 769, 772, 773
Baggs, T., 806
Baghdadli, A., 585
Baguio, F., 585
Baguley, D., 177
Bailey, H.A.T., 99
Bailey, H.A.T. Jr, 618
Bailey, K.A., 650
Bainbridge, K., 631
Baio, J., 585
Bakalov, V.K., 494
Baker, D., 585, 642
Baker, L.J., 649
Bakkouri, W.E., 233
Balaban, M., 172
Balch, D., 668
Baldwin, M., 148, 439–440
Baldwin, S.M., 120
Bale, J.F., 583
Balkany, T., 831
Balkany, T.J., 172, 465
Ballachanda, B., 633
Ballad, W.J., 760
Balog, J.Z., 493
Baloh, R.H., 412
Baloh, R.W., 381, 383–387, 389, 413
Balow, B., 468
Bamford, J., 69, 71
Bamford, J.M., 71
Banai, K., 325, 527, 532, 537–539
Bance, M., 167
Banerjee, A., 182
Banerjee, S., 69, 532, 720
Bankaitis, A.U., 728
Banoub, M., 299, 300, 307
Bara, D., 449
Barajas, J.J., 327
Baran, J., 330, 571
Baran, J.A., 231–246
Barany, E., 51
Barber, H.O., 235, 386, 411
Bardia, A., 650, 652
Barelli, P.A., 617
Barin, K., 385
Barker, D.H., 846
Barley, M., 855
Barmack, N.H., 392
Barney, H.L., 533–534
Barr, B., 53, 183
Barton, M.L., 586
Bartoshesky, L., 497
Bartosiewicz, C., 262
Barwick, K., 771
Basar, E., 318
Bassim, M.K., 232, 233
Bass, P.F., 452
Bass-Ringdahl, S., 148, 465
Bateman, D.E., 56
Bates, E., 845
Bathen, J., 492
Batra, R., 249
Battini, R., 583
Battmer, R., 172
Bauch, C.D., 251
Baughn, W.L., 599
Bauman, S., 504
Baumen, M., 243
Baum, J., 583
Bayley, N., 468
Bayliss, K., 238
Beagley, H., 224
Bear, M.F., 519
Beath, K., 632, 636
Beattie, C., 177
Beattie, R.C., 66, 68, 81
Beatty, J.D., 385
Beauchaine, K.A., 254, 258, 259
Bebin, J., 233
Beck, D.L., 113–117
Bederson, J.B., 233
Beer, D.E., 41, 262
Beers, A.N., 157, 159
Behl, D., 670
Behl, D.D., 453
Behrend, O., 516
Behrens, T.R., 440, 442
Beitel, R., 517
Bekesy, G., 51
Belger, N., 329
Belin, P., 534, 536, 541
Belkahia, A., 477
Bell, D., 146, 157
Bell, D.W., 62, 67
Bellis, T., 571, 574
Bellis, T.J., 520
Bellon-Harn, M., 574
Bellotto, R., 234
Ben Arab, S., 477
Bench, J., 69, 71
Benna, P., 197
Bennett, K., 561
Bennett, M., 148
Bennett, M.J., 170
Bennett, R., 676
Bennett, R.L., 481
Benninger, M.S., 232, 246
Benson, R.W., 62, 107
Bentler, R., 718, 730, 735, 740
Bentler, R.A., 717, 718, 766, 767
Bentley, B., 585
Ben-Yosef, T., 496
Beranek, L.L., 10
Berg, A.L., 262
Bergamasco, B., 197
Bergemalm, P.O., 243
Berger, E.H., 43, 80, 94, 595, 606, 609
Bergmans, J.A., 303
Berlin, C., 190
Berlin, C.I., 72, 120, 179, 207, 254, 257, 261, 262, 358, 451, 465, 488, 521
Berrettini, S., 583
Berrett, M.V., 78, 79, 80, 94
Berrittini, S., 842
Berry, G.A., 81, 86, 91, 99, 101, 103, 104
Bertelli, M., 585

Bertone, A., 586
Bertrand, J., 585
Bertrand, O., 326, 327
Besculides, M., 447–449, 448, 449
Bess, F., 676, 680
Bess, F.H., 66, 71, 72, 437, 442, 586, 587, 588, 592
Besson, M., 534
Bestelmeyer, P.E., 541
Beswick, R., 448
Betsworth, A., 72
Bettger, J.P., 448
Betz, J., 636
Beyea, S.D., 520
Bhatia, P., 448
Bhattacharyya, N., 399
Biagio, L., 667
Bianco, C., 197
Bielefeld, E.C., 596, 602–604
Bilger, R.C., 65, 360
Billings, C., 345, 349, 350
Billings, C.J., 350, 351
Binnie, C., 855, 856
Binns, M.A., 517
Binzer, S., 801
Birck, J.D., 71
Birkenshaw-Fleming, L., 730
Bischoff, A.M., 487
Björkman, G., 139
Black, F.O., 429, 431
Blackwell, K.L., 80
Blair, E., 307
Blair, R.L., 245
Blaiser, K., 771
Blake-Rahter, P., 687, 691
Blalock, L., 841
Blanchard, K.B., 839
Blanchard, S., 477
Bland, L., 120
Bleich, N., 198
Block, M.G., 167, 169, 170
Bloomberg, J., 427
Bloom, M.J., 307
Bluestone, C.D., 143, 144, 145, 148
Blumenfeld-Katzir, T., 520
Blythe, M.E., 81, 82, 86
Bobinski, M., 814
Bocca, E., 72
Bocchini, A., 452
Bocchini, J.A., 452
Boettcher, F.A., 274, 598, 603, 604
Bohmer, A., 419
Bohning, S., 159, 160
Boismier, T., 422
Bom, S.J., 487
Bondurant, L.M., 120
Bondy, C.A., 494
Bonen, L., 385
Bonfils, P., 364
Boothroyd, A., 40, 57, 66, 67, 71, 677, 679, 700
Bordelon, J., 254, 262
Boretzki, M., 764
Borg, E., 165, 166, 167, 171, 197, 243
Bork, J.M., 488
Bork, K., 146, 157
Born, J., 651
Bornstein, S.P., 72, 242
Borsad, N., 491
Borton, T.E., 232
Bottomy, M.B., 303
Bougatsos, C., 453
Bouscau-Faure, F., 654, 655
Boutros, N.N., 317, 324, 326, 329
Bove, F., 585
Bowen, C.V., 520
Bower, D., 72
Bower, D.R., 34
Boyages, S., 635
Boyd, C., 642
Boyev, K.P., 312
Boyko, E., 642
Boyle, C., 585
Boyle, W.F., 32
Boys Town National Research Hospital, 761, 762
Brackmann, D., 237
Brackmann, D.E., 199, 200, 231, 234, 235, 245, 251, 306
Bradley, J., 677, 679
Bradley, J.S., 676
Bradlow, A., 576–577, 578, 579
Bradlow, A.R., 521, 537
Brady, G., 813
Brady, S., 538
Braida, L., 691
Brand, A., 516
Brandt, T., 429
Brandy, W.T., 72
Brask, B.H., 585
Brask, T., 166
Brass, D., 360
Brattico, E., 533
Bray, C.W., 207
Bregenzer, N., 419
Brennan, M.A., 764
Breslin, M., 605
Brewer, C., 493
Brewer, C.C., 487–488, 495
Brewer, K.D., 520
Brewer, S., 766, 791, 796
Brey, R.H., 416
Brigell, M., 320
Bright, K.E., 360, 364
Brightwell, A.P., 56
Brine, M., 401
Brinsko, K.M., 182
Britton, L., 767
Brockett, J.E., 158
Brody, D., 508
Brody, H., 635
Brokx, J.P., 767
Brons, I., 717
Brooks, D.N., 143
Brown, A., 843
Brown, A.M., 357, 358, 363, 364, 367
Brown, A.S., 837, 847
Brown, C.J., 238, 251, 252, 280, 282, 352, 829
Brownell, R., 846
Brownell, W.E., 357
Browning, G.G., 31
Brown, S., 521
Brown, W.S., 487
Bruce, E.N., 413
Brugge, J.F., 516, 523
Brungart, D.S., 70
Brunskill, E., 664
Brunt, M., 503
Bryan, M.F., 717
Bryant, K., 144, 148
Buchman, C.A., 56, 238, 828, 832, 833
Buchwald, J., 187, 323, 324, 326
Buchwald, J.S., 231, 323
Buckley, K., 832
Budenz, C.L., 832
Buhrman, M., 670
Buhrmann, J., 448
Buitelaar, J.K., 586
Bukhari, I., 488
Bulen, J.C., 155, 159, 160
Bull, J., 197
Bullock, T.H., 318
Burge, D.M., 56
Burkard, R., 187–204, 241, 264, 268, 647, 654
Burkard, R.F., 338, 349
Burkhalter C.L., 841
Burkhard, M.D., 17
Burkhart, C.G., 752
Burkhart, C.N., 752
Burk, M.H., 534
Burks, C.A., 68, 69
Burlutsky, G., 636
Burney, B., 120
Burney, L., 621
Burns, E.M., 159, 160, 360, 598, 600
Burns, P., 92, 93, 94, 105
Burns, W., 596
Busby, P.A., 828
Bush, M.L., 236
Buss, E., 828
Butler, E.C., 92, 93, 94, 105
Butman, J.A., 487, 493
Butt, S., 172
Butts, S.L., 172
Buzsáki, G., 316
Byrne, D., 720, 735, 766
Byun, Y., 627

Cacace, A., 637, 638
Cacace, A.T., 318, 320, 324, 325, 647
Cacioppo, J., 631
Caissie, R., 686
Calaero, C., 72
Calandruccio, L., 144
Call, E., 590
Callø, J., 202, 203
Cama, E., 223
Cambron, N.K., 152, 154
Cameron, C., 764
Camiolo-Reddy, C.E., 417
Campbell, A.P., 56
Campbell, J. A., 319
Campbell, J.C., 72
Campbell, R.A., 623, 626
Campbell, V., 631
Campos, P.D., 668, 669
Canalis, R.F., 387, 391, 393
Caner, G., 172
Canfield, M., 588
Canlon, B., 634, 635
Cantekin, E.I., 38, 43
Caputo, D., 418
Caram, P., 104
Caraway, T., 764
CareerCast.com., 805
Carey, A., 846
Carey, J., 636
Carey, J.C., 484
Carey, J.P., 55, 182
Carhart, R., 18, 44, 54, 61, 65, 67, 68, 70, 620, 854
Cariani, P., 517–518, 530–531, 535
Carlisle, E.W., 239, 242
Carney, E., 38, 40, 43
Carney, L.H., 513–514
Carpenter, M., 167
Carpenter, M.B., 315
Carrell, T.D., 12, 538
Carr, G., 837, 847
Carrier, D., 232
Carroll, B., 812
Carrow-Woolfolk, E., 846
Caruthers, S.K., 418
Cascio, C.J., 586
Casey, K.A., 284
Cashman, M.Z., 235
Cason, J., 670
Caspary, D., 633–635
Cassinari, V., 72
Cass, S.P., 404, 417
Castelein, C.M., 484, 489
Castello, F.V., 586
Castro, S.L., 534
Caudle, S.E., 585
Cauley, J., 631, 632, 635
Cavanaugh, R., 148
Cawkwell, S., 40, 57
Cawthorne, T., 427, 429
CCSSI, 571
Cecola, R.P., 262
Celesia, G.G., 320, 324
Cenci, K.A., 417
Centers for Disease Control and Prevention (CDC), 399, 441, 444, 445, 449
Central Institute for the Deaf (CID), 105
Cesarani, A., 418
Cetas, J.S., 317
Cha b, H., 488
Chaiklin, J.B., 32, 41, 79, 80, 81, 94, 620–621
Chai, T.C., 753
Chai, T.J., 753
Chalupper, J., 764
Chambers, J.A., 57, 62, 64, 65, 81, 82, 104, 109, 622, 626
Chambers, R.D., 324
Champagne, S.C., 261
Champlin, C.A., 41, 57, 62, 64, 65, 81, 82, 104, 109, 621–623, 626
Chandler, J.R., 33
Chandrasekaran, B., 325, 338, 540
Chang, S., 669
Chang, W-P, 319
Chardenoux, S., 488
Charlet de Sauvage, R., 207, 215
Charlip, M., 675
Chase, S.G., 303
Chasin, M., 605, 609, 750
Chatrian, G.E., 322, 323
Chatterjee, R., 448
Chawarska, K., 585
Chays, A., 517, 520
Cheatham, M.A., 208, 209, 521
Chee, N.W., 405
Chenausky, K., 845
Chen, C., 320
Chen, D., 636
Chen, D.A., 417
Cheng, P.W., 418
Chermak, G.D., 243
Chermak, G. D., 571, 574
Cherry, E.C., 68
Chertoff, M., 215
Chial, M.R., 68
Chiappa, K.H., 235, 239
Chien, W.W., 55
Chilosi, A.M., 583
Ching, T., 766, 791, 796
Ching, T.C., 842
Ching, T.Y., 766, 767
Ching, T.Y.C., 764, 766–768
Chin, J., 638
Chiou, K., 717
Chisin, R., 307
Chisolm, T., 803, 853, 858
Chisolm, T.H., 68, 858
Choi, B.Y., 493
Choi, J.Y., 831
Choi, K.D., 405
Choo, D., 827, 842
Choo, Y.B., 54
Chordekar, S., 52
Chowdhry, A.A., 488
Christensen, E., 767
Christine, A., 675
Chung, K., 719, 743, 751
Cincinnati Children's Hospital Medical Center, 762
Çiprut, A., 587
City University of New York (CUNY), 67
Claes, C., 489
Clarey, J.C., 268
Clarke, S., 315
Clark, J.G., 6, 627, 851
Clark, J.L., 62, 662
Clark, M.S., 828
Clayton, M.I., 41, 54
Clayworth, C.C., 324
Clemis, J., 172, 177, 183
Clemis, J.D., 166, 760
Clemson, L.M., 399
Clench, D.A., 767, 772, 773
Clinard, C., 633

Coats, C.A., 175
Cobb, J., 635
Coelho, C., 647, 649, 653
Cohen, B., 390, 416
Cohen, H.S., 427, 429
Cohen, L.T., 274, 282
Cohen, M., 181
Cohen, N.L., 232, 827, 831, 832
Cohen, S., 589
Cohn, E., 488
Cohn, E.S., 157, 159
Colebatch, J.G., 418
Coleman, J.J., 763
Coles, R.R.A., 79, 86, 99, 103
Cole, W.A., 768
Collet, L., 358, 517, 520
Colletti, V., 149, 150
Collins, M., 642
Comeau, M., 521
Committee on Hearing and Equilibrium, 42
Comparini, A., 583
Condon, M.C., 469
Cone-Wesson, B., 41, 256, 262, 282, 284, 286, 349
Coninx, S., 842
Conn, M.J., 64
Connor, N.P., 687
Connors, B.W., 519
Conroy, P.J., 753
Conti, G., 207
Convery, E., 721
Cook, I.C., 585
Cooksey, F.S., 427
Coombes, S., 168
Cooper, W.A., 17
Cooper, W.A. Jr, 625
Corbett, A., 238
Cord, M., 720
Cord, M.T., 717
Corey, D.P., 383
Corfits, J., 197, 254
Corliss, E.L.R., 17
Cornelisse, L., 765, 766, 796, 797
Cornelisse, L.E., 766, 767
Cornwell, E., 632, 638, 639
Cortopassi, G.A., 484
Costa, P., 197
Costello, J.A., 307
Cotanche, D.A., 603
Couch, S., 586, 587
Coulter, D.K., 249, 459, 835
Counter, S.A., 167, 243
Coverstone, J., 814
Cowie, C., 631
Cox, C., 605
Cox, L., 197
Cox, R., 17, 669, 719
Cox, R.M., 69, 735, 762, 783
Coyne, J., 642
Crandell, C., 578, 579, 675–678, 680, 682, 686, 691, 782
Creeke, S., 45, 662
Creighton, J., 842
Cremer, P.D., 428
Cremers, C.W., 489, 818
Cremers, C.W.R.J., 764
Cremers, F.P., 489
Creten, W.L., 139, 151
Creutzfeldt, O., 531, 536
Crews, J., 631
Criado, J.R., 347
Cristobal, R., 583
Crocker, S.R., 653
Cronau, L., 197
Crotti, L., 492
Croutch, C., 53
Crowe, K., 767
Crow, G., 543
Crozier, S., 536
Cruickshanks, K., 637, 638
Cruickshanks, K.J., 142, 144
Crukley, J., 764, 767, 771, 772
Crum, M.A., 181
Crump, B., 120, 621
Cry, E., 465
Culbert, N., 439
Cullen, K.E., 386
Culpepper, B., 471
Cumming, R.G., 632, 636
Cummings, C.W., 31, 41
Cunningham, J., 521
Curhan, G.C., 508
Curhan, S.G., 508
Curry, J.M., 497
Curthoys, I.S., 403, 428
Cushing, E.M., 598, 600

DÆArcy, R.C.N., 520
Dagbjartsson, A., 583
D'Agostino, R., 633, 635
Dale, P.S., 845
Dallos, P., 188, 208, 209, 357
Dal Monte, E., 220
Dalpes, M., 844, 846
Dalton, D., 638
Daly, D.D., 235
Daly, D.M., 235
Dancer, J., 64
D'Angelo, W.R., 249
Danhauer, J.L., 67
Daniel, H.C., 649
Darley, F.L., 437
Daroff, R.B., 429
Dauman, R., 327, 647, 654, 655
Dau, T., 202, 254, 255
Davalos-Bichara, M., 636
Davies, D., 141, 144
Davies, H., 605
Davies, R., 405
Davis, A., 647, 670
Davis, H., 63, 65, 72, 81, 107, 188, 198, 249, 251, 252, 258, 357, 437
Davis, J., 493
Davis-O'Leary, L.L., 427
Davis, R.R., 484
Davis, T.C., 452
Dawson, G., 585
Dawson, G.D., 318
Day, J., 767
Dean, J., 584
Dean, M.S., 41, 94
Deavenport, A., 448
Debevc, M., 695
Decker, T.N., 10, 12
Declau, F., 485, 486
Decoufle, P., 585
Dees, T., 536
DeFilippo, C.L., 856
Degirmenci, S., 587
de Jonge, R., 143
de Jong, M., 52
Dekok, K., 767
De La Cruz, A., 56
Del Castillo, I., 220, 221, 223
De Leenheer, E.M., 486
Deletis, V., 296
Delgado, R.E., 241, 261
Delgutte, B., 516–517–518
Deluca, A., 487
Demiris, G., 700
Demorest, M.E., 783
Denenberg, L.J., 78–82, 99
Denoyelle, F., 488
Densert, B., 218
Dent, K.M., 460
Den, Z., 262
Department of Veterans Affairs, 63
DesGeorges, J., 760, 836, 844
Devine, O., 443
de Wolf, M.J., 818
deWolf, M.J.F., 764
D'Haese, P., 842
Dhar, S., 625
Diamond, B.E., 262
Diaz, F., 324
Dickins, J.R., 303
DiClemente, C., 852
Diefendorf, A.O., 466, 589
Dierking, D.M., 465
Diesch, E., 324, 533
DiGiovanni, D., 99
DiGiovanni, J.J., 169
Dillard, R.G., 583
Dille, M.F., 588, 590
Dillon, H., 681, 697, 720, 721, 735, 766–769, 777, 783, 791, 796
Dimitrijevic, A., 280, 284–286, 327, 464
Dinces, E.A., 42, 232
Ding, D., 43, 208
Dingle, R.N., 514, 516, 518, 522
Dinkes, R., 636
Din, N., 578
Dirks, D., 34, 36, 40, 54, 55, 58, 72, 107
Dirks, D.D., 34, 63, 65, 68, 82, 94, 99
Disher, M.J., 303
Dittberner, A.B., 717
Diviani, N., 671
Dixon, R.F., 618
Dobie, R.A., 274, 277, 278, 282, 324
Dobson, D., 307
Dockray, J., 148
Dodds, L.W., 357
Doerfler, L.G., 626
Doernberg, N., 585
Dohlman, G.F., 384
Doi, K., 218
Dolan-Ash, S., 172
Dolu, N., 329
Donaldson, A.I., 827, 832
Donaldson, I., 833
Donaldson, J.A., 160
Donatelli, A., 833
Donchin, E., 197
Don, M., 187–204, 237, 250, 251, 253, 254, 255, 257, 262
Doorenbos, A., 700
Dorman, M.F., 352, 538, 540, 828
Dorn, P.A., 364, 367–369, 465
Douglas, J.E., 169
Douniadakis, D., 619
Dowell, R.C., 41, 72, 262
Downs, D.W., 181
Downs, M.P., 38, 441–442
Doxey, D.D., 431
Doyle, DL., 481
Doyle, P.J., 68
Doyle, T.N., 44
Drake, A., 146
Dran, J., 631
Dreisbach, L.E., 372
Dreschler, W.A., 717, 721, 740
Driscoll, C., 159, 439, 448, 831
Driscoll, D.P., 43, 595
Dror, A.A., 485
Drotar, D., 839
Drullman, R., 286, 539
Dublin, W.B., 237, 242
Dubno, J., 637, 638
Dubno, J.R., 25, 44, 68, 125
Dudley, A.W., 303
Dudley, B., 63, 64
Dudulao, M., 813
Du Feu, M., 846
Dun, C.A., 818
Dunlop, J., 132, 477
Dunn, A., 772
Dunn, C.C., 650, 652
Dunn, D., 805
Dunn, H.K., 80, 91, 735
Duquesnoy, A., 681
Durch, J.S., 606
Durieux-Smith, A., 198
Durrant, J.D., 208, 218, 243, 244, 385
Dutia, M.B., 386
Dyrlund, O., 717, 720
E-A-R Auditory Systems, 79, 80
Earl, L., 633
Eaton, C., 638
Eaton, D.A., 753
Eaton, S.B., 592
Eavey, R., 508
Eavey, R.D., 148, 477
Ebinger, K.A., 592
Eby, T.L., 160, 232
Eckert, M.A., 42
Eddins, D., 634
Edelstein, D.R., 56
Edgerton, B.J., 66, 67, 81
Edlefsen, L.L., 651
Edmondson-Jones, M., 520
Edwards, B., 718
Edwards, B.M., 296, 297, 306, 310, 491
Edwards, C., 682, 691
Efrati, M., 56
Egan, J.P., 65, 66
Egge, J.L., 717
Eggermont, J.J., 197–200, 202, 207, 209, 211, 215, 218, 237, 251, 254, 257, 329, 337, 517, 520
Eichler, J.A., 38, 43
Eichwald, J., 438, 460
Eichwald, J.E., 440
Eickhoff, S.B., 517
Eikelbloom, R.H., 39
Eikelboom, R., 662, 664, 700
Eikelboom, R.H., 662, 669
Eilers, R.E., 468, 471
Eisenberg, L.S., 71, 831, 846
Eisenman, D.J., 832
Eiserman, W., 448
Eiserman, W.D., 448
Eiten, L., 577, 580, 581
Ekelid, M., 539
Elberling, C., 197, 202, 203, 209, 215, 250, 254, 255, 257, 271, 276
Elbert, T., 533
Eldert, E., 62, 107
Eldredge, D., 198
Eldredge, D.H., 251
El-Kashlan, H.K., 303, 832
Elkayam, J., 503–504
Elliot, L., 69, 71
Elliott, L.L., 473, 825
Ellison, J.C., 155, 156, 157, 159, 160, 184, 360
Elpern, B.S., 66, 94
Elridge, D.H., 598
Embleton, T.F.W., 600
Emde, R.N., 839
Emery, S.B., 489
Endicott, J., 675
English, K., 503–504, 507, 838–839, 851
English, K.M., 627
Enomoto, H., 418
Ensink, R.J., 486
Environmental Protection Agency, 596, 601
Epstein, R.M., 639
Erber, N., 691, 842
Erber, N.P., 854
Erdman, S.A., 783, 849, 851
Erlandsson, S., 649
Ertmer, D.J., 844
Erway, L.C., 484
Erwin, R., 323, 324, 326
Escolar, M.L., 587– 589
Estabrooks, W., 730
Esteban, E., 508
Etler, C.P., 829
Etymotic Research, 69, 70, 71, 131
Eulitz, C., 533, 538
Ewert, C., 780
Ewing, A.W.G., 437
Ewing, I.R., 437
Ezan, N., 578

Faber, H.T., 818
Fabry, D., 781
Fairbanks, G., 72
Fairey, A., 753
Fanaroff, A.A., 583
Fanning, R., 768
Fatima, A., 488
Fausti, S.A., 43, 603
Fay, R., 188
Feeney, M.P., 155–160, 165–184
Fein, D., 586
Feinstein, J.C., 149
Feldhake, L.I., 65
Feldhake, L.J., 120
Feldman, A.S., 80, 140
Feldmann, D., 488
Feldmann, H., 49, 61
Fenson, L., 845
Ferguson, A.S., 667
Ferguson, M.A., 520
Ferm, I., 255
Fernandez, C., 385
Ferrari, D.V., 668, 669
Ferraro, J.A., 219
Ferre, J., 571, 574
Ferre, J. M., 571
Ferreli, C., 753
Ferrero, P., 197
Ferrucci, L., 631
Festen, J.M., 539, 762
Feth, L.L., 677, 678
Fetter, M., 404
Fidell, S., 676
Fields, R.R., 488
Fifer, R.C., 72
Fife, T.D., 413
Fikret-Pasa, S., 715
Fineberg, H.V., 365
Finitzo-Hieber, T., 676, 678
Finitzo, T., 443
Fink, G.R., 517
Fink, N.E., 846
Finney, D.J., 64
Fino-Szumski, M.S., 588, 592
Finucane, M., 664
Fiordelli, M., 671
Firszt, J.B., 352, 826
Fiscella, K., 639
Fischel-Ghodsian, N., 489
Fischer, S.M., 491
Fisher, C.G., 855
Fisher, R., 488
Fisher, R.A., 496
Fitch, R.H., 538
Fitzgerald, T., 144
Fitzgerald, T.S., 372
Fitzpatrick, D., 156, 159, 160, 184, 465
Fitzpatrick, D.C., 249
Fitzpatrick, D.F., 155, 156, 157, 159, 160
Flamme, G.A., 717
Flatman, S., 177
Flax, M., 766, 791, 796
Flaxman, S., 664
Fleming, M., 504
Fletcher, H., 66, 67, 90
Fletcher, W.A., 404
Flexer, C., 675–679, 682, 688, 689, 691
Flexer, C.A., 120
Fligor, B.J., 605, 606, 610, 611
Flottorp, G., 605
Flynn, M.C., 72
Fobel, O., 254, 255
Foley, R., 764
Folmer, R.L., 651
Folsom, R., 468
Folsom, R.C., 254, 256, 258, 468, 589, 590
Fombonne, E., 585
Fontaine, A., 536
Food and Drug Administration (FDA), 705, 754
Forli, F., 583
Formby, C., 655
Forquer, B.D., 168
Forsman, I., 438, 447–449, 453
Fortnum, H., 177
Fortnum, H.M., 583
Forton, G., 200
Forton, G.E., 200
Foss-Feig, J.H., 586
Foust, T., 448
Fowler, C.G., 152, 153, 155, 243, 244
Frampton, C.M., 427
Franceschini, S.S., 842
Franks, P., 639
Frank, T., 57, 63, 64, 79, 625, 760
Fransen, E., 489
Frazer, N., 232
Fredrickson, J.M., 31, 41
Freed, D., 745
Freed, D.J., 720
Freedman, R., 329
Freeman, S., 307
Freer, C.B., 753
French, K.S., 481
French, N., 676
Frey, R.H., 43
Frøh, A., 492
Fria, T.J., 38, 43
Frick, K., 636
Friderici, K., 488
Friederichs, E., 576, 577, 579, 580
Friederichs, P., 576, 577, 579, 580
Friedland, P.L., 662, 664
Friedman, H., 583
Friedmann, G., 419
Friedman, R.A., 483
Friedman, T.B., 479, 483, 488, 495, 496
Fried, T., 642
Friesen, L.M., 345, 352
Frisch, S.A., 120
Frisina, R., 633–635, 667
Fristoe, M., 845
Froelich, T.M., 668
Frohman, E., 413
Frymark, T., 153
Fukuda, T., 404
Fukushima, K., 486
Furman, J.M., 404, 413, 417
Furst, M., 239, 242

Gacek, J., 633, 634
Gadzinowski, J., 583
Gaffney, M., 443
Gafni, M., 307
Gaggl, W., 826
Gajewski, B.J., 120
Galaburda, A., 317
Galaburda, A.M., 534
Galambos, M.P., 327
Galambos, R., 198, 249, 253, 257, 326, 327, 805
Galbraith, G.C., 325
Galiana, H.L., 391
Gallaudet Research Institute, 583
Gallun, F.J., 603
Galster, J.A., 764
Gandour, J., 530–531, 535
Gans, R.E., 426–432
Gantz, B., 649, 653, 831
Gantz, B.J., 176, 648
Garadi, R., 753
Gardener, J.C., 239, 242
Garde, S., 262
Garell, P.C., 539
Garza-Holquin, Y., 181
Gasaway, D.C., 600, 606
Gaskill, S.A., 363, 364, 367
Gatehouse, S., 167, 783
Gates, G., 632–635
Gates, G.A., 429
Gates, S., 399
Gauger, D., 609
Gawryluk, J.R., 520
Gazmararian, J., 642
Geers, A.E., 71, 831, 832
Gehringer, A., 647, 650
Gehringer, A.K., 649, 650, 652
Gelfand, E.T., 307
Gelfand, S., 678
Gelfand, S.A., 104, 125, 126, 167, 170–173, 175, 180, 181, 183
Gelhar, K., 618
Gencer, N.G., 320
Gengel, R.W., 36
Ge, N.N., 219
Genovese, E., 220, 224
Genther, D., 636
Georgantas, L.M., 144
George, C.F.P., 401
Georgsdottir, I., 583
Gerber, M., 806
Gerhardt, K.J., 43, 166
Gerken, G.M., 324
Gerling, I.J., 240
Geroso, A., 448
Geschwind, N., 534
Ge, X., 218
Gianna, C., 429
Giarbini, N., 485
Gibbens, N.D., 232, 246
Gibbs, F.A., 320
Gibson, P.J., 828
Gibson, W., 219
Gibson, W.P., 209
Gibson, W.P.R., 220
Gidley, P., 176
Gifford, R., 827, 828
Gilbert, S.J., 596
Gillespie, L.D., 399
Gillespie, W.J., 399
Gill, K., 238
Gilmore, C., 69, 762
Ginis, J., 777, 783
Ginsburg, W.H. Jr, 450
Ginsburg, W.H., Jr, 450
Ginter, S.M., 39
Giolas, T.G., 783
Gjini, K., 329
Gladstone, V.S., 97
Glasberg, B.R., 45, 662, 715
Glasscock, M.E., 234, 235, 239–241
Glasscock, M.E. III, 303
Glattke, T.J., 360, 364
Gleason, W.J., 618
Gleich, O., 635
Glenthøj, B.Y., 329
Gliklich, R.E., 312
Glista, D., 350, 715, 764, 772
Glorig, A., 72
Goberis, D., 844, 846
Gode, S., 153
Goebel, J., 789
Goedert, M.H., 444
Goetzinger, C.P., 618
Goff, W.R., 322–324
Goforth, L., 262, 488
Gogel, S.A., 649
Gogle, S., 650
Goines, L., 604
Goitein, K., 307
Goldberg, J.M., 385
Goldin, P.R., 650
Gold, M., 677
Goldman, R., 845
Gold, R., 638
Gold, S.L., 655
Gold, S.R., 218
Goldstein, D., 852
Goldstein, D.J., 583
Goldstein, D.P., 94
Goldstein, H., 574, 688
Goldstein, M., 209
Goldstein, R., 148, 170
Gollegly, K.M., 235, 240
Gonzales, L., 471
Gonzalez, J.E., 231–246
Goode, R.L., 51, 52
Goodhill, V., 19
Gooding, D.C., 329
Goodman, A., 38, 63, 65
Goodman, S.S., 33, 372
Gopinath, B., 632, 635, 636
Gordon, K.A., 329
Gordon, M.L., 232
Gordon-Salant, S., 631, 636
Gorga, M.P., 155–157, 159, 160, 254, 256, 258, 259, 367–372, 465
Goswami, U., 287
Goulios, H., 665
Gourévitch, B., 520
Goycoolea, H.G., 154
Grady, C.L., 517
Graham, M.D., 303
Grandori, F., 234, 362
Granneman, S., 488
Grantham, S.L., 396
Grant, I.L., 157, 158
Gravel, J., 832
Gravel, J.S., 120, 257, 260, 444, 451, 760
Gray, R.F., 728
Greenberg, D.B., 589
Greenberg, J.S., 310
Greenberg, S., 268
Green, D.M., 365
Green, D.R., 443
Green, J., 831
Green, J.A., 586
Green, W.B., 260
Greetis, E.S., 70
Gregory, M., 852, 858
Griest, S.E., 649
Griffith, A.J., 479, 483, 488, 494, 496
Griffiths, J.D., 67
Griffiths, S.K., 324
Griffiths, T.D., 517–518
Grimes, A., 767
Grine, L.E., 403
Groen, W.B., 586
Grose, J.H., 238, 274
Grosse, S.D., 443
Grossman, G.E., 413
Gross, R.D., 753
Grothe, B., 516
Groth, J., 704
Grover, N., 242
Gruenwald, J., 396
Grzanka, A., 449
Gstoettner, W., 823
Gudmundsen, G.I., 69, 78–81, 99, 532
Guede, C.I., 422
Guichard, J-P., 233
Guidetti, G., 404
Guilford, P., 477
Guinan, J.J., 358–360, 363
Gultekin, G., 172
Gu, M.J., 422
Gunter, M.B., 63, 64
Gustafson, S., 768
Gustafson, S.J., 762
Gutschalk, A., 269
Guy, R., 591

Haacke, N., 402
Haas, H., 679
Hackett, T.A., 315
Hack, M., 583
Hadar, E.J., 306
Haegerty, P., 642
Hafter, E., 634, 637, 641, 718
Haggard, M.P., 561, 618
Haggerty, H.S., 79, 80, 99
Hagler, L., 604
Hagler, P., 771
Haines, S., 42
Hain, T.C., 404
Hajicek, J., 678
Hakansson, B.E., 771

Hallam, R.S., 647, 649, 650
Hall, C.M., 53, 54, 55
Hallenbeck, S.A., 704
Hallewell, J.D., 618
Halliday, L.F., 534
Hall, J., 197, 578, 579, 634
Hall, J.H., 529, 540
Hall, J.W., 234, 235, 240–243, 625, 700
Hall, J.W. III, 120, 131, 486
Hall, M., 814
Hall, S.E., 517–518, 520, 522
Halmagyi, G.M., 403, 418, 428
Halpin, C., 326
Hamade, M.A., 157, 159
Hamel, G., 251, 261
Hamernik, R.P., 43
Hamilton, A.E., 231
Hancock, K.E., 516
Hanin, L., 67
Hanks, W.D., 148, 465
Han, M.K., 489
Hanna, T.E., 36
Hannley, M., 104, 176, 180
Hansen, M.R., 649, 653
Haraldsson. A., 583
Hardardottir, H., 583
Harford, E., 172, 177, 183, 805
Harigai, S., 590
Harker, L.S., 31, 41
Harm, D.L., 431
Harmon, C., 564
Harrington, I.A., 522
Harris, C., 12
Harris, D., 357
Harris, F., 584
Harris, F.J, 326
Harris, F.P., 363, 364, 367, 372
Harris, K.C., 344, 596, 602–604
Harrison, R.V., 513–514
Harrison, W.A., 366
Harris, S., 492
Harris, T., 631, 635
Harris, T.B., 527
Harrys, R., 148
Hartel, D.M., 448
Hartley, D., 764
Hartley, L., 764
Hartung, J.P., 845
Harvey, D.J., 753
Hashimoto, I., 322
Haskell, G., 650
Haskins, H., 473
Haslwanter, T., 403, 405
Hasselblad, V., 448
Hatch, D., 832, 833
Hate, N., 52
Hatton, J., 464
Hausler, R., 327, 419
Hawkins, D., 789
Hawkins, D.B., 17, 858
Hawkins, J.E., 90
Hawkins, J.E. Jr, 63, 66, 107
Hawkley, L., 631
Hawley, M.S., 19
Hayes, C.S., 94
Hayes, D., 65, 119, 462, 750
Hayes, E., 531, 538, 562
Hayes, M., 846
Haynes, D.S., 828
Hazard, L., 831
Hazell, J.W.P., 654
Health Resources and Services Administration (HRSA), 455
Healy, E., 631
Hearing Industries Association, 703
Heavner, K.S., 827, 832
Hebb, D.O., 519
Hecht, B.F., 448
Hecker, M.H.L., 67
Hecox, K., 198
Hecox, K.E., 241, 253, 257
Hedelund, U., 651
Heimdal, K., 492
Heitger, M.H., 427
Heithaus, D., 590
Heitmann, J., 371
Helbig, S., 823
Heller, J.W., 141
Hellgren, J., 719, 720
Hellweg, F.C., 531, 536
Helzner, E., 631, 632, 635
Hemenway, W.G., 442
Henderson, D., 43, 596, 598, 602–604
Henderson, T.L., 599, 604
He, N-J, 371
Henning, R., 730, 735, 740
Henry, J.A., 43, 603, 649
Henry, J.L., 650–652, 655
Hentona, H., 236
Hepler, E.L., 43
Hepler, E.L. Jr, 166
Herdman, A., 464
Herdman, A.T., 269, 273, 280, 288
Herdman, S.J., 403, 426–428
Herer, G., 626
Herman, B.S., 33
Hermann, B.S., 182
Herman, P., 233
Herring, H., 841
Hersbach, A.A., 715
Hersey, P., 839
Herzog, H., 52
He, S., 352
Hetu, R., 45
Heydemann, P.T., 261
Heyworth, T., 627
Hickson, L., 168, 171, 636, 858
Highstein, S.M., 385
High, W.S., 72
Hildebrand, M.S., 487
Hildesheimer, M., 831
Hill, M., 842
Hillman, E., 427
Hill, M.M., 585
Hillyard, S., 197, 198
Hillyard, S.A., 253, 325, 520
Himelfarb, M.Z., 148
Hintermair, M., 839
Hiraiwa, F., 418
Hirakawa, S., 486
Hirsch, A., 183, 306
Hirsch, B.E., 218
Hirsch, J.E., 144
Hirsh, I.J., 62, 63, 65, 66, 107
Hirsh, S.K., 252, 258
Hjorth, B., 320
Hnath-Chisolm, T., 67
Hochberg, I., 17
Hochstein, S., 535
Hodges, A., 831
Hodges, A.V., 172
Hodgetts, W.E., 771
Hodgson, W., 94
Hodgson, W.R., 71
Hoeker, G., 750
Hoekstra, C., 767
Hoffman, H., 631
Hoffman, Y., 839
Hofman, P.M., 522
Hofstetter, S., 520
Ho, K., 659
Holcomb, M.A., 587– 589
Holden, L., 801
Holden, L.K., 826
Holden, R., 591
Hollich, G., 831
Holmes, A., 508
Hol, M.K., 818
Hol, M.K.S., 764
Holte, K., 465
Holte, L., 148, 472
Holzinger, D., 837, 847
Homnick, D.N., 465
Honaker, J.A., 404
Honrubia, V., 383–386, 389
Hood, J.D., 62, 65, 82, 90, 95, 96, 97
Hood, L., 190
Hood, L.J., 120, 179, 204, 207, 249–265, 325, 451, 465, 488, 521
Hood L.J., 451
Hood, W.H., 623, 626
Hoover, B., 762, 764, 845
Hoover, B.M., 465
Hopkinson, N.T., 624
Hopkins, T., 670
Horak, F.B., 427, 428, 431
Hornickel, J., 325, 531, 537–538, 576–579
Horn, J., 782
Hornsby, B.W., 710
Horton, N.J., 157
Hosford-Dunn, H., 79, 80, 99, 805
Hostler, M., 764
Houben, R., 717
Houlden, D., 418
Hou, S., 767
House, A.S., 67
House, J.W., 232, 233, 306
Houston, D.M., 831
Houston, G.D., 232
Houston, K.T., 453, 670
Houtgast, T., 539, 762
Howard, M.A., 539
Howe, D.M., 662
Howell, P., 12, 13
Huang, C., 187, 231
Huang, J.Q., 257
Huang, T., 223
Huang, T.W., 418
Hu, B.H., 596, 602–604
Hudgins, C.V., 63, 66, 107
Hudson, T.M., 471
Hudspeth, A.J., 383
Huebner, W.P., 413
Hug, G.A., 783
Hughes, G.B., 303
Hughes, L., 633–635
Hughes, M., 829
Hughson, W., 36
Huis in't Veld, F., 253
Hulecki, L.R., 49, 284
Hullar, T.E., 487
Humes, L., 637, 638
Humes, L.E., 72, 534, 606
Humiston, S.G., 452
Hunter, L.L., 137–161, 172, 178, 183
Hunter, M.L., 662, 664
Huotilainen, M., 533
Hur, I.A., 487
Hurley, A., 254
Hurley, R.M., 169, 234–236, 239, 241
Hurtig, R., 718
Hussain, D.M., 366, 367
Hutman, T., 585
Hutton, C.L., 623, 626
Huygen, P.L., 487–489
Huy, P.T., 233
Hyde, K.L., 586
Hyde, M., 196, 340, 342
Hyde, M.L., 245
Hygge, S., 678

Ida Institute, 852
IEC 60318-4, 732
Iino, Y., 590
Illing, R., 634, 635
Imamura, Y., 590
Institute of Electrical and Electronics Engineers (IEEE), 69
Institute of Medicine, 849
International Electrotechnical Commission (IEC), 9, 11, 19, 21
International Organization for Standardization (ISO), 9, 11, 19, 599
Internet World Stats, 665
Iosif, A.M., 585
Ireland, P.E., 437
Irene, T., 812
Ireys, H., 447–449, 448, 449
Irvine, D., 268
Irvine, D.R.F., 520
Isaacson, B., 489
Iseli, C., 219
Ishimoto, S.I., 238
Ison, J., 634
Itoh, A., 418
Ito, K., 238
Ito, M., 387
Ives, T.E., 606

Jackler, R.K., 56
Jackson, A., 159, 579
Jackson, C.G., 303
Jackson, D.F., 262
Jackson, G.L., 448
Jackson, P., 855
Jacobs, M., 56
Jacobson, G.P., 317, 328, 392, 396, 402, 428, 429, 783
Jacobson, J., 6, 197
Jahn, A.F., 38
Jahnke, M., 827
James, A., 777, 783
James, M., 444, 451
Jamieson, D.G., 766
Jang, M., 518
Janky, K., 55
Janky, K.L., 182
Jannetta, P.J., 231, 232, 238, 303, 325
Janssen, T., 372
Jasper, H., 190
Jasper, H.H., 318
Jastreboff, P.J., 650
Jauhiainen, T., 618
Jayakody, D.M.P., 669
Jeffers, J., 855
Jeffress, L.A., 516, 522
Jeng, P.S., 159, 160
Jenstad, L., 780
Jenstad, L.M., 351, 716, 767
Jerger, J., 36, 65, 69, 104, 120, 140, 149, 172, 176, 177, 180, 181, 183, 233, 243, 245, 462, 621, 626
Jerger, J.F., 64, 72, 119, 175
Jerger, S., 38, 71, 104, 176, 177, 180, 233
Jesteadt, W., 35, 258, 259
Jewett, D., 189, 253
Jewett, D.L., 295
Jezewski, M.A., 839
Jiang, D., 516, 522
Jiang, Z.D., 224
Ji, H., 647, 649, 653
Jin, S.H., 43
Jirsa, R.E., 232, 234–236, 239–241, 244
Joellenbeck, L.M., 606
John, A., 578, 579, 764
John, A.B., 119–132, 676– 678, 681, 686
John, M.S., 269, 271, 273, 276, 284
Johns Hopkins Medical Hospital (Baltimore), 437
Johnson, A., 715, 764
Johnson, C., 39, 581, 676, 836, 843, 844
Johnson, C.D., 501, 503, 504, 508–510, 836, 844
Johnson, C.E., 149
Johnson, D., 12, 599, 856
Johnson, E.E., 771
Johnson, E.L., 427
Johnson, E.W., 199
Johnson, G.D., 235, 240
Johnson, J., 444, 451
Johnson, K., 243, 245, 528
Johnson, T.A., 251, 252, 282, 364, 371
Johnsrude, I.S., 517–518
Johnston, D.R., 497
Johnstone, B.M., 208
Johnstone, P., 470
Johnstone, P.M., 762
Johnston, K., 578, 579

Joint Committee on Infant Hearing (JCIH), 120, 131, 249, 370, 440, 442, 444, 447, 448, 450–452, 454, 759, 836, 839, 847
Jones, C., 766
Jones, R.D., 427
Jones, R.O., 236, 239, 241–243
Jones, S.M., 485
Jones, T.A., 485
Jonsdottir, G.M., 583
Joosten, F.B., 489
Jordan, H., 649
Joris, P.X., 517
Joseph, A., 72, 590
Josey, A.F., 72, 234, 235, 239, 240, 241
Joutsiniemi, S.L., 328
Jungner, G., 438, 455

Kaas, J.H., 315
Kaga, K., 418, 585
Kahn, A., 209
Kahn, J.B., 310, 312
Kalb, J.T., 596, 603
Kaldo-Sandström, V., 670
Kaldo, V., 670
Kalikow, D.N., 69
Kalluri, R., 372
Kalluri, S., 638, 718
Kamath, V., 539
Kambayashi, J., 386
Kamerer, D.B., 218
Kaminski, J.R., 254, 258, 259
Kamm, C.A., 65
Kamm, D., 72
Kanaya, M., 418
Kaneko, Y., 386
Kania, R.E., 233
Kankkunen, A., 171
Kan, S.W., 439
Kaplan, H., 39, 97
Kaplan-Neeman, R., 831
Kaprio, J., 632
Karapurkan, T., 585
Kardatzke, D., 143
Karino, S., 238
Karl, A., 329
Karlin, J.E., 63, 66, 107
Karpa, M., 632, 636
Karzon, R.K., 487
Kasai, N., 486
Kasden, S.D., 626
Kashio, A., 632
Katbamna, B., 465
Kates, J., 718, 720
Katsch, R., 766
Katz, D., 71, 473, 825
Katz, J., 6, 34, 36, 72, 77, 82, 94, 99, 561, 562, 564, 570, 618
Katz, R.C., 232
Kaylie, D.M., 392
Keamy, D.G., 477
Keats, B., 488
Keats, B.J., 488–489
Keats, B.J.B., 120
Keefe, D.H., 155, 156, 157, 159, 160, 183, 184, 360, 364, 372, 465
Keenan, D., 579, 715, 777
Keidser, G., 720, 721, 766, 791, 796
Kei, J., 148, 156, 159, 168, 171, 448
Keirsey, D., 806
Keith, R.W., 148, 181
Kellett, A.J.C., 261
Kelley, P.M., 488
Kelly, A., 575, 578
Kelly, A.S., 128, 329
Kelly-Ballweber, D., 324
Kelly, J., 590
Kelly, J.B., 517
Kelly, T., 665, 666, 671
Kelsell, D.P., 132, 477
Kemink, J.L., 303, 328
Kemp, D.T., 357–364
Kemper, A.R., 448
Kemperman, M.H., 487
Kemp, R.J., 728
Kennalley, T., 444, 451
Kennedy, C.H., 590
Kennedy, C.R., 443
Kenneson, A., 460
Kentish, R.C., 653
Kent, R.D., 97
Kerber, K.A., 381, 383, 386, 387, 412, 422
Kerivan, J.E., 80, 94
Kermany, M.H., 418
Kessler, D.T., 71
Kettel, J., 718
Khan, A., 159
Kiang, N., 209
Kiang, N.Y.S., 209
Kibbe, K., 236, 241
Ki, C.S., 54
Kidd, S.A., 517
Kiefer, J., 823
Kiely, K., 635
Kieper, R.W., 609
Kiernan, J.A., 386
Kieszak, S., 508
Kifley, A., 631, 637
Kikuchi, Y., 636
Kileny, P., 198, 251
Kileny, P.R., 296, 297, 303, 306, 307, 322, 325, 328, 491, 825
Killion, M.C., 32, 37, 61, 68, 69, 78–81, 99, 120, 532, 609, 715, 760
Killion, M.D., 17
Kim, A.H., 306
Kimberling, W.J., 491
Kim, C.Y., 405
Kim, D.O., 357, 358
Kim, H.J., 405, 488
Kim, H.N., 831
Kim, J.H., 405
Kim, J.R., 345
Kim, J.S., 405, 717
Kim, T.B., 489
Kimura, D., 72
Kim, Y.S., 418
Kindermann, S., 650
King, A.M., 41, 262
King, C., 531, 538
King, C.D., 521
King, J.E., 172
King, K., 493
King, K.A., 493–494
King, W., 636
King, W.M., 538
Kirazli, T., 153
Kirby, B.J., 371, 372
Kirkegard, C., 148
Kirkim, G., 153
Kirk, K.I., 71, 474, 831
Kirksunov, L., 52
Kishon-Rabin, L., 67, 831
Kitamura, K., 492
Kjaer, M., 197
Klatte, M., 678
Kleidel, W.D., 33, 34
Klein, A., 44
Klein, A.J., 125
Klein, B., 638
Klein, B.E.K., 142, 144
Kleineck, M., 579
Klein, R., 142, 144, 638
Klin, A., 585
Klinepeter, K.L., 583
Klockhoff, I., 42
Klucharev, V., 533
Kluka, E., 254
Knecht, H.A., 677, 678
Knight, J.J., 80
Knight, R.D., 359, 363
Knight, R.T., 324, 327, 328
Knutson, C.L., 472
Kobayashi, T., 386
Koch, D.B., 521, 538
Kochhar, A., 491
Kochkin, S., 651, 720, 727, 746, 781
Kodera, K., 325
Koebsell, K.A., 143
Kokesh, J., 667
Kok, M.R., 364
Koller, K., 667
Kollmeier, B., 183, 202, 255
Komatsuzaka, A., 492
Komatsuzaki, A., 236
Kondo, K., 632
Kongstvedt, P., 814
Kong, Y.Y., 521
Konigsmark, B.W., 477
Konishi, M., 522
Konishi ,Y., 242
Konkle, D.F., 44, 61, 63, 66, 81, 86, 91, 99, 101, 103, 104
Konrad-Martin, D., 465
Kontorinis, G., 619
Kontrogiaannis, A., 619
Koo, J.W., 405
Koop, C. E., 441
Kopp, C., 468
Kopun, J., 764
Korhonen, P., 715
Kornreich, L., 56
Korzyukov, O., 324
Koskenvuo, M., 632
Kosky, C., 71
Kotlarz, J.P., 232
Kowal, A., 69
Kozak, F.K., 157, 159
Krainz, W., 52
Kramer, P., 426
Kraus, N., 189, 207, 224, 261, 268, 320, 324, 325, 327–329, 338, 346, 350, 521, 527, 530–532, 534, 536, 537–540, 561, 562, 576–579, 633
Krausz, H.I., 253
Kravitz, R.L., 639
Kreisman, B., 676– 678, 681, 686
Kreisman, B.M., 119–132
Kreisman, N., 578, 579, 676, 677
Kricos, P., 853
Krieg, E.F., 484
Krishnan, A., 530–531, 533, 535, 633
Kristensen, S.G.B., 255
Kronenberg, J., 831
Kruel, E.J., 62, 67
Kruger, B., 19, 78, 768
Kruglikov, S.Y., 318
Krumbholz, K., 517
Krumm, M., 668
Kryter, K.D., 67, 598
Krzan, M.J., 296
Kubicek, L., 839
Kuehn, L.A., 385
Kühn-Inacker, H., 842
Kujawa, S.G., 43, 602, 647
Kuk, F., 197, 579, 715, 748, 764, 777
Kuk, F.K., 649
Kuklinski, A.L., 79, 80, 99
Külekçi, S., 587
Kumar, M., 41, 54
Kummer, P., 364, 367, 372
Kunst, H.P., 486
Kuo, A.A., 448
Kuo, M.J., 590
Kurdziel, S.A., 183
Kurima, K., 488
Kuriyama, M., 242
Kurs-Lasky, M., 760
Kusakari. J., 386
Kusuki, M., 370
Kuwada, S., 249, 268, 269
Ku, Y., 149
Kwakye, L.D., 586
Kwan, K.Y., 383
Kwong, B., 199, 200, 202, 237
Lachmann, T., 678
Lafaille, P., 534, 541
Lagziel, A., 496
Lahiri, A., 533
Laipply, E., 67
Laird, D.A., 604
Lalwani, A.K., 484, 489
Lalwani, E., 312
La Malfa, G., 585
Lambert, P.R., 387, 391, 393
Lamb, S., 783
Lam, C.F., 44, 125
Lance, J.W., 429
Lane, G.I., 99
Langer, D.R., 315
Langford, J., 508
Lang, J.S., 67
Langlan, L., 686
Langman, A.W., 754
Lansing, C., 855
Lanska, D.J., 413
Laoide, S., 439–440
Lapsley Miller, J.A., 159, 160, 370
Laroche, C., 45
Larsby, B., 678
Larsen, H.C., 670
Lasky, R.E., 273
Lassi, S., 585
Laszig, R., 172
Latinus, M., 541
Lau, C., 579, 764
Lau, C.C., 715
Launer, S., 766
Laurent, C., 667
Laurnagara, D., 765, 766
Laurynagay, D., 796, 797
Lavigne-Rebillard, M., 357
Law, C.M., 443
Law, S.K., 320, 321
Lawson, G.D., 592
Lawton, B.W., 155
Layne, M., 43, 595
Leal, S.M., 486, 488
Leandri, M., 319
Leavitt, R., 679
Le, C.T., 54
Lee, C.C., 315, 517, 522
Leech, H.M., 763
Leeder, S., 636, 639
Lee, F., 44, 125
Lee, F.S., 42
Lee, H., 385
Lee, H.M., 303
Lee, S.E., 831
Lee, W.S., 831
Lee, W.W., 239–241, 328
Lee, Y.S., 322, 323
Legatt, A.D., 307
Lehiste, I., 66, 535
Lehman, S., 497
Lehnhardt, E., 172
Leicher, L., 61
Leigh, I.M., 132
Leigh, R.J., 389, 390, 395, 413
Leinonen, L., 328
Lempert, B.L., 599, 604
Lench, N.J., 132, 477
Lenthall, R., 177
Leonard, C.M., 538
LePage, E.L., 605
Leren, T.P., 492
Lerman, J., 71, 473
Lesperance, M.M., 486, 489
Leventhal, A., 635
Levi, E.C., 159, 274, 275
Levilliers, J., 477
Levine, R.A., 239, 242, 647
Levine, S., 42
Levine, S.C., 172, 178, 183
Levin, S., 670
Levinson, M.J., 56
Levitt, H., 67, 638, 640, 854, 855

Lewine, J.D., 317, 328
Lewis, D., 762
Lewis, D.E., 156, 159, 160
Lewis, J.D., 770
Lewis, M., 782
Liang, J.N., 132, 477
Liang, L., 531
Liang, Z.A., 603
Liberman, M.C., 43, 220, 227, 357, 602, 647
Lichtenhan, J., 215
Lichtenstein, V., 366
Liden, G., 166, 171
Lidén, G., 54, 92–94, 99, 103, 138–140, 149
Lieberman, L.J., 587
Liegeos-Chauvel, C., 322
Lightfoot, G., 177, 255, 264
Light, G.A., 329
Lijffijt, M., 329
Li, L., 262, 451
Li. L., 120
Lilly, D.H., 183
Lilly, D.J., 25, 156
Li, M.W., 418
Linden, D., 261
Lindsay, R., 635
Lindström, B., 244, 245
Lin, E., 312
Lin, F., 631, 632, 635, 636
Lin, F.R., 527
Ling, L., 634, 635
Ling, R., 155
Ling, X., 484
Lin, H.W., 399
Lin, J., 585
Lins, O.G., 268, 272–274, 277, 284
Lisberger, S., 426
Lister, J., 637, 638
Litovsky, R., 828
Littler, T.S., 80
Littman, T.A., 120
Litvan, H., 319, 325
Liu, Q.J., 489
Liu. X.Y., 224
Liu, Y., 155, 156
Liu, Y.W., 156, 159, 160, 184
Li, X., 439
Li, Y., 42, 631
Lloyd, L.L., 39, 97, 586
Lobarinas, E., 43
Lockwood, A.H., 647, 654
Lodwig, A., 264
Lofqvist, L., 197
Lombardino, L.J., 538
Lomber, S.G., 517
Long, G.R., 371, 372
Long, Q., 489
Longridge, N., 146, 157
Longridge, N.S., 403
Lonsbury-Martin, B.L., 363, 364, 465
Loovis, C., 642
Lopes da Silva, F., 318, 320
Lorente de No, R., 316
Lot, G., 233
Lotze, A., 854, 855
Love, J.T., 303
Lowery, K.J., 470
Lubrica, P., 472
Luce, T., 533
Luders, H., 303
Ludvigsen, C., 740
Lumio, J.S., 618
Lu, N., 845
Lundberg, T., 667
Lundberg, Y.W., 385
Lundborg, T., 244, 245
Lunner, T., 669, 719
Lupi, G., 224
Luria, A., 561, 564
Luszcz, M., 635
Lu, T., 531
Luterman, D., 851
Lutolf, J., 175
Luxford, W., 262
Luxford, W.M., 172, 183
Luxon, L., 402
Lynch, C., 618
Lynch, R.M., 416
Lynn, D.J., 175
Lyons, M.J., 165
Lyttkens, L., 650, 670
Lyxel, B., 243

Macari, S., 585
MacDonald, E., 521
MacDonald, M., 636
MacFarland, S.Z.C., 588
Macharia, I., 439
Machin, D., 753
Macleod, A.D., 427
Madell, J.R., 120
Madeo, A.C., 487, 493
Magdziarz, D.D., 42
Magnan, J., 517, 520
Magnavita, V., 211
Magne, C., 534
Magnusson, M., 218
Magruder, A., 120
Mahendran, A., 41
Mahendran, S., 54
Maheshwar, A., 54
Maheshwar, S., 41
Mahncke, H., 540
Mahomed, F., 662
Mahoney, T.M., 440
Mai, C., 588
Maier, N., 823
Mair, A., 596
Makeig, S., 249
Makela, A.M., 532
Makinen, V., 532
Makishima, T., 487, 494
Malandrino, A., 772
Malandrino, A.C., 767, 772, 773
Malhotra, S., 517
Mallinson, A.I., 403
Malmierca, M.S., 517–518
Malmivuo, J., 339
Malmquist, C., 54, 99
Maloff, E., 204
Maloff, E.S., 255
Manchaiah, V.K., 669, 852
Mancini, F., 54
Mancl, L.R., 589
Mandell, D.S., 585, 586
Manichaikul, A., 493
Manley, G.A., 357
Mann, Y.M., 449
Manolidis, S., 310
Mantokoudis, G., 422
Marcell, M.M., 589
Marchant, C.D., 149, 170
Marchbanks, J.R., 303
Marchbanks, R.J., 56
Marchbanks, T., 623
Margoliash, D., 518
Margolis, R.H., 44, 58, 63, 65, 141–144, 148, 151, 154, 156, 157, 170, 326, 465, 662, 665, 667
Marion Downs Hearing Center, 442
Markham, C.H., 390
Markides, A., 79, 81
Markman, T.M., 831
Marks, J.H., 465
Marlin, S., 488
Marlowe, J.A., 450
Marple, B.F., 753
Marquardt, T., 516
Marques, C., 534
Marquet, J., 200
Marquet, J.F., 200
Marres, H.A., 486
Marryott, L.P., 157, 158, 184
Mars, A., 585
Marsella, P., 494
Marsh, A., 12, 22
Marshall, L., 35, 36, 370
Marshall, N., 197
Marshall, S., 764
Marsh, C., 538, 540
Marsh, J.T., 487
Martin, A., 599
Martin, A.M., 56
Martin, B.A., 260, 349–351
Martin, E., 419
Martinez, A.S., 71
Martin, F., 6
Martin, F.N., 31, 34, 41, 57, 62, 64, 65, 81, 82, 86, 92, 93, 94, 97, 99, 104, 105, 109, 168, 618, 621–623, 626
Martin, G.K., 363, 367
Martin, J.S., 623
Martin, W.H., 257, 651
Marynewich, S., 350, 351
Mascarenhas, M., 664
Mascia, J., 591
Mascia, N., 591
Mason, S.M., 329
Massie, R., 686
Masson, E., 402
Mast, F., 419
Mastroianni, M.A., 484, 489, 493
Masuda, A., 197, 199, 200, 237, 257
Masuda. A., 254
Masure, M.C., 536
Matefi, L., 42
Maternal and Child Health Bureau (MCHB), 443, 444
Mathers, C.D., 664
Matkin, N., 584
Matkin, N.D., 86, 470
Matsui, J.I., 603
Matsunaga, T., 218, 238
Matsuo, V., 390
Matteucci, F., 842
Matthews, L., 44
Matthews, L.J., 42, 125
Mattingly, K.R., 120, 451
Matzker, J., 72
Mauermann, M., 372
Mauk, G.W., 440
Mauldin, L., 120, 176, 177, 621
Maxia, C., 753
Maxon, A.B., 442
Maxwell, S., 460
Ma, Y., 635
May, M.E., 590
Mayne, A., 846
May, P., 532
May, P.J.C., 523
Mazerolle, E.L., 520
Mazevski, A., 144
Mazlan, R., 168, 171
Mazzoli, M., 485
McAleer-Hamaguchi, P., 571
McAlpine, D., 516, 522
McAnally, K.I., 287
McArdle, R., 68, 69, 70, 72, 649, 803, 858
McCall, A., 467, 471
McCann, D.C., 443
McCarthy, P., 853
McCaslin, D.L., 392, 396
McCleary, E., 845
McCormick, C.A., 234, 235, 236, 239, 241
McCreery, R.W., 156, 159, 160, 763, 764, 766, 770, 771
McCreery, T.M., 621
McCullough, J.A., 71
McCullough, J.K., 183
McDermott, A.L., 590
McDermott, H.J., 715
McDonald, P., 427
McDonald, W.J., 43
McFarland, D.J., 318, 320, 325
McFarland, W.H, 326
McGee, T., 189, 324, 327, 538
McGee, T.J., 538
McGowen, R.S., 845
McGrath, A.P., 145
McHugh, R.K., 483
McKenna, L., 649, 652
McKenna, M., 182
McKenna, M.J., 55, 56, 182, 312
McKibben, N., 719
McLennan, R.O., 621
McMahon, C., 631, 635, 636, 637
McMahon, C.M., 220
McManus, M., 803
McMillan, P.M., 149, 170
McMordie, S.J., 487
McPherson, B., 669, 686
McPherson, D., 262
McPherson, D.L., 325
McPherson, J.H., 416
McPhillips-Tangum, C., 449
Mehl, A.L., 249, 443, 459, 835
Mehra, S., 477
Mehta, U.C., 586
Meikle, M., 651
Meikle, M.B., 649
Meinke, D., 508
Meinzen-Derr, J., 827, 842
Meis, M., 678
Meldrum, S.C., 639
Mellert, V., 202, 255
Mellor, D.H., 329
Melnick, W., 10, 596, 598, 602
Menapace, C., 831
Menard, M., 285
Mendrek, A., 586
Merchant, G.R., 157, 159
Merchant, S.N., 33, 44, 55, 56, 177, 182
Meredith, R., 465
Merks, I., 720
Merzenich, M.M., 518, 540–541
Mesibov, G.B., 585
Metson, R.B., 312
Metter, E., 631
Metz, D., 845
Metz, O., 165, 169
Meyer-Bisch, C., 465
Meyer, R., 588
Michaelides, E.M., 145
Michalewski, H., 210, 220, 221
Michalewski, H.J., 219, 220, 521
Michelini, S., 207
Michikawa, T., 636
Middlebrooks, J.C., 517, 522
Middleweerd, M., 691
Migilavacca, F., 72
Mikawa, H., 242
Mikulec, A.A., 182
Milani, S., 211
Miles, B., 590
Miller, E., 814
Miller, G.A., 66
Miller, J.D., 78, 598
Miller, M.H., 170
Miller, S., 538
Mills, J., 632–634
Millward, K.E., 534
Minassian, A.L., 650
Mineau, S.M., 36
Minges, M., 665, 666, 671
Minich, N., 583
Minor, L.B., 55, 181, 405, 418
Mintz, S., 448
Miranda, T., 148
Miranda, T.T., 521
Mishkin, M., 315
Mispagel, K., 777
Mitchell, A., 642
Mitchell, P., 631, 635, 637, 639
Mitchell, R.B., 590
Mitchell, S.A., 41
Mittermaiser, R., 61

Miyamoto, R., 831
Miyamoto, R.T., 71, 831
Mizutari, K., 636
Mkwanazi, H., 662, 667
Mngemane, S., 662, 667
Moberly, A., 831
Mody, M., 538
Moeller, M.P., 444, 447, 459, 762, 771, 837, 845, 847
Moeneclaey, L.L., 200
Mohfoud, L., 157, 159
Mohr, A.M., 651
Molemong, S., 662, 667
Moller, A., 187–189
Moller, A.R., 171, 231, 232, 235, 239, 268, 303, 318, 322, 325, 360
Møller, M.B., 235
Moncrieff, D., 575
Money, K.E., 385
Mongeau, L., 719
Monro, D.A., 622, 626
Montano, J., 858
Montano, J.J., 849, 850, 851
Montaudo, R., 432
Montgomery, A., 789
Montgomery, E., 120, 179, 262
Montogomery, A., 855
Monzani, D., 404
Moodie, K.S., 260, 767
Moodie, S., 761, 766, 768–770, 796, 797
Moodie, S.T., 260, 767, 772, 773
Moog, J.S., 71
Moon, I.S., 831
Moon, S.P., 627
Moore, B., 676, 679
Moore, B.C., 44, 662
Moore, B.C.J., 39, 45, 251, 715, 786
Moore, D.R., 520, 534
Moore, J.K., 257
Moore, J.M., 468, 471, 587, 589
Moore, J.N., 735
Moore, L.E., 386
Moore, R., 844
Morehouse, C.R., 169
Morell, R.J., 488
Moreno, S., 534
Morera, C., 823
Morgan, D., 107
Morgan, D.E., 34, 63, 64, 65, 68, 665
Morimoto, N., 238
Mori, N., 218
Morin, T.M., 527
Morizono, T., 54
Morlet, T., 120, 179, 262, 451, 497
Morris, L.J., 109
Morris, L.O., 417
Morris, M., 197
Morris, M.D., 306
Morrow, S.L., 687
Mortensen, L.B., 440
Morton, N.E., 477
Moschovakis, A.K., 385
Moser, T., 219, 220
Mosko, S.S., 323
Moss, K.A., 65, 120
Moul, M.L., 43
Moushegian, G., 324, 543
Moushey, J.M., 845
Moxon, E., 209
Mucha, A., 417
Muchnik, C., 274, 831
Mudry, A., 817
Mueller, G., 718, 750, 791, 803
Mueller, H.G., 131, 710
Mueller, R.F., 132
Mueller, R.J., 238
Mugwe, P., 439
Mujtaba, G., 488
Mukari, S., 578
Mullee, M., 443
Muller, S.P., 41
Mulrow, C., 675
Mundy, M., 144, 148
Munjal, S.K., 243
Muñoz, K., 438, 450, 771
Munro, K., 780, 798
Murata, K., 243
Murnane, O.D., 418
Murphy, C., 585
Murphy, M.R., 232
Murray, N., 766
Murray, N.M., 605
Musacchia, G., 530–531, 537
Musch, H., 751
Musiek, F., 575
Musiek, F.E., 71, 231–246, 286, 320, 328, 330
Myburgh, H.C., 662
Myers, A.M., 429
Myers, P.J., 603
Mylanus, E.A.M., 764

Naatanen, R., 339, 346, 347, 533
Nabelek, A., 72, 676, 677, 679
Nábelek, A.K., 762
Nabelek, I., 676, 677, 679
Nadeau, J., 483
Nadol, J.B., 160
Naeve-Velguth, S., 838–839
Nagarajan, S., 536, 540
Nagel, R.F., 624
Nageris, B., 56
Naik, K., 209
Nakajima, H.H., 156, 157, 159, 182
Nam, B., 633
National Association of the Deaf, 443
National Athletic Trainers' Association, 422
National Bureau of Standards (NBS), 13
National Center for Hearing Assessment and Management (NCHAM), 439, 442, 444, 447, 448, 449, 452, 453
National Consortium on Deaf-Blindness, 591
National Institute for Occupational Safety and Health, 40, 596, 599, 600, 601, 604, 606, 607, 614
National Institute on Deafness and Other Communication Disorders, 42
National Institutes of Health (NIH), 296, 310, 370, 425
National Institutes of Health to the March of Dimes, 450
Naunton, R.F., 94, 99
Nayak, A., 325
Naz, S., 484, 489
Nearey, T.M., 535
Needleman, A., 675
Neely, J.G., 200
Neely, S., 33
Neely, S.T., 363, 364, 465, 770
Neely, T., 465
Neff, W.D., 33, 34
Nelson, H.D., 453
Nelson, J.R., 668
Nelson, L., 451
Nelson, M.D., 326
Nelson, P.B., 43, 677, 678
Nelson, R., 237
Nelson, R.A., 199, 200
Nelson, R.S., 845
Nesbitt, L., 857
Neuman, A., 678
Neumann, J., 183
Newall, P., 631, 637, 766, 767
Newborough, B., 497
Newby, H.A., 618
Newlander, J.K., 484
Newman, C.W., 402, 428, 429, 783
Newman, J., 324
Newman-Toker, D.E., 422
Newton, J.R., 177
Newton, V., 485
Newton, V.E., 439
New York BSC., 809
Nguyen, K.D., 182
Nguyen, P.H., 306
Nicholas, J.G., 831, 832
Nicholson, G., 238
Nicholson, J., 591
Nicholson, N., 369
Nicol, T., 530–531, 537–540, 562
Nicol, T.G., 538
Niemczyk, K., 449
Niemoeller, A.R., 78
Nikolopoulos, T., 177
Nikolopoulos, T.P., 177
Nilsson, G., 92–94, 99, 103
Nilsson, M., 69, 131, 532, 715
Nilsson, N., 668
Nilsson, R., 166
Niparko, J.K., 303, 831, 846
Niquette, P.A., 68, 69, 120, 532
Nishida, H., 492
Nishiwaki, Y., 636
Nishizaki, K., 486
Niskar, S., 508
Nittrouer, S., 845
Niwa, H., 518
Nixon, J.C., 62, 67
Noack, C., 854, 855
Nober, E.H., 56, 57
Noble, B., 649
Noble, W., 647, 649, 650, 652
Noguchi, Y., 492
Noh, M.D., 539
Nondahl, D., 638
Nondahl, D.M., 142, 144
Norena, A.J., 520
Northern, J., 6
Northern, J.L., 38, 441
Northrop, C., 220, 227
Norton, S.J., 364, 366, 370, 371
Novak, M.M., 585, 586
Nozza, R., 471
Nozza, R.J., 143, 144, 146
Nunez, P.L., 317
Nusbaum, H.C., 527
Nuwer, M.R., 300
Nyffeler, M., 764
Nygren, P., 453

OÆConnell, S., 534, 540
OÆNeal, J., 443
Oates, P., 252, 326, 327
Oba, S., 262
Obleser, J., 533–538
O'Brien, A., 764
O'Brien, R., 631
Occupational Safety and Health Administration (OSHA), 9, 40, 599, 600, 603, 612–614
Ockene, J., 638
O'Connor, A.F., 823
O'Connor, C.A., 232
Oden, C., 854, 855
O'Donoghue, G., 177
Offeciers, F.E., 200
Oghalai, J.S., 583, 585
Ogut, F., 153
Ohyama, K., 386
O'Leary, D.P., 427
Oleson, J., 771
Olgun, L., 172
Oliver, T.A., 181
Oliviera, R., 750
Olsen, L., 751
Olsen, W., 251
Olsen, W.O., 65, 86, 107, 183, 761, 770
Olson, D.J., 43
Olson, L.M., 44
Olson, R.J., 472
Oluwasamni, A., 41, 54
Ondrey, F.G., 493
O'Neal, J., 443
O'Neill, C., 177
O'Neill, W., 633, 635
Onitsuka, T., 326
Oostrik, J., 488
Oranje, B., 329
Orchik, D.J., 218, 219
Orentlicher, D., 814
Orga, M.P., 184
Orlando, M.S., 254, 258
Orrison, W.W., 317, 328
Orten, D.J., 488
Orzan, E., 224
Osberger, M.J., 71, 474
O'Shea, T.M., 583
Osterhammel, D., 170
Osterhammel, P., 170, 253
Ostroff, J.M., 344
O'Sullivan, C., 238
Ototo, B., 439
Ouellette, J., 251
Ou, H., 771
Outerbridge, J.S., 391
Overfield, T., 752
Oviatt, D.L., 167, 169, 170
Owen, G., 197
Owens, E., 67, 71, 89, 783
Owens, J.J., 364
Owsiak, J., 449
Oyler, R.F., 80
Özdamar, O., 207, 224, 241, 261, 327, 328
Ozonoff, S., 585

Pacifico, C., 494
Palmer, A.R., 516, 522
Palmer, C., 718
Palmer, C.V., 853
Paloski, W.H., 431
Palumbos, J.C., 460
Palva, T., 63, 65
Panda, N.K., 243
Pandya, D.N., 315
Pannu, S.S., 591
Pan, T., 649
Panus, P.C., 418
Papanicolaou, A.C., 324
Paparella, M.M., 148, 160
Paperella, M.M., 38, 54
Pappas, J.J., 99
Paradise, J.L., 144, 148, 437, 442, 453, 760
Paradiso, M.A., 519
Parbery-Clark, A., 534, 537, 540, 541
Parish, B., 750
Parisier, S.C., 56
Parker, D.J., 200, 237
Parker, S., 588
Parkinson, A., 766, 828
Park, L.R., 832, 833
Parnes, L.S., 401
Parry, G., 132, 477
Parsa, V., 715, 764
Parving, A., 215, 485
Pascal, C., 585
Pascoe, D., 795–797
Pasman, J., 330
Passchier-Vermeer, W., 599
Pass, H.E., 521
Patel, A., 631, 632, 635
Patel, I., 586
Patel, R., 780, 798
Pathak, A., 243
Patient Protection and Affordable Care Act, 849
Patil, N.G., 659
Patrick, J.F., 828
Patricoski, C., 667
Patterson, J., 262
Patterson, R.D., 517–518
Patuzzi, R.B., 208, 220, 665
Patzkó, A., 238
Paul, R., 585
Pavlovic, C.V., 44

Pawelczyk, M., 484
Payton, K., 691
Peacock, M.E., 252, 258
Peake, T., 177
Peake, W.T., 33, 44
Pearson, J., 596
Pearsons, K., 676
Peck, J.E., 617, 618, 627
Pedersen, K., 596, 599
Pederson, O.T., 67
Peek, B.F., 79, 80
Peled, M., 56
Pelerin. J., 207, 215
Peljhan, Z., 695
Peng, S-C., 845
Penhune, V.B., 534
Penner, M.J., 360
Pennings, R.J., 487, 489
Penrod, J.P., 71
Penry, K., 320
Pensak, M.L., 181
Perdew, A.E., 831
Perez-Abalo, M., 464
Perez, N., 403
Perez, R., 52
Pero, H., 504
Perra, M.T., 753
Perreau, A., 649
Perry, B.P., 648
Pesznecker, S.C., 429
Peterson, A., 826
Peterson, C.R., 175
Peterson, G., 66
Peterson, G.E., 533–535
Peterson, H.J., 470
Peterson, J.L., 139
Petrou, S., 443
Pfaltz, C.R., 42
Pfeifferbaum, A., 324
Phillips, D.P., 167, 514, 516, 518–519, 521–523
Picard, M., 583
Pichora-Fuller, K., 638, 640
Pichora-Fuller, M.K., 521
Pickard, J., 56
Picot, M.C., 585
Picou, E., 791, 803
Picou, E.M., 764
Picton, T., 190, 197, 198, 251, 533
Picton, T.W., 207, 251, 253, 260–262, 272–274, 277, 280, 281, 283–285, 288, 324, 325, 327, 337, 340, 345–348, 464–465, 518, 520–521
Pienkowski, M., 520
Piercy, M., 538
Pike, B., 534, 541
Piker, E.G., 396
Pikora, M., 764
Pillion, A.L., 69
Pillsbury, C., 828
Pillsbury, H.C., 828
Pinnington, L.L., 694
Pipp-Siegel, S., 839
Pirsig, W., 171
Pirzanski, C., 749, 750
Pisa, J., 668
Pisano, D., 157, 159
Piskorski, P., 156, 157, 159
Pisoni, D.B., 71, 474
Pittman, A., 768, 838–839
Pittman, A.L., 760, 762, 770, 771
Place, C., 488
Placidi, G.F., 585
Plack, C.J., 514, 518
Plomp, R., 61, 68, 539, 676, 681, 691
Plourde, G., 327
Plyler, E.L., 470
Poch, N.E., 181
Poelmans, H., 287
Poldrack, R.A., 536
Polich, J., 347, 348
Poling, G.L., 372
Polka, L., 146, 148, 153, 157
Polovoy, C., 668
Ponton, C., 197
Ponton, C.W., 197, 199, 257, 349, 352
Poole, J.P., 62, 65
Popelka, G.R., 25, 148
Popper, A., 188
Porter, H., 589
Porter, L.S., 44
Portnuff, C.D.F., 605, 606
Potts, L., 781, 795, 801
Pou, A.M., 218
Poulsen, C., 274
Powell, L.E., 429
Powers, B.J., 448
Prasher, D., 181
Prass, R., 303
Prass, R.L., 303
Pratt, H., 179, 198, 253, 262
Pratt, R.E., 307
Pratt, S., 631, 632, 635
Preece, J.P., 62, 72
Preis, M., 56
Preminger, J., 857, 858
Pressman, L., 839
Price, G.R., 596, 603
Prickett, J.G., 472
Priede, V.M., 79, 86, 99, 103
Prieve, B.A., 144, 156, 159, 362, 364–366, 371, 372
Primus, M., 469–471
Prince, M.M., 596
Probst, R., 361, 364, 372
Prochaska, J., 852
Proffit, T.M., 418
Proops, D., 833
Proops, D.W., 590
Propes, S., 159
Prosek, R., 789
Prosser, S., 207, 245
Protopapas, A., 536, 540
Proud, G.O., 618
Provost, E., 667
Pryor, S.P., 493, 496
Pry, R., 585
Psarommatis, L., 619
Psarros, C., 842
Pugnetti, L., 418
Pula, J.H., 422
Pu, M., 635
Pumford, J., 260, 761, 766, 768– 770
Pumford, J.M., 716
Punch, J., 72
Puranik, C.S., 538
Purcell, D.W., 287
Purchase-Helzner, E., 527, 631, 635
Purdy, S., 575, 578
Purdy, S.C., 465
Puria, S., 52
Putman, C., 845
Puttermann, D., 802
Pyle, G.M., 306
Pynchon, K.A., 320
Pyykko, I., 632

Qaqish, B.F., 56
Quar, T.K., 767
Quigley, S.M., 754
Quinlan, C.K., 514, 516
Quittner, A.L., 831, 846
Qui, W.W., 166

Radeloff, A., 823
Radziszdsska-Konopka, M., 449
Raffin, M.J.M., 66
Raggio, M., 79, 80, 99
Raggio, M.W., 71
Rakerd, B., 72
Rall, E., 838–839
Rama-Lopez, I., 403
Ram, B., 177
Ramirez, G.M., 284
Ramsey, M.J., 182
Rance, G., 41, 238, 262, 273, 279, 280, 282, 286
Rantanen, T., 632
Rao, A., 372
Rao, S.Q., 489
Raphan, T., 390, 416
Rapin, I., 832
Ratib, S., 520
Rauch, S.D., 182
Rauschecker, J.P., 315, 517
Ravecca, F., 842
Raveh, E., 56
Ray, J., 833
Reardon, W., 465
Rebillard, G., 357
Recanzone, G.H., 518, 541
Reed, N., 207, 224, 261
Refaie, A., 647
Reid, A.P., 590
Reid, M.J., 586
Reiser, M., 419
Reite, M., 324
Rekart, D., 832
Relkin, E.M., 38
Renshaw, J.J., 466
Rentmeester, L., 255
Reschke, M.F., 431
Rescorla, L.A., 846
Resnick, S., 67, 631
Ress, B.D., 465
Resta, RG, 481
Reuven, M., 371, 372
Revit, L.J., 69, 532, 784, 798
Reyes, S.A., 269
Reynolds, E.G., 62, 107
Reznick, J.S., 845
Rhode, W.S., 268
Rhode, W.W., 357
Riazuddin, S., 483–484, 489
Ribera, J., 668
Ricci, A.J., 357
Rice, C., 585
Rice, C.G., 605
Richardson, L.L., 670
Richert, F.M., 767, 772, 773
Rickard, N., 578
Rickard R, 588
Rickards, F.W., 273, 275, 280
Rickenmann, J., 419
Rickert, M., 43
Ricketts, T.A., 592, 764
Ries, D.T., 43, 169, 172, 178, 183
Riggs, D., 676, 680
Riley, A., 520
Ringwalt, S., 670
Rintelmann, W.F., 44, 61, 81, 82, 91, 96, 99, 101, 103, 104
Ritter, W., 343
Rivera, V., 181
Rivier, F., 315
Roach, R., 18
Roberts, J., 144, 148
Roberts, L.E., 520
Robertson, C.M., 583
Robertson, D., 520
Robertson, M.C., 399
Robertson, V.S., 762
Roberts, R.A., 426, 427, 428, 432
Roberts, T.P., 329
Robinette, M.S., 362, 364, 370
Robins, D.L., 586
Robinson, D.W., 599
Robinson, K., 231
Robinson, M., 626
Robinson, P., 72
Robinson, R.O., 587
Robinson, S.K., 650
Rochtchina, E., 631, 637
Rochtchina, R., 632, 636
Rochtina, E., 635
Rodin, E., 320
Rodríguez-Ballesteros, M., 223
Rodriguez, G.P., 43
Roeser, R.J., 62, 235
Rogers, C., 167
Rokugo, M., 386
Roland, J.T., 831
Roland, P., 633
Roland, P.S., 753
Romano, M.N., 295
Romanski, L.M., 517
Rönnberg, J., 669, 678
Roosli, C., 157, 159
Roper, R.G., 605
Rose, K., 262
Rose Mattingly, K., 262
Rosenberg, G., 687, 691
Rosenblatt, C., 468
Rosen, B.R., 239, 242
Rosenfeld, R., 633
Rosenhall, U., 244, 246, 596, 599
Rosenhamer, H.J., 244, 245
Rosen, S., 12, 13, 286, 530
Rosler, G., 167
Rosowski, J.J., 33, 44, 55, 56, 156, 177, 182
Ross, B., 269, 318, 325, 342, 344, 520, 633
Ross, M., 71, 473, 624, 766, 853
Rosso, A., 638
Rotenberg, B., 401
Rothpletz, A.M., 592
Roup, C.M., 141, 142, 144
Rousch, J., 169
Roush, J., 144, 146, 148, 587–589, 760
Roush, P., 771
Roush, P.A., 238, 587–589, 766, 767, 832, 833
Rovatti, V., 404
Rowe, J., 197
Rowe, S., 627
Roy, R.J., 325
Royster, J.D., 43, 595, 598, 609
Royster, L.H., 43, 595, 609
Ruah, C.B., 148, 160
Rubel, E., 633
Ruberry, J., 838
Rubin, C., 508
Rubin, M., 52
Rubin, R., 468
Rubinstein, A., 678
Rubinstein, J.T., 176, 653
Ruckenstein, M., 414
Ruder, L., 617
Rudge, P., 231
Rudmose, W., 17
Rudnick, E., 56
Ruggero, M.A., 268
Ruhm, H.B., 625
Rupp, A., 330
Russell, B.A., 120
Russell, D., 649
Russo, N., 530–531, 537, 562
Russo, N.M., 527, 536
Rutter, M., 585
Ryan, M.M., 238
Ryerson, S., 224
Ryugo, D.K., 517–518

Sabers, D.L., 503
Saber-Tehrani, A.S., 422
Sabes, J., 889, 890
Sabes, J.H., 854
Sabo, D.L., 760
Saccone, P., 57
Sachdev, V., 487
Sachs, M.B., 527, 533
Sachs, R.M., 17
Saenz, M., 315
Safady, S.H., 141, 142, 144
Safran, A.B., 419
Sagi, Y., 520
Sagle, C., 235
Sainz, M., 823

Saito, H., 636
Sakamoto, T., 632
Sakashita, T., 370
Sala, F., 296
Salamon, D.L., 72
Salamy, A., 257
Salem, N., 488
Salminen, N.H., 523
Salomon, G., 215, 675
Saltzman, A., 447–449
Salvini, R., 585
Salvi, R., 43, 208
Salvi, R.J., 647, 654
Saly, G.L., 156, 157
Salz, T., 536
Sammeth, C., 828
Sammeth, C.A., 79, 80
Samo, C.N., 166
Samson, F., 586
San Agustin, T.B., 486
Sanchez, S., 44
Sanders, D.A., 856
Sanders, J.W., 77, 82, 86, 91, 97
Sanders, S., 257
Sando, I., 197
Sanford, C., 156, 159, 160
Sanford, C.A., 137–161, 168, 183, 184
Sanides, F., 317
Sankhyan, N., 242
Santarelli, R., 207–228
Santos, A., 534
Santos, M., 534
Santos-Sacchi, J., 38
Sarampalis, A., 718
Sarff, L., 686
Sass, K., 218, 219
Sato, H., 197, 243, 676
Saunders, G.H., 618
Savio, G., 273
Scalchunes, V., 827, 832
Scarinci, N., 858
Schachern, P.A., 148, 160
Schafer, E., 579, 764, 821
Schaffer, H.I., 43
Schairer, K.S., 156, 165–184, 360
Scharf, B., 517, 520
Schatterman, T., 633–635
Scheibel, M., 635
Scheperle, A., 33
Scherg, M., 318–321, 323, 324, 327
Schery, T., 586, 587
Schiff, S.J., 318
Schissel, H., 586, 587
Schlauch, R.S., 36, 172, 178, 183
Schmerber, S., 418
Schmida, M.J., 470
Schmidt, R., 694
Schmiedt, R., 633, 634
Schmiedt, R.A., 42, 357, 367, 371
Schneider, B.A., 521
Schneider, J., 636
Schochat, E., 318, 321, 575
Schoen, C.J., 489
Schon, D., 534
Schooling, T., 153
Schoonhoven, R., 254
Schotland, L.I., 598
Schow, R., 856
Schraders, M., 488
Schrauwen, I., 489
Schreiner, C., 531, 536
Schreiner, C.E., 518, 534, 541
Schrott, A., 199
Schubert, A., 299, 300, 307
Schubert, E., 783
Schubert, E.D., 67, 71, 89
Schubert, M., 636
Schubert, M.C., 403, 427, 428
Schuck, P., 243
Schuknecht, H., 42, 633, 634
Schuknecht, H.E., 387
Schuknecht, H.F., 42
Schultz, A., 422
Schultz, J.M., 488, 495–496
Schulz, P.J., 671
Schum, D., 691
Schum, R., 650, 652
Schupf, N., 588
Schwaber, M.K., 242
Schwander, T., 125, 126, 170, 173, 183
Schwartz, D., 197
Schwartz, D.M., 89, 306, 307
Schwartz, M., 232, 233
Schwartz, P.J., 492
Schwartz, S., 633
Scimemi, P., 220, 223
Scollie, S., 715, 730, 735, 740, 761, 764, 766, 768–770, 772, 796, 797
Scollie, S.C., 716
Scollie, S.D., 260, 285, 764–767, 769, 771–773
Scorpecci, A., 494
Scott, B.L., 856
Scott, R.E., 659
Scott, S.K., 538
Scusa, M.F., 583
Searchfield, G., 651, 655, 656
Seaton, J., 509–510, 836, 844
Seaver, L., 836, 837, 844, 847
Sedey, A., 459, 842, 845, 846
Sedey, A.L., 249, 831, 832, 835, 839, 845, 846
Seelisch, A., 764
Seely, D.R., 754
Seewald, R., 715, 730, 735, 740, 761, 766–770, 796, 797
Seewald, R.C., 260, 716, 764–767, 769
Selby, G., 429
Selesnick, S.H., 232
Sells, J.P., 169
Selters, W.A., 231, 234, 235, 245
Semar, V., 845
Semple, M.N., 516
Sensesynergy, 855
Sensimetrics Corporation, 857
Serbetcioglu, B., 153
Sersen, E., 588
Setzen, G., 328
Seung, G.Y., 627
Sexton, J.E., 146
Seyfried, D.N., 80
Shahnaz, N., 139, 141, 143, 144, 146, 148, 151, 153, 156, 157, 159
Shahnaz. N., 146, 157
Shakeel, M., 177
Shallop, J.K., 120
Shankar, S., 634
Shanks, J.E., 141, 142, 152, 154
Shannon, R., 262
Shannon, R.V., 539
Shanon, E., 148
Shantz, J., 767
Sharbrough, F., 197
Sharbrough, F.W., 231, 239, 240
Shargorodsky, J., 508
Sharma, A., 352, 538, 540, 728
Sharma, M., 575, 578, 767
Sharma, M.L., 242
Sharma, R., 242
Sharpe, R.M., 325
Shaw, E.A., 78, 155
Shaw, E.A.G., 33, 34
Shea, J.J. Jr, 218, 219
Shehata-Dieler, W., 328
Shelhamer, M., 426
Shemesh, R., 56
Shepard, N.T., 243, 404, 406, 409–411, 413, 414, 416, 422, 429
Shepherd, R.K., 41, 262
Shera, C.A., 33, 44, 177, 358, 359, 360, 363, 372
Sherbecoe, R.L., 44
Sherrington, C., 399
Shestakova, A., 533
Sheykoholeslami, K., 418
Shields, C.G., 639
Shindo, M., 585
Shinn, J.B., 232, 234–236, 239, 240, 241–243, 244, 286
Shipp, D.B., 622, 623, 626
Shisler, L., 447–448
Shi, Y., 198, 241, 651
Shotland, L.I., 493
Shott, S.R., 590
Shulman, S., 447–449
Shumrick, D.A., 38
Shumway-Cook, A., 427, 428, 431
Shurin, P.A., 149, 170
Shy, M.E., 238
Siciliano, G., 842
Siebein, G., 677
Siegel, B., 586
Siegel, J.H., 357, 358, 372
Siem, G., 492
Silman, S., 25, 97, 125, 126, 170, 171, 173, 175, 181, 183, 678
Silva, P., 561
Silverman, C.A., 97, 170, 175
Silverman, F.H., 10
Silverman, S.R., 62, 66, 81, 107
Silverman, W., 588
Simmons, J.L., 156, 157, 159
Simon, H., 854, 855
Simon, U., 171
Simpson, A., 715
Sinclair, J., 676, 680
Sinclair, S.T., 768
Sininger, Y., 207, 465, 521
Sininger, Y.S., 179, 254, 256, 257, 261, 262, 264, 370, 767, 832
Sinininger, Y., 190
Sipilä, P., 54
Sirigu, P., 753
Sirimanna, T., 465
Sismanis, A., 56, 303
Sivakumaran, T.A., 489
Skaar, D., 841
Skinner, B.F., 466
Skinner, M., 801
Skinner, M.W., 719, 826
Sklare, D.A., 78–82, 99
Skoe, E., 531, 532, 536, 538, 561
Sladen, D., 586, 587
Slattery, W.H.III, 522
Sliwerska, E., 489
Sliwinska-Kowalska, M., 484
Smaldino, J., 503, 675–678, 680, 682, 686, 691
Small, B., 634, 637, 641
Small, S.A., 49, 284, 327, 464
Smart, J., 578
Smart, J.L., 119–132
Smirga, D., 805
Smith, A., 251
Smith, B.L., 79, 81
Smith, C.G., 144, 148, 760
Smith, E.L., 604
Smith, J., 639
Smith, J.C., 487
Smith, P., 141, 142, 504
Smith, P.S, 561
Smith, R.J., 596
Smith, R.J.H., 486–487, 583
Smith, S.D., 486–489
Smith, T., 633
Smolakm, L., 181
Smoski, W., 503
Smriga, D., 744, 745
Smucny, J., 318
Snell, D.L., 427
Snell, K.B., 856
Snik, A.F., 489, 767
Snik, A.F.M., 764
Snoeckx, R.L., 488
Snook-Hill, M.M., 592
Snyder, J.M., 41, 79, 81, 82
Sockalingam, R., 686
Sohmer, H., 52, 253, 307
Sokoloff, M., 385
Soli, S., 69, 131, 668, 745
Soli, S.D., 532, 715, 720, 771
Solomon, D., 181
Soloviev, A., 533
Someya, S., 632
Songer, J.E., 182
Song, J.H., 325, 527, 532
Sorri, M., 632
Sotnik, P., 839
Soulières, I., 586
Souza, P., 633, 642
Sowerby, L.J., 401
Spangler, C., 503–504
Sparacino, G., 211
Spazzolini, C., 492
Speaks, C., 12, 69, 191
Speaks, C.E., 65
Spencer, L.J., 845
Sperry, J.L., 68, 72
Spicer, J., 694
Spink, U., 324
Spiro, M.K., 766
Spitzer, M.W., 517
Spitzer, S.B., 262
Spivak, L., 256, 370
Spoendlin, H., 187, 199
Spradlin, J.E., 586
Sprague, B.H., 148, 170
Spratford, M., 771
Squires, M., 504
Sridhar, K.S., 465
Stacey, P.C., 583
Stach, B.A., 120, 181, 183
Stagner, B.B., 465
Stahle, J., 42
Stanberry, B., 700
Stangl, E., 718
Stangl, S., 255
Stanton, S.G., 235
Stapells, D., 198
Stapells, D.R., 120, 252, 260, 261, 273, 275, 276, 279, 280, 283, 284, 288, 326, 327, 349, 366, 464
Starr, A., 179, 190, 207, 210, 219–221, 223, 231, 239, 253, 257, 261, 262, 325, 465, 488–489, 521
Starr, K., 832
Stavroulaki, P., 370
Stayner, L.T., 596
Stecker, G.C., 522
Steeneken, H.J.M., 539
Stefanick, M., 638
Steiger, J.R., 56, 57
Steinberg, J., 66, 67, 676, 767
Stein, J.F., 287
Stein, L., 189, 207, 224, 261
Steinmann, I., 269
Steinschneider, M., 539
Stelmachowicz, P.G., 760, 762, 764, 770, 845
Stenfelt, S., 52, 156, 159
Stephens, D., 465, 669, 852
Stephenson, W.A., 320
Stevens, G., 664
Stevens, H.P., 132, 477
Stevens, J., 255
Stevens, K.N., 69
Stevens, S.S., 63, 66, 90, 107
Stewart, B.J., 649
Stewart, I., 561
Stewart, J., 831
Stewart, K., 626
Stewart, M.G., 310, 312, 325
Stillman, R.D., 543
Stinson, M.R., 155
Stinson, M.S., 503
St. John, P., 120, 179, 254, 262
Stockard, J., 197
Stockard, J.E., 231, 239, 240, 254

Stockard, J.J., 231, 239, 240, 254
Stockwell, C.W., 386, 411
Stoel-Gammon, C., 844
Stoinska, B., 583
Stollman, M.H., 767
Stone, M.A., 715, 786
Stone, W.L., 586
Stoppenbach, D.T., 65, 120, 141, 142, 144
Storey, L., 766
Stouffer, J.L., 650, 652
Stover, L., 363, 364, 367, 372
Strait, D.L., 534, 540, 541
Straka, H., 386
Strange, P.H., 80
Straumann, D., 405
Stredler-Brown, A., 503
Strickland, A.E., 359
Stromgren, T., 650
Strömgren, T., 670
Strom, L., 650, 670
Strom, T.M., 488
Strother, D.E., 120
Strouse, A., 69, 70, 71
Strutz, J., 635
Stuart, A., 167, 260
Stuck, C., 751
Stucker, F., 166
Studdert-Kennedy, M., 538
Studebaker, G., 19, 39, 78
Studebaker, G.A., 44, 57, 58, 67, 77, 79–82, 85, 92–94, 99, 101, 104, 105
Stufflebeam, S.M., 239, 242
Stürzebecher, E., 271
Subramaniam, M., 598, 603, 604
Suffield, J.B., 261
Suga, N., 540
Suga N., 518
Sugata, K., 486
Sulkers, J., 764
Sullivan, J., 69, 668
Sullivan, J.A., 131, 532, 715
Summerfield, A.Q., 583
Summerfield, Q., 786
Sunderland, S., 306
Surr, R., 89, 720
Surr, R.K., 717
Suter, A.H., 600
Sutherland, G., 698
Sutton-Tyrrell, K., 631, 632, 635
Suzuka, Y., 633
Svanborg, A., 596, 599
Svihovec, D.A., 66
Svihovec, D.V., 81
Svirsky, M.E., 831
Swanepoel, D., 662, 664, 667, 700
Swanepoel, D.W., 39, 662, 669
Sweetow, R., 853, 889, 890
Sweetow, R.W., 854
Swets, J.A., 365
Swindeman, J.G., 94
Swinkels, S., 586
Synnes, A.R., 583
Szabo, P., 262
Szalda, K., 268
Szymko-Bennet, Y.M., 493

Tack, D., 487
Taiji, H., 238
Tait, C., 169
Taitelbaum-Swead, R., 831
Takahashi, H., 197
Takebayashi, T., 636
Talbott, E., 631, 632, 635
Talebizadeh, Z., 486
Tallal, P., 536, 538
Tallon-Baudry, C., 326, 327
Talmachoff, P.J., 249
Talmadge, C.L., 358, 359, 371
Tamai, F., 585
Tanaka, C., 199, 200, 237
Tanaka, H., 236
Tanaka, Y., 328, 585, 590
Taniguchi, I., 518
Tannahill, C., 503
Tao, P., 647
Taveras, J.M., 328
Tavor, I., 520
Taylor-Jenafreau, J., 120
Taylor, J.L., 534
Taylor, R., 177
Taylor, W., 596
Teaching Research Institute, 591
Teagle, H.F., 832, 833
Teagle, H.F.B., 238
Teas, D., 198
Teas, D.C., 251
Tedesco, S., 262
Tegel, L., 396
Teich, M.O., 268
Teig, E., 492
Teissier, N., 249
Telian, S., 833
Telian, S.A., 306, 406, 409, 410, 411, 413, 414, 416, 429, 832
Temple, E., 536
Tenhaaf Le Quelenec, J., 772
Terkildsen, K., 138–139, 253
Tetzlaff, J.E., 299, 300, 307
Thal, D., 831, 845
Thal, D.J., 831
Tharpe, A.M., 462, 466, 470, 586–589, 592, 760, 764, 769
Theissing, G., 61
Theodoros, D., 686
Thiele, N., 764
Thisted, R., 631
Thivard, L., 536
Thomas, E., 830
Thompson, D., 504
Thompson, D.J., 17
Thompson, G., 467, 471, 589, 590
Thompson, M., 467, 471
Thompson, M.D., 472
Thomsen, K.A., 138–139
Thomson, V., 443
Thordardottir, E.T., 65, 120
Thorkelsson, T., 583
Thorne, M.C., 489
Thornton, A.R., 33, 44, 66, 177, 237, 318, 320, 325, 326
Thornton, A.R.D., 200
Thornton, C., 325
Thorpe, R., 631
Thurlow, W.R., 81
Tibbils, R.P., 219
Tieri, L., 494
Tierney, A., 534, 541
Tiitinen, H., 523, 532
Tillman, T., 94, 676, 678
Tillman, T.W., 64, 65, 67, 68, 107
Timothy, K., 492
Tinetti, M., 642
Tinker, J., 197
Tjellstrom, A., 817
Tlumak, A.I., 283
Tobey, E.A., 72, 826, 831, 832, 846
Tobias, J.V., 94
Todd, M.J., 403
Tomblin, J.B., 845
Tomiyasu, U., 635
Tomlin, D., 273
Tomlinson, R.D., 418
Tong, H., 638
Tong, H.M., 405
Tonndorf, J., 49–54, 61, 94
Towers, A.D., 668
Towers, H.M., 262
Towey, K., 504
Towle, C., 700
Toyoda, K., 328
Tran, K., 735
Trautwein, P., 832
Treffers, P.D., 846
Tremblay, K., 349, 350, 352, 353, 633, 634
Tremblay, K.L, 340, 342, 345, 349
Trine, M.B., 144
Trine, T., 720
Trivette, C.P., 70
Truex, R.C., 315
Trychin, S., 857, 858
Tsakanikos, M., 619
Tsiakpini, L., 842
Tsilou, E.T., 488, 495
Tsurukiri, J., 325
Tsutsumi, T., 492
Tubaugh, L., 159
Tubs, J.L., 717
Tueller, S.J., 453
Tuley, M., 675
Tun, P., 634, 637, 641
Turczyk, V.A., 149
Turner, C., 326
Turner, J., 634, 635
Turner, R.G., 99, 119, 462
Turno, F., 753
Turpin, L.L., 252, 258
Turrini, M., 224
Turunen-Rise, I., 605
Tusa, R.J., 403, 413, 427, 428
Tutshini, S., 662, 667
Tvete, O., 605
Tweed, T.S., 142, 144
Tye-Murray, N., 697, 698, 857
Tyler-Krings, A., 845
Tyler, R., 647, 649, 651, 653
Tyler, R.S., 647, 649–653
Tzur-Moryosef, S., 520

Uchanski, R., 691
Ueki, Y., 486
UK National Screening Committee, 449
Ulanovski, D., 56
Ulmer, E., 517, 520
Umat, C., 578
United States Preventive Services Task Force (USPSTF), 443
Uppenkamp, S., 183, 517–518
Urbano, R., 471
Urben, S.L., 232, 246
Ursino, F., 842
US Department of Education, 835
US Department of Health and Human Services (HHS), 442, 814
US Department of Health, Education, 437
Usher, L., 583
US Preventive Services Task Force, 437
Uyehara-Isono, J., 838–839
Uygen, P.L., 488

Valente, M., 777, 781, 782, 789, 795, 802, 803
Valtonen, J., 532
Van Bourgondien, M.E., 585
van Buuren, R.A., 762
Van Campen, L.E., 79, 80
Van Camp, G., 485–487, 490
Van Camp, K.J., 139, 151
Van de Heyning, P., 823
van den Borne, S., 767
van der Gaag, R.J., 586
Vandermosten, M., 287
Van der Reijden, C.S., 275
Vander Werff, K.R., 286
Van Dun, B., 275
Van Dyke, D.C., 472
Van Eldik, T., 846
Vanhuyse, V.J., 139, 151
van Kamp, I., 605
Van Maanen, A., 279, 283
Van Opstal, A.J., 522
van Orsouw, L., 586
Van Riper, L.A., 491
Van Riswick, J.A., 522
Van Roon, P., 464
Van Rybroek, J.M., 487
Van Tasell, D., 780
Van Tassell, D.J., 65
Varga, R., 488
Vasant, K., 750
Vavrek, M.J., 79
Veeman, J.W., 846
Velez, R., 675
Venediktov, R.A., 763
Venem, M., 750
Ventry, I., 631, 638
Ventry, I.M., 32, 41, 64, 116, 620–621
Verhagen, W.I., 487
Verhulst, F.C., 846
Vermiglio, A., 197
Vernon, J., 651
Verschurr, C., 402
Vershuure, H., 740
Vesterager, V., 675
Vethivelu, S., 472
Vibert, D., 419
Vibert, N., 386
Vidal, P.P., 386
Viemeister, N.F., 278
Viirre, E.S., 650
Viljanen, A., 632
Villchur, E., 32, 78
Vincent, C., 488
Vinck, B.M., 366, 367
Virre, E., 668
Vivion, M.C., 325
Vizioli, L., 328
Vohr, B.R., 256, 370
Volding, L., 587
Volkmar, F., 585
Volkov, I.O., 539
Vollrath, M.A., 383
von Ammon, K., 233
von Baumgarten, R.J., 419
von Cramon, D., 318, 321, 323, 324, 327
von Hapsburg, D., 470
Voss, S.E., 33, 44, 155–157, 177
Vostanis, P., 846
Vosteen, K.H., 42
Vowler, S.L., 728

Waespe, W., 416
Wahlgreen, D., 202
Wahlgreen, O., 276
Waite, L., 632, 638, 639
Walden, B., 720, 789
Walden, B.E., 717
Waldman, D., 760
Waligora, J., 488
Walker, E.A., 771
Walker, K.M.M., 517
Walker, M.F., 405
Wallace, M.T., 586
Wallace, R., 638
Wall, G.M., 753
Wall, R., 592
Wall, R.S., 592
Walsh, T.E., 81
Walters, K.Z., 670
Walton, J., 633–635
Walton, J.P., 667
Waltzman, S.B., 827, 831, 832
Wang, J., 208, 635, 639
Wang, J.J., 632, 636
Wang, N.Y., 831
Wang, Q., 489
Wang, Q.J., 489
Wang, X., 531
Wang, Y., 588, 635
Ward, C.D., 694
Ward, W.D., 43, 172, 178, 183, 598, 600
Ware, J.E., 429
Waring, M., 257
Wark, D., 849
Warner, D., 668
Warren, J., 846
Warrier, C.M., 531, 538
Warr, W.B., 357

Wartinger, F., 610
Washburn, L.K., 583
Watkin, P.M., 439–440
Watson, C.S., 36
Watson, D.R., 232, 243, 244, 246
Watson, K.N., 56, 57
Watson, R., 541
Watt, M.J., 583
Watts, K.A., 70
Waxman, G.M., 465
Wayner, D., 698, 699
Weakley, D.G., 69, 70
Weatherby, L.A., 170
Weaver, R.S., 385
Weber, F., 325
Weber, J., 710
Weber, S.C., 175
Webster, D., 188
Webster, J.C., 243
Wedenberg, E., 183
Weegerink, N.J., 488–489
Wegel, R.L., 99
Wegner, O., 202, 255
Weichbold, V., 842
Weider, D.J., 238
Weinberger, N.M., 518, 520
Weinstein, B., 631–633, 635, 636, 638, 642, 643, 698
Weinstein, B.E., 116, 783
Weinstein, M.C., 365
Weiss, E., 19
Weissenbach, J., 477, 488
Weissler, P., 17
Weiss, M., 635
Welgampola, M.S., 182
Werner, L.A., 159, 589
Wertz, D., 575
Wesendahl, T., 668
Wesilender, P., 71
West, D., 508
Westerberg, B.D., 146, 157, 159
Westermann, S., 740
Westlake, H., 36
Westmoreland, B., 197, 254
Weston, M.D., 488
Wetzig, J, 419
Wetzig, J., 419
Wever, E.G., 207
Whitaker, S., 218
Whitechurch, M., 238
White, K., 460
White, K.R., 438–440, 442–444, 447–453, 450, 583
Whitelaw, G.M., 677, 678
White-Schwoch, T., 537
White, S.D., 91, 735
Whitney, I., 504
Whitney, S.L., 417
Whyte, A., 182
Wible, B., 538–539
Wichmann, W.W., 233
Widarsson, J., 670
Widen, J., 471
Widen, J.D., 468
Widen, J.E., 120, 256, 369, 371, 444, 451, 468, 590
Wiegand, D.A., 181
Wiener, W.R., 592
Wie, O.B., 828
Wiet, R.J., 42, 232
Wiggin, M., 845
Wigney, D., 767
Wilber, L., 65
Wilber, L.A., 10, 15, 18, 19, 78–81, 99
Wilcox, E.R., 483
Wild, E.L., 449
Wilensky, D., 120, 179, 254, 262, 451
Wiley, S., 827, 842
Wiley, T., 638
Wiley, T.L., 65, 68, 120, 141, 142, 144, 148, 152, 153, 155, 167, 169, 170
Wilkinson, A.E., 418
Wilkinson, A.R., 224
Williams, C.E., 67
Williams, D.A., 70
Williams, J., 590
Williams, M.J., 465
Williams, S., 752
Williams, V., 634, 637, 641
Williams, W., 607
Willis, M., 215
Williston, J., 189, 253
Williston, J.S., 295
Willott, J., 350
Wilmington, D.J., 603
Wilson, B.A., 828
Wilson, B.S., 821
Wilson-Costello, D., 583
Wilson, I., 71
Wilson, J.M.G., 438, 455
Wilson, L., 232, 312
Wilson, M.J., 274, 277, 278, 282
Wilson, P.H., 650–652, 655
Wilson, R., 107, 637, 638
Wilson, R.H., 44, 61–65, 68–72, 152, 154, 169, 170, 183
Wilson, W.R., 468, 471, 589, 590
Winer, J.A., 315, 317
Winnick, J.P., 587
Wisniewski, S., 635
Witt, S., 649
Witt, S.A., 649, 650, 652, 653
Wolfe, J., 764, 821
Wolf, M., 642
Wolf, P., 635
Wolf, S.J., 120
Wolpaw, J.R., 320
Wong, P.C., 536
Won, J.H., 344, 352
Woodard, J., 832, 833
Wood, C.C., 320
Wood, D.L., 324, 327, 328
Woodworth, G.G., 176
Wootton, R., 659
World Health Organization (WHO), 438, 439, 601, 659, 664, 665, 849, 850
Wormald, P.J., 167
Worrall, L., 858
Worsfold, S., 443
Wouters, J., 285
Wright, B.A., 538
Wright, D.C., 760
Wu, B.J., 257
Wu, H., 638
Wu, Y., 718
Wu, Y.Y., 224
Wygonski, J., 539

Xia, J., 527, 631, 635
Xiao, Z., 540
Xue, Q., 631, 635
Xue, Q.L., 527
Xue, Y., 489
Xu, Y., 530–531, 535
Xu, Z-M, 327

Yacullo, W.S., 78, 79, 81, 92–94, 96–98, 104, 105, 107, 109
Yaffe, K., 527, 631, 635
Yamasoba, T., 632
Yamoah, E.N., 385
Yang, C.H., 285
Yang, E.W., 260
Yang, E.Y., 284
Yang, F., 489
Yang, W.S., 831
Yang, Y., 260
Yardley, L., 402
Yasan, H., 54
Yasargil, M.G., 233
Yasui, Y., 583
Yates, G.K., 208
Yavor, R.A., 403
Yeager, D., 12, 22
Yeargin-Allsopp, M., 585
Yi, H.G., 531
Yilmaz, Y., 587
Yin, R., 224
Yin, T.C.T., 517
Yip, F., 578
Yokoyama, K., 243
Yoo, J., 858
Yoon, T.H., 148, 160
Yoshinaga-Itano, C., 249, 459, 835, 838, 839, 842–846
Yoshioka, K., 418
Yost, W.A., 828
Young, E.D., 527, 533
Young, L., 63, 64
Young, Y.H., 418
Yrttiaho, S., 523
Ysunza, A., 284
Yueh, B., 642
Yüksel, S., 587
Yvert, B., 317

Zalewski, C., 493
Zalewski, C.K., 487, 494
Zapala, D.A., 148, 465
Zappia, J.J., 232
Zatorre, R.J., 534, 541
Zdanski, C.J., 238
Zecker, S., 531, 538, 540, 562, 576–579
Zecker, S.G., 537–539
Zee, D.S., 181, 389, 390, 392, 394, 395, 404, 405, 413, 426
Zeisel, S., 144, 148
Zelterman, D., 148, 160
Zeng, F.G., 219, 220, 262, 521, 539
Zettel, M., 635
Zhang, J., 631
Zhang, V., 767
Zhang, Y., 489
Zhao, X., 385
Zhao, Y.L., 489
Zheng, J., 357
Zhiqi, L., 149
Zhiwu, H., 149
Zhou, B., 56
Zhou, Y., 224, 635
Zhu, X., 633, 635
Zigman, W., 588
Zilbovicius, M., 536
Zinreich, J.S., 181
Zmuda, J., 635
Zonderman, A., 631
Zong, L., 489
Zubritsky, C.D., 585, 586
Zuniga, M., 636
Zuniga, M.G., 182
Zwack, G., 667
Zwicker, E., 362
Zwislocki, J., 78–80, 732
Zwislocki, J.J., 17
Zwolan, T., 830, 832
Zwolan, T.A., 825, 827, 832, 833

SUBJECT INDEX

Note: Page number followed by f and t indicates figure and table respectively.

AAA. *See* American Academy of Audiology (AAA)
AAA Childhood Hearing Screening Guidelines, 147–148
A-ABR. *See* Automated auditory brainstem response (A-ABR)
AAP. *See* American Academy of Pediatrics (AAP)
ABA. *See* American Board of Audiology (ABA)
Abbreviated Profile of Hearing Aid Benefit (APHAB), 640t, 783
ABIs. *See* Auditory brainstem implants (ABIs)
ABR. *See* Auditory brainstem response (ABR)
ACC. *See* Acoustic change complex (ACC)
ACE. *See* Advanced Combined Encoder (ACE)
ACGIH. *See* American Conference of Governmental Industrial Hygienists (ACGIH)
Acoustic admittance, peak-compensated static, 141–143
Acoustical Society of America (ASA), 10, 505
Acoustic calibrators, 13
Acoustic change complex (ACC), 344–345
Acoustic immittance
  devices, 25–26
  measurements, 621–622
Acoustic masking procedures, 92, 103–109
Acoustic modifications of room, for hearing loss population, 680. *See also* Room acoustics and auditory rehabilitation technology
Acoustic onsets. *See also* Human brain
  defined, 537
  in human brain, 537–539
  role in perception of speech, 537
Acoustic reflex, 120, 121t
  and audiologic test battery, 465
  in diagnosis of nonorganic hearing loss, 621
  otosclerosis and, 489
  word recognition scores and, 487
  X-linked nonsyndromic hearing loss and, 488
Acoustic reflex thresholds (ARTs), 119
Acoustic signals
  central auditory processing disorder and, 545
  in digital signal processor, 707
  earmolds and, 709
  frequency modulation amplification, 682
  hyperacusis and, 654
  and language development, 677
  neural processing of, 536
  telecoil-equipped hearing aids and, 688
Acoustic stapedius reflex (ASR), 165
  adaptation of, 183
  anatomy related to, 165
  automated tests, 169
  contralateral pathway, 165, 166f
  decay test, 171–172, 172f
  electrically evoked, in patients with cochlear implants, 172–173
  functional significance, theories of, 166–167
  ipsilateral pathway, 165, 166f
  pathways of, 165–166, 166f
  response growth, 165–166, 166f
  threshold. *See* Acoustic stapedius reflex threshold
Acoustic stapedius reflex threshold
  gender and age effects, 170–171
  ipsilateral and contralateral, patterns of, 173, 173f
  measurement of, 167–171
    activators, 167
    admittance, 167
    instrumentation, 168, 168f
    methodology, 168–169, 169f
    new methods of, 183–184
    probe, 167
    terminology, 167–168
    test conditions, 168, 168t
  normative data, 169–170
  and otologic disorders, 173
    auditory neuropathy spectrum disorder, 179–180
    central nervous system disorders, 177–181
    conductive hearing loss, 176–177
    demyelinating and cortical disorders, 181
    extra-axial brainstem disorders, 180
    facial nerve paralysis, 175–176, 176f
    functional hearing loss, 183
    hearing loss of cochlear origin, 173–175, 174f, 175f
    intra-axial brainstem disorders, 180–181
    retrocochlear and brainstem disorders, 177–181
    superior canal dehiscence, 181–182
    vestibular schwannoma, 177–179
  pharmacological effects, 171
  use of, 167
Acoustic telephone programs, 755
Acoustic transients, calibration of, 21–25, 25f
Acoustic tumors, 42. *See also* Vestibular schwannomas
  ABR measure for, 198–200
  ABR testing and MRI in, 232–233
Active head rotation (AHR), 413
ADA. *See* Americans with Disabilities Act (ADA)
Adaptation test (ADT), 422
Adaptive signal processing strategies, 681
ADC. *See* Analog-to-digital converter (ADC)
Adenine, 871
ADT. *See* Adaptation test (ADT)
Adults, nonorganic hearing loss in, 617–618
Advanced Bionics (AB), 819
Advanced Combined Encoder (ACE), 821
AEPs. *See* Auditory-evoked potentials (AEPs)
AGC. *See* Automatic gain control (AGC)
Age-related hearing loss (ARHL), 631
  aging auditory mechanism, 632–635
  audiologic findings, 636–638
  diagnostic and management protocol, 639–642
  future perspectives, 643
  geriatric syndromes associated with, 635–636
  hearing screening, 642–643
  and medical comorbidities, 631–632
  psychosocial consequences of, 638–639
  risk factors for, 632
Aging
  effect of
    on ABR interwave intervals (IWIs), 197
    on ASR thresholds, 170
    on tympanometry measures, 144
  and hearing loss, 42
Aging auditory mechanism, 632–635
AHR. *See* Active head rotation (AHR)
AICA. *See* Anteroinferior cerebellar artery (AICA)
Aided audiogram, 772
Aided sound-field thresholds, 802
Air-bone gap, 38
Air conduction
  hearing aids, 590
  HPD and, 609
  puretone thresholds, 636
  signal, 620
Air-conduction audiometry, cross-checks for, 119
Air-conduction testing
  cross-checks for, 120
  masking, 77–82
Air-conduction (AC) thresholds, 31
ALD. *See* Assistive listening device (ALD)
Aliasing, 193–194
Allele, 871
Alport syndrome, 490–491
Amblyaudia, treatment for, 575. *See also* Central auditory processing disorder (CAPD)
American Academy of Audiology (AAA), 3, 9, 502, 545, 663
American Academy of Audiology Clinical Practice Guidelines, 682
American Academy of Audiology Guideline for the Audiologic Management of Adult Hearing Impairment, 639
American Academy of Pediatrics (AAP), 444
American Board of Audiology (ABA), 510
American Conference of Governmental Industrial Hygienists (ACGIH), 600, 886
American Institute of Balance (AIB) Dynamic Visual Acuity Test, 430
American National Standard Method for Coupler Calibration of Earphones, 731
American National Standards Institute (ANSI), 9, 505, 604, 675
  audiometers, specification for, 32
  classroom acoustics, 505
  contact information, 10
  hearing aid tests, 732–739, 733–734t
  SLM standards, 675
  standards for acoustic immittance systems, 146–147
  standards for audiometers and audiometric testing, 11t

American Sign Language (ASL), 832, 836
American Speech-Language-Hearing Association (ASHA), 9, 545, 663
  central auditory processing disorder, 545
  guidelines for audiologic screening, 147
  pediatric audiologic practices, 463
  spondaic words, 63, 64t
Americans with Disabilities Act (ADA), 694, 836
American Telemedicine Association (ATA), 659
Amino acid, 871
Aminoglycoside ototoxicity, in older adults, 635
Ampclusion, 748
Amplification, 577–581. *See also* Central auditory processing disorder (CAPD)
  behavioral verification of, 581
  candidacy for, 579
  classroom amplification systems, 579
  conventional hearing aids, 579
  electroacoustic verification of, 580, 580f
  remote microphone hearing aids, 577, 577f
  research, 577–579
  school and teacher guidance, 581
Amplitude compression, 763
Ampulla, 384
AN. *See* Auditory neuropathy (AN)
Analog oscilloscope, 13
Analog-to-digital converter (ADC), 190, 192–194
  aliasing, 193
  bioamplifier gain, 193
  dynamic range, 193
  least significant bit (LSB), 193
  low-pass filter, 194
  Nyquist rate, 193–194
  quantization, 193
  quantization error, 193
  sixteen-bit, 193
Aneuploidy, 484, 871
Annoyance hyperacusis, 654
Annoyance, of everyday sounds, 892
ANSD. *See* Auditory neuropathy spectrum disorder (ANSD)
ANSI. *See* American National Standards Institute (ANSI)
ANSI/ASA. *See* The American National Standards Institute/Acoustical Society of America (ANSI/ASA)
ANSI/ASA S3.35, Method of Measurement of Performance Characteristics of Hearing Aids under Simulated Real-Ear Working Conditions, 735
ANSI/ASA S3.46, Methods of Measurement of Real-Ear Performance Characteristics of Hearing Aids, 736
ANSI/ASA S3.42/Part2 Methods for Characterizing Signal Processing in Hearing Aids with a Speech-Like Signal, 735–736, 735f
ANSI/ASA S3.42 Part 1, Testing Hearing Aids with a Broad-Band Noise Signal, 735
ANSI/ASA S3.22, Specification of Hearing Aid Characteristics, 732–733, 735
  troubleshooting using, 736–739
Anterior lenticonus, 871
Anterior vestibular artery, 385
Anteroinferior cerebellar artery (AICA), 385
Anteroventral cochlear nucleus (AVCN), 515
Antialiasing filter, 194
APDs. *See* Auditory processing disorders (APDs)
APHAB. *See* Abbreviated Profile of Hearing Aid Benefit (APHAB)
APTO. *See* Auditory pattern temporal ordering (APTO)
AR. *See* Audiologic rehabilitation (AR)
ARHL. *See* Age-related hearing loss (ARHL)
Artificial ear method, 17–18
ASA. *See* Acoustical Society of America (ASA)
ASD. *See* Autism spectrum disorder (ASD)
ASHA. *See* American Speech-Language-Hearing Association (ASHA)
ASL. *See* American Sign Language (ASL)
Asperger's disorder, 585
ASR. *See* Acoustic stapedius reflex (ASR)
Assistive listening device (ALD), 577, 675
Assistive technology device, defined, 876
Assistive technology service, defined, 876
ASSRs. *See* Auditory steady-state responses (ASSRs)
Asynchronous telehealth. *See* Store-and-forward telehealth
AT. *See* Auditory training (AT)
ATA. *See* American Telemedicine Association (ATA)
Ataxia, 400–401
Attenuator linearity, checking of, 16
Audiograms, 37–38, 37f
  classification, 38, 38t
  conductive hearing loss (CHL)
    from osteomas, 53, 53f
    from otitis media, 54, 55f
    from otosclerosis, 54, 54f
  hearing-loss configuration and, 39, 39t
  large vestibular aqueducts, hearing loss from, 56, 56f
  mean CHL from children, 54, 54f
  mixed hearing loss, 55, 55f
  normal-hearing sensitivity, 53, 53f
  pseudoSNHL from intracranial hypertension, 57f
  SNHL from presbycusis, 54, 55f
  superior semicircular canal dehiscence, hearing loss from, 56f
  tympanic membrane perforations, 43–44, 44f
  type of hearing loss and, 38–39, 38f
  unmasked and masked puretone thresholds, 100–101, 100f
Audiologic evaluation, of hearing loss, 825. *See also* Cochlear implants (CIs)
Audiologic intervention, telehealth service for, 668. *See also* Teleaudiology
Audiologic practice, building and growth of
  cost calculation, case study on, 812
  equipment needs and space consideration, 811–812
  first decisions, 806–809
  historical perspectives, 805
  marketing, 813
  office as marketing tool, 812
  paperwork for, 814
  personnel, 812–813
  pricing, 813–814
  private practice and personality types, 805–806, 806f
  space requirements, 809–811
Audiologic procedures using telehealth, 667t
Audiologic rehabilitation (AR), 849–851, 850t
  candidacy types, 852t
  counseling, 851–853
  future perspectives, 858–859
  options for therapeutic AR intervention, 854–858
Audiologic test battery, 119. *See also* Diagnostic audiology
  for birth to 6 months of age infants
    electrophysical assessments, 463–465
    immittance, 465
  for infants 6 months of age and older, 465–466
    behavioral observation audiometry, 466
    conditioned play audiometry, 472
    instrumental/operant conditioning, 466
    test room arrangement, 467, 467f
    visual reinforcement audiometry, 468–472
Audiologists
  cost assessment, case study on, 812
  counseling role in, 851–853
  decisions of, 806–809
  glove use guidelines for, 863t
  hand hygiene guidelines for, 864, 864t
  marketing by, 813
  masks, eye protection, and gowns for, 864
  office as marketing tool, 812
  paperwork for, 814
  personnel hiring, 812–813
  pricing, 813–814
  private practice and personality types, 805–806, 806f
  role of, 640, 642–643
  space requirements, 809–811
Audiology, 3
  defined, 3, 875
  evolution of, 3–4
  infection control in, 861
    implementation of infection control principles, 862–865
    relevance of, 861–862, 861t
    terms, definition of, 865t
    written infection control plan, 865–867
  telehealth service for, 664–666
Audiometers, 31
  automatic, 21
  calibration, 9. *See also* Calibration
  price of, 811
  puretone, 14–16
    basic signal, 14
    biologic check, 14–16, 15f
    frequency check, 16
    harmonic distortion check, 16
    linearity check, 16
    rise–fall time, 16
  speech, 19
  types of, 32
Audiometric interpretation, 37–39
Auditory and nonauditory consequences of hearing loss, 851t
Auditory brainstem implants (ABIs), 817
Auditory brainstem response (ABR), 205, 207, 249, 307, 451, 463, 529, 659, 825. *See also* Auditory-evoked potentials (AEPs)
  age effects, 257

air-conducted tone burst stimuli and, 258–259
audiologic test battery, 463
auditory-evoked potentials, 625
auditory function, estimation of, 250
and auditory nerve involvement, 233
auditory nerve aplasia/agenesis, 238
auditory nerve hypoplasia, 238
auditory neuropathy spectrum disorder, 237
Charcot-Marie-Tooth syndrome, 238
vascular loop syndrome, 238–239
vestibular schwannoma, 233–237
auditory neuropathy/auditory dys-synchrony and, 262–263
behavioral thresholds and thresholds of, 260
bone-conducted stimuli and, 259–260
brain activity measurement, 529
and brainstem involvement
absence of waves, 240–241
absolute latency delay, 239
brainstem disorders, 241–243
contralateral effect, 241
interear latency comparisons, 240
interwave latency delay, 239–240
lesions of auditory brainstem pathway, 239
repetition rate shifts, 241
sensitivity and specificity, 241
wave V to wave I amplitude ratio, 240
case studies, 263
moderate hearing loss, 263–264
normal hearing, 263
cautions and considerations in use of, 261
ear canal collapse, 261
middle-ear function, 261
neural abnormalities, 261
reporting test results, 261–262
subject noise, 261
chirps, 202–204
CI candidacy process, 825
click polarity and, 197–198
click rate and, 198
cochlear hearing loss, effect of, 243–245
high-frequency hearing loss, formula for, 245–246
derived-band, 200–201, 200f
diagnostic aspects, 233
differential diagnosis, 231–246
frequency-specific stimuli, 250
frequency specificity *vs.* neural synchrony, 251
frequency specificity *vs.* place specificity, 251
types of, 250–251
use of, 250
gender effects, 257
generator sites of, 231–232
in head injury, 243, 243f
hearing loss phenotype, 485
hearing sensitivity, estimation of, 249–264
in heavy metal exposure, 242–243
human, 189, 189f
in hyperbilirubinemia, 242
and imaging studies in acoustic tumors, 232–233
masking, use of, 261
in multiple sclerosis, 241–242
peak generators, 187–188
for pediatric patients, 257–260, 258t
approaches, 257–258
neural integrity, 258, 258f
recording, 255
electrode montages, 256
filter settings, 256
number of averages and noise quantification, 256–257
one- *vs.* two-channel, 256
time window, 256
stacked ABR amplitude, 201
stacked ABR approach, 199–202
stimulus, 252
chirps, 254–255
envelope of, 252
frequency, 252
intensity, 252–253
polarity, 253–254
rate, 253
stimulus manipulations on, effects of, 198–204
test efficiency and accuracy, maximizing of, 260–261
test protocol for older children and adults, 261
threshold testing
recording considerations in, 255–257
stimulus considerations in, 251–255
subject considerations in, 257
tone bursts
2-1-2 envelope, 252
envelope characteristics, 252
use of, 251
tumor detection, 198–199
in tumors of brainstem, 242
use of, 250
Auditory cortex (AC), 315
speech envelope in, 540
Auditory dyssynchrony (AD), 825
Auditory-evoked potentials (AEPs), 187, 188, 249, 462, 625, 668. *See also* Nonorganic hearing loss
analog-to-digital conversion, 190, 192–194
auditory brainstem response. *See* Auditory brainstem response (ABR)
auditory steady-state response, 189
bioamplifiers, 190
classification, 188–189
compound action potential, 189
conceptual framework of, 316–318
populations of neurons in CNS, 316–317, 317f
cortical, 337
aging effects, 349
amplitudes of, 337
and auditory training, 352–353
clinical applications, 349–353
and cochlear implants, 351–352
EEG *vs.* MEG, 339
electrode configurations and acquisition parameters, 339–340
exogenous and endogenous aspects, 338–339
hearing loss and hearing aids, 349–351
latency of, 337–338
maturational effects, 349
passive recordings, 338
patient subject factors, 349
preattentive, 338–339
types of, 340–348
use of, 337
digital microprocessor, 191
analog representation of sinusoid, 191, 192f
binary number system, 191–192
digital-to-analog conversion, 194
electrodes for, 188, 190–191
event-related potential, 189
for hearing assessment, 759
for hearing screening, 190
instruments, 21–25
for intraoperative monitoring, 190
long-latency, 328
in mapping of cochlear implants in children, 190
middle-latency, 315
age effects, 324–325
analysis strategy, 320–322
anatomic frame of reference, 315, 316f
bandwidth, 319
clinical use, 327–329
in cochlear implant evaluation, 328–329
gender effects, 324–325
handedness effect, 325
hearing loss on, effects of, 327
in neurologic disease, 324
Pa, 322–323
Pb, 323–324
in psychiatric research, 329–330
recording, 318–320
reference electrode, 320
site-of-lesion testing, 327–328
and speech-evoked ABR, 325
state variables, 325
stimulus considerations, 325–327
subject variables, 324–325
threshold estimation, 327
in tinnitus, 324–325
TP41, 324
waveform, 319, 319f, 320, 321f
middle latency response, 189
noise reduction, 194–197
filtering, 195–196
signal averaging, 196–197
in nonorganic hearing loss, 625
normative aspects, 197–198
response recording, 190–191
common lead, 191
electrode application, 190–191
inverting lead, 191
noninverting lead, 191
scalp locations, 190
scalp preparation, 190
for site-of-lesion testing, 190
slow vertex potential, 189
stimulus variables, 197–198
subject variables, 197
time-domain signal averaging, 190, 196
uses of, 190
Auditory function, intraoperative monitoring of, 306–309

Auditory hallucinations and tinnitus, 648
Auditory nerve, 187–188, 513–515, 514f
  asynchronous activity in, 634
  function , loss of, 634
Auditory nerve aplasia/agenesis, ABR findings in, 238
Auditory nerve hypoplasia, ABR in, 238
Auditory nerve response telemetry (ART), 829
Auditory neuropathy (AN), 41, 190, 207, 219
  ECochG potentials in, 219–224
Auditory neuropathy spectrum disorder (ANSD), 179–180, 262, 451
  ABRs in, 237, 262–263
  and ASR threshold, 179–180
  clinical presentation, 262
Auditory oral communication approach, 841
Auditory pattern temporal ordering (APTO), 546
Auditory processing disorders (APDs), 684
Auditory processing evaluation, referral for, 131
Auditory sense organ, critical risk factor for, 633
Auditory skills assessment (ASA), 554
Auditory steady-state responses (ASSRs), 189, 267–268, 463, 759
  in adults, 273–274
  averaging and, 276
  calibration, 278
  case study, 288, 289f
  clinical applications, 278
    ASSR *vs.* ABR, 281–282
    audiogram prediction, 278–281
    bone conduction, 284
    cochlear implant mapping, 284–285
    hearing aid fitting, 284–285
    hearing threshold estimation, 278–281
    40-Hz ASSR threshold tests, 282–284
    other, 286–287
    phonemic awareness and discrimination, 287
    sensory *vs.* neural losses, 286
    speech perception abilities, determination of, 286–287
    temporal gap detection, 287
  detection methods, 276
    time- and frequency-domain methods, 276–278
  electrode montage, 275–276
  filtering, 275
  in humans, 269
  in infants, 273, 274–275
  magnitude-squared coherence (MSC) methods, 277
  modulation frequency–subject state, 274
  neural generators, 268–269
  phase coherence measures, 276–277
  signal processing and acquisition variables, 275–278
  spectral measurements, 277–278
  stimulus factors
    AM modulation depth, 269, 271
    carrier frequency, 269, 270t
    chirps, 271
    cochlear place specificity, 273
    FM modulation depth, 269, 271
    mixed modulation (MM), effect of, 269, 271, 271f, 272f
    modulation type, 271
    multiple modulation/carrier frequencies, 272–273
  stimulus–subject interactions, 274
  subject factors, 273–274
  threshold estimation protocol, 287
    acquisition in, 287
    patient factors in, 287–288
    stimulus in, 287
    test method, 288
  threshold rules for tests, 288–289
    examples of, 289
    stopping rules, 290
  thresholds, interpretation of, 288
Auditory system, 632
  age-related changes in, 632–635
Auditory training (AT), 561, 849, 853–855
Auditory verbal communication, 841
Aural rehabilitation process, 696
Autism spectrum disorder, 585–586
  management considerations, 586–587
  testing considerations, 586
Autism spectrum disorder (ASD), 585–586
  management considerations, 586–587
  testing considerations, 586
Autocoils, hearing aids with, 754
Automated audiometry, 44–45
Automated auditory brainstem response (A-ABR), 439
Automated puretone audiometry, 667
Automatic gain control (AGC), 736
Autosomal/autosomes, 871
Autosomal dominant, 871
Autosomal recessive, 871
AVCN. *See* Anteroventral cochlear nucleus (AVCN)
Averaging, and ASSR detection, 276

Background noise, 675. *See also* Room acoustics and auditory rehabilitation technology
BAHAs. *See* Bone-Anchored Hearing Aids (BAHAs)
Balance Master unit, 431
Balance system function, laboratory studies of, 405–422
  active head rotation, 413
  caloric irrigation test, 411–413
    directional preponderance, 412
    fixation index/fixation suppression, 412–413
    unilateral weakness, 411–412
  computerized dynamic posturography, 419–422
    adaptation test, 422
    motor control test, 420–422
    sensory organization test, 419–420
  Dix–Hallpike maneuver in, 409–410, 410f
  dynamic positioning, 409–410
  electronystagmography, 406–413
  horizontal and vertical eye position, 417f
  nystagmography, 406–413
  ocular motility test, 408–409
    gaze stability test, 408
    optokinetic nystagmus test, 409
    saccade test, 409
    smooth pursuit test, 408–409, 408f
  otolith function test, 417–418
  rotational chair test, 413–414, 414f, 415f
  saccular evaluation, 418–419
  sinusoidal harmonic acceleration test, 414–416, 415f
    gain, 415–416
    phase, 416
    symmetry, 416
  static positional test, 410–411
  utricular evaluation, 419
  velocity step test, 416–417
  videonystagmography, 406–413
  visual–vestibular interaction, 417
Baltimore Longitudinal Study, 631
Bamford–Kowal–Bench (BKB), 686
Bamford–Kowal–Bench Speech-in-Noise Test (BKB-SIN), 131
Bamford–Kowal–Bench (BKB) Standard Sentence Lists, 69
Band-pass filter, 195, 195f
Band-reject filter, 195, 195f
Bardet–Biedl syndrome, 591
BAS. *See* Bone Anchored Solutions (BAS)
Base pairs, 871
Batteries
  future concerns in auditory processing test, 556–557
  in hearing aids, 755–756. *See also* Hearing aids
  malfunctioning, 752
  maximizing life of, 756
  zinc-air, 708
Bayley Scales of Infant Development, 468
Beamforming microphone arrays on hearing aids, 764
Beamforming technology, 764
Bedside screening, for dizziness, 402–405
  dynamic visual acuity test, 403
  Fukuda step test, 404
  head impulse test, 403
  headshake test, 404
  hyperventilation test, 405
  ocular motility, 402–403
  Romberg test, 404
Behavioral observation audiometry (BOA), 120, 121t, 465, 829
Behind-the-ear (BTE), 703–704, 703f, 706f
  for children, 762
Bekesy audiometer, 44
Bellis/Ferre model, of central auditory processing, 548–549, 548t. *See also* Central auditory processing (CAP)
  auditory decoding deficit, 549
  integration deficit, 549
  prosodic deficit, 549
  secondary profiles
    auditory associative deficit, 549
    output-organization deficit, 549–550
Bell palsy, 175–176
Benign paroxysmal positional vertigo (BPPV), 426
Biallelic, 871
Bias signal test, 788–789
BICROS. *See* Bilateral contralateral routing of signal (BICROS)
Bilateral contralateral routing of signal (BICROS), 723

Bilateral hearing loss, 39
Bilateral *vs.* monaural amplification, 782
Bimodal-bilingualism approach, 840
Binaural Fusion test, 72
Binaural processing in hearing aids, 723
Biologic check, of audiometer, 14–16, 15f
Biotinidase deficiency, 871
BKB. *See* Bamford–Kowal–Bench (BKB)
Blackberry communicators, usage of, 755
Black box technology, 310
Blackman window, 252
Bluetooth, application of, 694–695
Bluetooth-enabled cell phones, 755
BMQ-R. *See* Buffalo Model Questionnaire—Revised (BMQ-R)
BOA. *See* Behavioral observation audiometry (BOA)
Body-worn hearing aids, 724
Bone-Anchored Hearing Aids (BAHAs), 719, 723–724, 724f, 817–818, 817f
Bone-Anchored Solutions (BAS), 817
Bone-conduction evaluation/testing
  apparatus, 50
  conductive hearing loss with air–bone gaps
    of middle ear origin, 53–54
    of outer ear origin, 53
  cross-checks for, 120
  inner ear bone conduction component, 52
  intracranial hypertension with pseudoSNHL, 56, 57f
  large vestibular aqueducts, 56, 56f
  masking, 82
  middle ear bone conduction component, 51–52
  mixed hearing loss, 55, 55f
  normal-hearing sensitivity, 52–53, 53f
  outer ear bone conduction component, 51
  procedures, 50–52
  sensory/neural hearing loss, 54, 55f
  superior semicircular canal dehiscence, 55–56, 56f
  technical issues
    interaural attenuation, 57
    masking and occlusion effect, 57
    mastoid vs.forehead placement, 57–58, 57t
    threshold accuracy and air–bone gap, 58
    vibrotactile responses, 56–57
  transmission routes, 52, 52f
Bone-conduction hearing, 49
  early writings on, 49–50
  Rinne tuning fork test, 50
  Weber tuning fork test, 50
Bone-conduction hearing aids, 724
Bone-conduction (BC) thresholds, 31, 40–41. *See also* Bone conduction evaluation
Bone vibrators, 34, 34f, 50
  calibration
    artificial mastoid procedure, 19
    real ear procedures, 18–19
Bony labyrinth, 381, 382f
Boot technology, 684
BOR. *See* Branchio-oto-renal syndrome (BOR)
BPPV. *See* Benign paroxysmal positional vertigo (BPPV)
Brain activity, measures of
  brainstem responses, 529
  cortical responses, 529–530
Brain, age-related changes in, 635
Brainstem, auditory, 187–188
Brainstem disorders
  extra-axial, and ASR threshold, 180
  intra-axial, and ASR threshold, 180–181
  retrocochlear and, 177–181
Branchio-oto-renal syndrome (BOR), 491
British Society of Audiology Position Statement, 546
Broadcast media reception difficulty, technology for, 692
Bruit, 41
BTE. *See* Behind-the-ear (BTE)
Buffalo Model Questionnaire—Revised (BMQ-R), 553
Bullying, defined, 504. *See also* Educational audiology
Business considerations for audiologist, 807t
Business vocabulary, for private practitioner, 807t

CADS. *See* Classroom audio distribution systems (CADS)
CAEPs. *See* Cortical auditory-evoked potentials (CAEPs)
Calibration
  acoustic immittance devices, 25–26
  acoustic immittance systems, 146–147
  ancillary equipment
    masking generator, 20
  auditory-evoked potential instruments, 21–25
  auditory steady-state responses, 278
  automatic audiometers, 21
  basic equipment, 14
  bone vibrator
    artificial mastoid procedure, 19
    real ear procedures, 18–19
  compact disc and tape players, 20–21
  earphones, 16, 33
    artificial ear method, 17–18
    calibration worksheet, 18f
    real ear method, 16–17
  of effective masking level, 91–92
  instrumentation, 10
    frequency counter, 12
    multimeter, 12
    oscilloscope, 13
    sound level meter, 12–13
    spectrum analyzer, 13–14
  monitoring meter, 19–20
  otoacoustic emission devices, 25
  parameters of, 10
  puretone audiometers, 14–16
    basic signal, 14
    biologic check, 14–16, 15f
    frequency check, 16
    harmonic distortion check, 16
    linearity check, 16
    rise–fall time, 16
  reasons for, 9–10
  sound field testing, 20
  speech audiometers, 19
  test room standards, 26
  of wideband acoustic immittance, 155
California Consonant Test (CCT), 67, 89
Caloric irrigation test, 411–413
  directional preponderance, 412
  fixation index/fixation suppression, 412–413
  unilateral weakness, 411–412
Canadian Association for Speech-Language Pathologists and Audiologists (CASLPA), 663
Canalith repositioning maneuvers (CRM), 426
Candida, 728
CANS. *See* Central auditory nervous system (CANS)
CAP. *See* Central auditory processing (CAP); Compound action potential (CAP)
CAPD. *See* Central auditory processing disorder (CAPD)
Captioned media, for individual with hearing loss, 692–693
Carhart, Raymond, 4
Carhart's notch, 41
Carrier frequency (CF), and auditory steady-state response, 269, 270t
Case history, 113
  index of suspicion, 113
  interview techniques, 113–116
  medical model for, 113
  questionnaires, 116
    pencil and paper presentations, 116
    verbal presentation, 116
  red flags, 113
  SOAP format, 116
    assessment section, 117
    objective section, 117
    plan, 117
    subjective section, 116–117
  tools, 113
CASLPA. *See* Canadian Association for Speech-Language Pathologists and Audiologists (CASLPA)
CASP. *See* Conditioned Assessment of Speech Production (CASP)
CCT. *See* California Consonant Test (CCT)
CDC-P. *See* Centers for Disease Control and Prevention (CDC-P)
CDI. *See* Communicative Development Inventories (CDI)
CDP. *See* Computerized dynamic posturography (CDP)
CDT. *See* Cubic difference tone (CDT)
CEBA. *See* Central effect of biological aging (CEBA)
Cell phone amplifiers, applications of, 693
Center for Epidemiological Studies-Depression Scale (CES-D), 636
Centers for Disease Control and Prevention (CDC-P), 459
  precautions issued by, 863t
Centers for Medicare and Medicaid Services (CMS), 826
Central auditory hypothesis, for speech recognition difficulty, 637
Central auditory nervous system (CANS), 545
Central auditory pathways
  auditory brainstem, 515–517, 515f
  auditory forebrain, 517–518

Central auditory processing (CAP), 513, 561, 637
auditory processing test batteries, future concerns in, 556–557
background of, 545–546
models
Bellis/Ferre model, 548–550, 548t
Buffalo model, 550–551, 550t
minimal test battery, 547–548
spoken-language–processing model, 551, 552t
screening
questionnaires, 553
tests, 553–555
test battery
approach, 546
and electrophysiological measures, 551–553
tests and central auditory nervous system, 547t
third-party reimbursement, 555–556
Central auditory processing disorder (CAPD), 520, 545
amblyaudia, treatment for, 575
auditory training and language therapy, 575–576
Buffalo model therapies, 561–562
phonemic synthesis, 564–566, 566f
phonemic training program, 562–564, 562f
short-term auditory memory program, 568–570
words-in-noise training, 566–568
classroom accommodations and hearing assistance
amplification, 577–581, 577f
classroom environment, 576
M3 therapies, 571–574
treatment effectiveness, 574–575
test, 555
Central auditory system, acoustic features of speech in, 528t
Central effect of biological aging (CEBA), 637
Central effect of peripheral pathology (CEPP), 637
Central Institute for the Deaf (CID) W-1 and W-2 tests, 63
Central nervous system disorders, 177–181
Central vestibular system, 386–387
cerebellum, 386–387
vestibular nuclei, 386
vestibulocerebellum, 387
Centromere, 871
CEOAEs. *See* Click-evoked otoacoustic emissions (CEOAEs)
CEPP. *See* Central effect of peripheral pathology (CEPP)
Cerebellopontine angle (CPA), 234, 302
Cerebellum, 386–387
Cerebrovascular disease (CVD), 632, 635
Cerumen impaction, 632–633
CES-D. *See* Center for Epidemiological Studies-Depression Scale (CES-D)
CF. *See* Characteristic frequency (CF)
CHABA. *See* Committee on Hearing and Bioacoustics (CHABA)
CHAPPS. *See* Children's Auditory Processing Performance Scale (CHAPPS)
CHAPS. *See* Children's Auditory Performance Scale (CHAPS)
Characteristic frequency (CF), 514
Charcot-Marie-Tooth (CMT) syndrome, 238
CHARGE syndrome, 491–492. *See also* Hearing loss (HL)
Chief complaint (CC), 113
CHILD. *See* Children's Home Inventory for Listening Difficulties (CHILD)
Children
with cochlear implant (CIs)
rehabilitative needs for, 830
speech perception skills, 831–832
congenital severe-to-profound hearing loss in, 478f
cross-check considerations for, 120
directional microphone for, 764
hearing aids fitting for
assessment and verification, 767–772
clinical challenges, 773
pediatric vs.adult hearing aid fitting, 759–762
prescriptive formulae, 765–767
selecting and prescribing, 762–765
suprathreshold perceptual testing, 772–773
with hearing loss
assessing children's progress, 842–846
guidelines and practices, 836–838
special education laws, 835–836
supporting families, 838–842
hearing loss in
age-appropriate assessment of, 461–462
continued surveillance, 462–463
early detection facilitates favorable outcomes, 459–460
etiology of, 460–461, 460t
evidence supports early detection of, 459
multicultural considerations, 462
physical and cognitive/intellectual conditions accompanying, 461t
test battery approach, 462
hyperacusis in, 656
language development, 845–846
with learning disabilities
CADS for, 686–688
nonorganic hearing loss, 618–619
nonorganic hearing loss in, 618–619
personal FM system for, 682–684, 683f
suprathreshold perceptual testing in, 772–773
testing speech thresholds and recognition in, 472–474
tinnitus in, 653
vocal development, 844–845
Children's Auditory Performance Scale (CHAPS), 503, 581
Children's Auditory Processing Performance Scale (CHAPPS), 553
Children's Home Inventory for Listening Difficulties (CHILD), 503
Children's Peer Relationship (CPR), 504
Chirps
auditory brainstem response, 202–204, 254–255
auditory steady-state responses, 271
CHL. *See* Conductive hearing loss (CHL)
Choana, 871
Cholesteatoma, 139t
Chromosome, 871
CID Auditory Test W-22 (CID W-22), 65–66
CID Everyday Sentences, 66
Cincinnati Auditory Skills Checklist, 842
CIPP. *See* Colorado Individual Performance Profile (CIPP)
Circumaural earphones, 33, 78
CIS. *See* Continuous Interleaved Sampling (CIS)
CIs. *See* Cochlear implants (CIs)
City University of New York (CUNY) Sentences, 67
Classroom acoustic standard, status of, 680–681. *See also* Room acoustics and auditory rehabilitation technology
Classroom audio distribution systems (CADS), 505, 682, 685–688, 685f, 686f. *See also* Room acoustics and auditory rehabilitation technology
Clear speech procedures, 691
Click-evoked otoacoustic emissions (CEOAEs)
example, 360f
growth function, 362f
levels, 362
waveform, 360–361
Clicks, 250
Client-Oriented Scale of Improvement (COSI), 640t, 697, 777
Clinical Test of Sensory Integration of Balance (CTSIB), 402, 427
Closed-jaw impressions, 750
CM. *See* Cochlear microphonic (CM)
CMAP. *See* Compound muscle action potential (CMAP)
CM response. *See* Cochlear microphonic (CM) response
CMRR. *See* Common-mode rejection ratio (CMRR)
CMS. *See* Centers for Medicare and Medicaid Services (CMS)
CN. *See* Cochlear nuclei (CN)
CNS disorders, ECochG potentials in, 224–226
Coaching sessions, for hearing aid fitting, 771–772
Cochlear Americas of Centennial, 819, 820f
Cochlear conductive presbycusis, 634
Cochlear fluids, 52
Cochlear hearing loss
ECochG potentials in, 214–218
effects of, on ABR
degree and configuration, 243–245
high-frequency hearing loss, 245–246
Cochlear imaging, 825
Cochlear implants (CIs), 817, 819–823, 821f
candidacy requirements for, 823–828
evaluation, MLAEPs in, 328–329
surgery, auditory system in, 668. *See also* Teleaudiology
telehealth service for, 669. *See also* Teleaudiology
Cochlear microphonic (CM), 464
response, 188, 207
Cochlear nuclei (CN), 187–188
Cochlear Pty. Ltd., 821
Codon, 871
Cognitive function decline and hearing loss, 631, 635–636

Cognitive hypothesis, for speech recognition difficulty, 637–638
Cognitive performance changes and speech understanding difficulty, 638
Coloboma, 871
Colorado Individual Performance Profile (CIPP), 838
Colorado Quality Standards for Programs and Services for Children who are Deaf or Hard of Hearing, 837
Committee on Hearing and Bioacoustics (CHABA), 598
Common cochlear artery, 385
Common-mode rejection ratio (CMRR), 191
Communication in room settings, improvement of. *See also* Room acoustics and auditory rehabilitation technology
  background room noise, 675–677
  distance, 678–680
  noise and reverberation, effects of, 678
  reverberation, 677–678
Communication needs assessment, selected instruments, 640t
Communication strategies training, 697–698
Communicative Development Inventories (CDI), 845
Compact disc and tape players, 20–21
Compensation
  tympanometric, 140
  vestibular, 392–395, 393f
Completely-in-the-canal (CIC), 705
Complex auditory brainstem response (cABR), 338
Compound action potential (CAP), 189, 207
  and auditory nerve function, 307–308
Compound heterozygote, 871
Compound muscle action potential (CMAP), 301, 304–305
Computed tomography (CT), 825
Computer Assisted Speech Perception Software (CASPER), 67
Computer-based speechreading training, 856–857
Computerized dynamic posturography (CDP), 419–422, 420f
  adaptation test, 422
  motor control test, 420–422
  sensory organization test, 419–420
Computer-operated equipment, for telehealth, 661
Concentration, importance of, 652
Condenser microphones, 12
Conditioned Assessment of Speech Production (CASP), 844
Conditioned play audiometry (CPA), 465, 472, 586, 829
Conductive hearing loss (CHL), 49. *See also* Bone conduction evaluation
  amplification for children with, 771
  and ASR threshold, 176–177
  in infant, 374
  prediction of, by WAI tests, 159
Congenial hearing loss, 438
Connected Speech Test (CST), 69
Connexin 26 mutation, hearing loss for, 132
Consanguineous, 871
Consanguinity, 482, 871
Contemporary bone-anchored hearing devices, 817
Contemporary implantable middle-ear devices, 818–819
Continuous artifact, 298
Continuous Interleaved Sampling (CIS), 821
Continuously attended model, IOM, 310
Contralateral hearing aid, use of, 831
Contralateral masking
  during bone-conduction audiometry, 84–86, 85f
  during puretone air-conduction audiometry, 82–84, 83f
  purpose of, 90
Contralateral routing of signal (CROS), 723
Conversation Made Easy program, 857
Cortical auditory-evoked potentials (CAEPs), 337
  aging effects, 349
  amplitudes of, 337
  and auditory training, 352–353
  clinical applications, 349–353
  and cochlear implants, 351–352
  EEG *vs.* MEG, 339
  electrode configurations and acquisition parameters, 339–340
  exogenous and endogenous aspects, 338–339
  hearing loss and hearing aids, 349–351
  latency of, 337–338
  maturational effects, 349
  passive recordings, 338
  patient subject factors, 349
  preattentive, 338–339
  types of, 340
    mismatch negativity, 346–347, 346f
    N400/P600, 348–349
    P300, 347–348, 347f
    P1-N1-P2 change complex, 344–346, 345f
    P1-N1-P2 complex, 340–343
  use of, 337
COSI. *See* Client-Oriented Scale of Improvement (COSI)
Counseling. *See also* Rehabilitation
  in audiologic rehabilitation, 851–853
  Audiologists role in, 851–853
  for children with hearing loss, 839–840
  educational audiology and, 507–508
  hearing aid, 802–803
  recommendations and, 889–890
CPA. *See* Cerebellopontine angle (CPA); Conditioned play audiometry (CPA)
CPR. *See* Children's Peer Relationship (CPR)
CPT. *See* Current Procedural Terminology (CPT)
Cranial motor nerves. *See also* Intraoperative neurophysiological monitoring (IOM)
  monitoring of, principles of, 304–306
  triggered electrical stimulation of, 303–304
Cranial nerve stimulation, nonacoustic occlusion, 748–749
Cristae ampullaris, 383f, 384–385
Critical band concept, 90–91
CRM. *See* Canalith repositioning maneuvers (CRM)
CROS. *See* Contralateral routing of signal (CROS)
Cross hearing, 77. *See also* Interaural attenuation (IA)
"Cross-talk," 15
CT. *See* Computed tomography (CT)
CTSIB. *See* Clinical Test of Sensory Integration of Balance (CTSIB)
Cubic difference tone (CDT), 363
Cued speech, for D/HH children, 840
CUNY Nonsense Syllable Test (CUNY-NST), 67
Cupula, 384–385
Current Procedural Terminology (CPT), 555, 649
Custom earmolds, 709–710, 709f. *See also* Hearing aid
CVD. *See* Cerebrovascular disease (CVD)
Cytogenetics, 871
Cytosine, 871

DAC. *See* Digital-to-analog converter (DAC)
DAI. *See* Direct audio input (DAI)
Dau chirp, 202
DCN. *See* Dorsal cochlear nucleus (DCN)
DDT. *See* Dichotic Digit Test (DDT)
Deaf-blindness, defined, 876
The Deaf Child's Bill of Rights, 837–838
Deafness, defined, 876
Deafness modifier genes (DFM), 489
Deaf or hard of hearing (DHH), 437, 836
Decibels (dB), 29
Deletion, 871
Dementia and hearing loss, 631
Demyelinating and cortical disorders, 181
De novo mutation, 871
Deoxyribonucleic acid (DNA), 872
Depression and hearing loss, 636
Desired Sensation Level (DSL) formula, 740, 765
Development, effect of, on tympanometry measures, 144
DFM. *See* Deafness modifier genes (DFM)
DHH. *See* Deaf or hard of hearing (DHH)
DHI. *See* Dizziness Handicap Inventory (DHI)
Diabetes and hearing loss, 631
Diagnostic audiology, 119, 119t
  case studies on, 121–130
  cross-checks, 121t
    electrophysiological tests as, 120
    pediatric testing, 120
    principle, 119
    for puretone air conduction, 119
    for puretone audiometry, 120
    for puretone average, 120
  limitations, 130
  order of tests, 121
  and referrals, 130–131
    for auditory processing evaluation, 131–132
    for genetic evaluation, 132
    for medical otolaryngologic evaluation, 131, 131t
    for vestibular evaluation, 132
  speech-in-noise test, 120
Dichotic Digits, 72
Dichotic Digit Test (DDT), 555
Dichotic Interaural Intensity Difference (DIID), 575
Dichotic Sentence Identification test, 72

Differential amplification, 191
Differential Screening Test for Processing (DSTP), 554
Diffusion tensor imaging (DTI), 329
Digenic inheritance, 483, 872
Digital hearing aids, 707
Digital noise reduction, 717–718
Digital oscilloscope, 13
Digital signal processing, 782
Digital sound processor, 707
Digital subscriber line (DSL), 661
Digital-to-analog converter (DAC), 194
Digital transmission technology, advances in, 690. *See also* Room acoustics and auditory rehabilitation technology
Digital wireless technology, 722–723
DIID. *See* Dichotic Interaural Intensity Difference (DIID)
Direct audio input (DAI), 709, 755
Direct electrical connect systems, 696
Directional hearing aids
  directional microphone
    for children, 764
    technology, 681
  testing of, 743–744
Directional preponderance (DP), 412
Distortion product otoacoustic emissions (DPOAEs), 25, 238, 463
  characteristics, 363–364
  clinical applications, 367–368
  cumulative distributions, 368f
  frequency separation, 363
  graph, 363f
  hearing loss diagnosis, 371–372
  hearing status prediction, 372
  high-frequency stimuli, 372
  input/output function, 372
  level as function of frequency, 371f
  levels, 363–364, 367, 367f, 368–369, 369f, 371
  measurement, 362–363, 375f, 465
  patient characteristics, 364
  "primaries," 362
  ROC curves, 367–368
  spectrum, 362f
  TEOAEs *vs.*, 364
Dix–Hallpike maneuver, 409–410, 410f
Dizziness and balance disorders
  bedside screening, 402–405
    dynamic visual acuity test, 403
    Fukuda step test, 404
    head impulse test, 403
    headshake test, 404
    hyperventilation test, 405
    ocular motility, 402–403
    Romberg test, 404
  clinical presentation, 399–405
  dysequilibrium and, 400
  gait instability and, 400–401
  light-headedness and, 400
  neurotologic case history, 401, 401t
  overview, 399
  pre-syncope and, 400
  screening and diagnostic vestibular tests, 400t
  self-assessment inventories, 402
  signs and symptoms, 400–401
  vertigo and, 400
  vestibular *vs.* nonvestibular, 402t
Dizziness Handicap Inventory (DHI), 402
DNLL. *See* Dorsal nucleus of the lateral lemniscus (DNLL)
Doerfler–Stewart test, 626. *See also* Nonorganic hearing loss
Dorsal cochlear nucleus (DCN), 515
  aging changes in, 634
Dorsal nucleus of the lateral lemniscus (DNLL), 516
Down syndrome, 484
  causes of, 484
  characteristics of, 484
DP. *See* Directional preponderance (DP)
DPOAEs. *See* Distortion product otoacoustic emissions (DPOAEs)
DSL. *See* Desired Sensation Level (DSL) formula; Digital subscriber line (DSL)
DSL I/O Version 5.0, for hearing aid verification, 797–798
DSP. *See* Digital signal processing (DSP); Digital sound processor (DSP)
DSTP. *See* Differential Screening Test for Processing (DSTP)
Duplication, 872
Dynamic positioning, 409–410
Dynamic visual acuity (DVA) test, 403
  for vestibular function, 427
Dysequilibrium, 400
Dyslexia, 287
Dysmorphic, 872
Dystopia canthorum, 872

EABR. *See* Electric auditory brainstem response (EABR)
Ear disorders
  effects of, on wideband acoustic immittance, 157–159, 158f
  signs of, 131, 131t
Ear impression techniques, 763
Early hearing detection and intervention (EHDI), 437
  national goals for, 444–449, 445t, 446f
  protocols used in, 445t
  in the United States, 441–443
Early Listening Function (ELF), 503, 843
Early Speech Perception (ESP), 826
Earmold technology, advance in, 783–784
Earphones, 32–33
  calibration
    artificial ear method, 17–18
    calibration worksheet, 18f
    real ear method, 16–17
  circumaural, 33
  insert, 32–33, 32f
  supra-aural, 32, 32f
E-ARTONE 5A, 80
EARtrak®, 778, 779f
Ear wax, 752–754
EAS. *See* Electric–acoustic stimulation (EAS) devices
ECMO. *See* Extracorporeal membrane oxygenation (ECMO)
ECochG. *See* Electrocochleography (ECochG)
EDA. *See* Electrodermal audiometry (EDA)
Edgerton–Danhauer NST, 67
Educational audiology
  amplification, 506
  assessment, 502
    auditory processing assessment, 505
    bullying and hearing loss, 504
    classroom acoustics, 505
    classroom communication, instruction and administrative support, 505
    classroom listening assessment, 503–504
    communication access assessment, 502–503
    eligibility for services, 505–506
    hearing assessment, 502
    hearing loss adjustment and self-advocacy, 504
  counseling, 507–508
  ethics and conduct in education settings, 511
  habilitation, 506–507, 507t
  identification, 501–502
  individual education program team, role, 510
  models of service provision, caseloads, and licensure, 509–510, 510f
  prevention, 508–509
  program development and evaluation, 510–511
  services according to IDEA, 501
Educational services for school-aged D/HH children, 837
EEG. *See* Electroencephalogram (EEG)
EHDI. *See* Early hearing detection and intervention (EHDI)
eHealth, 659
Eight-pole filter, 195
Electric–acoustic stimulation (EAS) devices, 823
Electrical artifacts, 298
Electrically evoked stapedial reflexes (ESRT), 829
Electric auditory brainstem response (EABR), 825
Electricity, for tinnitus treatment, 653
Electroacoustic procedures, for DSP Features Assessment, 786–789
Electrocochleography (ECochG), 207, 307
  normal response, 212–214, 213f, 214f
  potentials, 207
    in auditory neuropathy, 219–224
    clicks, stimulation with, 210
    in CNS disorders, 224–226
    in cochlear hearing loss, 214–218
    cochlear microphonic, 207–208
    compound action potential, 209
    extratympanic techniques, 210
    in infants discharged from NICU, 226–228
    intratympanic procedures, 209–210
    in Ménière disease, 218–219
    recording of, 211, 212f
    recording site, 209–210
    signal processing and CAP extraction, 211–212, 211f
    stimuli for, 210–211
    summating potential, 208–209
    tone-burst stimulation, 210–211
    transtympanic approach, 209
  responses, 188–189
Electrodermal audiometry (EDA), 626
Electrodes
  in auditory steady-state responses, 275–276

in ECochG recording, 209–210
middle-latency auditory-evoked potentials, 320
Electroencephalogram (EEG), 464
Electromagnetic induction loop systems, 688–690, 689f. *See also* Room acoustics and auditory rehabilitation technology
Electronic medical record (EMR), 778
Electronystagmography, 406–413
Electro-oculograms (EOG)
with horizontal (HEOG) electrodes, 340
with vertical (VEOG) electrodes, 340
Electrophysiological test
as cross-checks, 120
of hearing loss, 825. *See also* Cochlear implants (CIs)
Electrostatic discharge (ESD), 820
ELF. *See* Early Listening Function (ELF)
EM. *See* Environmental microphone (EM)
e-mail technology, for telehealth, 661
EMR. *See* Electronic medical record (EMR)
Encephalomyopathy, 872
Endolymph, 381, 384–385
Endolymphatic potential (EP), 634
Energetic masking, 70
Enlargement of vestibular aqueduct (EVA), 493
Environmental characteristics, in audiology, 662–663
Environmental classification, 721
Environmental microphone (EM), 682
Environmental Protection Agency (EPA), 596
EOAEs. *See* Evoked otoacoustic emissions (EOAEs)
EoWPVT-3. *See* Expressive One Word Picture Vocabulary Test (EoWPVT-3)
EP. *See* Endolymphatic potential (EP)
EPA. *See* Environmental Protection Agency (EPA)
EPSPs. *See* Excitatory postsynaptic potentials (EPSPs)
ERPs. *See* Event-related potentials (ERPs)
Escherichia coli, 753
ESD. *See* Electrostatic discharge (ESD)
ESP. *See* Early Speech Perception (ESP)
ESRT. *See* Electrically evoked stapedial reflexes (ESRT)
Esteem® Hearing Implant, 818, 819f
Estimated hearing level (eHL), 260
ET. *See* Eustachian tube (ET)
Etymotic model ER-3A insert earphones, 32, 32f, 33
Euploidy, 484, 872
Eustachian tube (ET), 143, 144
dysfunction, 138, 143
dysfunction testing, 811
function tests, 144–145
patulous, 145
EVA. *See* Enlargement of the vestibular aqueduct (EVA)
Event-related potentials (ERPs), 189, 337. *See also* Cortical auditory-evoked potentials (CAEPs)
Evoked otoacoustic emissions (EOAEs), 359
clinical, 371
criteria, 369
differential diagnosis, 370
input/output function, 372
levels, 364, 365
measurement, 364
test result, 364, 365
Excitatory postsynaptic potentials (EPSPs), 220
Exome, 872
Exon, 872
Expansion, 872
Expression, 872
Expressive One Word Picture Vocabulary Test (EoWPVT-3), 846
Extended wear hearing aid, 724, 724f
Extra-axial brainstem disorders, 180
Extracorporeal membrane oxygenation (ECMO), 497
Extraocular muscle (EOM), 388
semicircular canals and, 388t
Extratympanic techniques, for ECochG recording, 210, 210f
Eyeglass hearing aids, 724
Eye movements
rapid, 389
smooth pursuit, 389
Eye position
electrode montage for monitoring, 406f
horizontal, 417f
vertical, 417f

Facial nerve, anatomy of, 302–303, 302f
Facial nerve paralysis, 175–176, 176f
and ASR threshold, 175–176, 176f
Familiarization method, for threshold measurement, 36
FAPE. *See* Free and appropriate public education (FAPE)
FAPI. *See* Functional Auditory Performance Indicators (FAPI)
Fast Fourier transform (FFT), 13, 276
FCC. *See* Federal Communications Commission (FCC)
FDA. *See* Food and Drug Administration (FDA)
Fear hyperacusis, 654
Federal Coal Mine Health and Safety Act, 600, 886
Federal Communications Commission (FCC), 682
Feedback suppression systems, 745–746
FFR. *See* Frequency-following response (FFR)
File transfer protocol (FTP), 661
Filtering, for reducing unwanted noise, 195, 195f
Filter skirt, 195
Financial decisions, of audiologists, 807–809. *See also* Audiologists
Fixation index, 412–413
Fixation suppression, 412–413
Fixed loss pad, 16
FLE. *See* Functional Listening Evaluation (FLE)
Floating mass transducer (FMT), 819
FM. *See* Frequency modulation (FM)
FMT. *See* Floating mass transducer (FMT)
Foam pads, hearing aids with, 754
Food and Drug Administration (FDA), 690, 705
approved candidacy criteria for contemporary CI systems, 824t, 826
Fourier transform, 276
Frameshift mutation, 872
Free and appropriate public education (FAPE), 505
Frequency counter, 12
Frequency-following response (FFR), 325, 338, 530
Frequency modulation (FM), 681
and tones, 34
use of, 751
Frequency-specific auditory brainstem response (FS-ABR), 759, 760f
Frye Electronics" hearing aid and real-ear analyzers, 786, 787
FS-ABR. *See* Frequency-specific auditory brainstem response (FS-ABR)
F-test, 277–278
FTP. *See* File transfer protocol (FTP)
Fukuda step test, 404
Functional Auditory Performance Indicators (FAPI), 503, 842–843
Functional hearing loss, and ASR threshold, 183
Functional Listening Evaluation (FLE), 503, 581, 844
Functional skills screening, for children with hearing loss, 877–881

GABAergic inhibition, age-related downregulation of, 634, 635
GABA neurotransmission, age-related changes in, 635
Gait instability, 400–401
Gallaudet Research Institute (GRI), 583
Gans repositioning maneuver (GRM), 432
Gap junction beta-2 *(GJB2)* mutation, testing for, 132
GAR. *See* Group aural/audiologic rehabilitation (GAR)
Gaze stability test, 408
GDS. *See* Geriatric Depression Scale (GDS)
Gender, influence on hearing status, 636–637
Gene, 872
General Education Inclusion Readiness Checklist, 838
Gene therapy, 872
Genetic evaluation, referral for, 132
Genetic markers, 872
Genetic modifiers. *See* Modifier genes
Genome, 872
Genotype, 872
Geriatric Depression Scale (GDS), 636
Geriatric syndromes and hearing loss in elderly, 635–636
GFTA-2. *See* Goldman Fristoe Test of Articulation-2 (GFTA-2)
Glove use guidelines for audiologists, 863–864, 863f, 863t
Glutamate receptors, age-related changes in, 635
Goldenhar syndrome, 591
Goldman Fristoe Test of Articulation-2 (GFTA-2), 845
Grason Stadler Model 1720 otoadmittance meter, 139
GRI. *See* Gallaudet Research Institute (GRI)
GRM. *See* Gans repositioning maneuver (GRM)
GROUP. *See* Group Rehabilitation on-Line Utility Pack (GROUP)
Group aural/audiologic rehabilitation (GAR), 857

Group Rehabilitation on-Line Utility Pack (GROUP), 858
Guanine, 872
Guidelines for Audiometric Symbols, 37
Guidelines for Manual Pure Tone Audiometry, 35

Habituation, 430f
HAC. *See* Hearing aid compatible (HAC)
HAC Act. *See* Hearing Aid Compatibility Act of 1988 (HAC Act)
HAE. *See* Hearing aid evaluation (HAE)
Haemophilus influenzae, 753
HAF. *See* Hearing aid fitting (HAF)
HA-1 hearing aid coupler, 731–732, 731f
HA-2 hearing aid coupler, 731–732, 731f, 732f
Hair cells
  activation, 383f, 386f
  cuticular plate, 383
  excitatory responses, 383f
  kinocilium, 383
  stereocilia, 383
  structure, 383–384
  tip links, 383
  type I, 383, 384f
  type II, 383, 384f
Hardwired systems, 690. *See also* Room acoustics and auditory rehabilitation technology
HAT. *See* Hearing assistive technology (HAT)
Head injury, ABR in, 243, 243f
Head-related transfer function (HRTF), 522
Headshake nystagmus (HSN), 404
Health Maintenance Organization Act of 1973, 814
Health-related quality-of-life (HRQOL), 675
Hearing aid analyzers
  digital features verification, 742–746
  schematic representation of, 730f
  troubleshooting with, 730–732
Hearing aid candidacy, assessment of, 760–762
Hearing Aid Compatibility Act of 1988 (HAC Act), 694
Hearing aid compatible (HAC), 709
Hearing aid counseling, 802–803
Hearing aid evaluation (HAE), 779–789, 825
Hearing aid fitting (HAF), 780, 789–802
Hearing aids, 876
  electroacoustic performance, 711–712
  fitting for adults, 777–778
    hearing aid evaluation, 779–789
    hearing aid fitting, 789–802
    outcome measures, 802
    prefitting data, 778–779
  fitting for children
    assessment and verification, 767–772
    clinical challenges, 773
    pediatric vs.adult hearing aid fitting, 759–762
    prescriptive formulae, 765–767
    selecting and prescribing, 762–765
    suprathreshold perceptual testing, 772–773
  other styles of, 723–724
  prevention and troubleshooting tips for
    American National Standards Institute hearing aid tests, 732–739
    effective troubleshooting of problems, 727–732
    problem patterns, 727
  signal processing
    audibility, 712–716
    comfort improvement, 717–720
    improving signal-to-noise ratio, 716–717
    personalization, 720–721
    tinnitus management, 721–722
  styles of, 703–705
  technology of, 706–711
  troubleshooting tips for
    batteries, 755–756
    digital features verification, hearing aid analyzers in, 742–746
    ear wax, 752–754
    feedback, 749–752
    nonadaptive hearing aid characteristics assessment, 739–742
    occlusion and ampclusion, 748–749
    prevention of, 756
    proper fit, 746–748
    telephones, 754–755
  use of, 651
Hearing assistive technology (HAT), 503, 561, 576, 639, 675, 780
  legislation for, 699
Hearing Handicap Inventory (HHI), 640, 642
Hearing Handicap Inventory for the Elderly—Screening Version (HHIE-S), 116
Hearing handicap, self-reported, 636
Hearing Health Care Intervention Readiness Questionnaire (HHCIR), 640, 641t
Hearing impairment
  adverse effects of untreated, 638–639
  defined, 872
Hearing Industries Association (HIA), 703
Hearing-in-Noise-Test (HINT), 69, 131, 668, 715, 777
Hearing level (HL), 29
Hearing loss, 596
  age-related, 631
    aging auditory mechanism, 632–635
    audiologic findings, 636–638
    diagnostic and management protocol, 639–642
    future perspectives, 643
    geriatric syndromes associated with, 635–636
    hearing screening, 642–643
    and medical comorbidities, 631–632
    psychosocial consequences of, 638–639
    risk factors for, 632
  assessment
    age-appropriate assessment, 461–462
    continued surveillance, 462–463
    early detection and favorable outcomes, 459–460
    etiology of, 460–461, 460t
    multicultural considerations, 462
    physical and cognitive/intellectual conditions accompanying, 461t
    test battery approach, 462
  auditory processing deficit individuals, technologies for, 691–694
  in children
    age-appropriate assessment of, 461–462
    audiology, service delivery in, 461–463
    continued surveillance, 462–463
    early detection facilitates favorable outcomes, 459–460
    etiology of, 460–461, 460t
    evidence supports early detection of, 459
    multicultural considerations, 462
    physical and cognitive/intellectual conditions accompanying, 461t
    test battery approach, 462
  cochlear, 173–175, 174f, 175f
    and ASR threshold, 173–175, 174f, 175f
    ECochG potentials in, 214–218
    effects of, on ABR, 243–246
  conductive, 49, 176–177, 374. *See also* Conductive hearing loss (CHL)
  cortical auditory-evoked potentials and, 349–351
  differential diagnosis of, 370
  early identification of, 249. *See also* Auditory brainstem response (ABR); Auditory-evoked potentials (AEPs)
  educational audiology assessment for children with, 503t
  effects of, on MLAEPs, 327
  in elderly. *See also* Room acoustics and auditory rehabilitation technology
    acoustic modifications of room for, 680
    aging auditory mechanism, 632–635
    audiologic findings, 636–638
    diagnostic and management protocol, 639–642
    future perspectives of, 643
    geriatric syndromes associated with, 635–636
    hearing screening, 642–643
    and medical comorbidities, 631–632
    psychosocial consequences of, 638–639
    and tinnitus, 651, 652
  functional, 183
  identification, OAEs in, 368–371
  middle-latency auditory-evoked potentials, effect on, 327
  noise-induced, 43. *See also* Noise-induced hearing loss (NIHL)
  nonsyndromic
    auditory neuropathy, autosomal dominant, 489
    autosomal dominant, 486–487
    autosomal recessive, 487–488
    deafness modifier genes, 489
    mitochondrial hearing loss, 489
    otosclerosis, 489
    X-linked, 488–489
    Y-linked, 489
  phenotype, 485–486
  relative handicap of, 893
  sensory/neural, 49, 373–374, 675
  syndromic, 489–490
    Alport syndrome, 490–491
    branchio-oto-renal syndrome, 491
    CHARGE syndrome, 491–492
    Jervell and Lange-Nielsen syndrome, 492
    mitochondrial encephalomyopathy, 492–493
    pendred syndrome, 493
    Stickler syndrome, 493–494

Treacher Collins syndrome, 494
Turner syndrome, 494
Usher syndrome, 494–495
Waardenburg syndrome, 495–496
unilateral, 39. *See also* Unilateral hearing loss (UHL)
Hearing protection devices (HPD), 600, 606
acoustics of, 607–608, 608f
computation of attenuated exposure, 606–607
earmuffs and earplugs, 608–609, 608f
electronic, 610
fittings, 609
history of, 606
maximum attenuations, 609
reportable shift, 614
standard threshold shift, 613–614, 613t
uniform frequency attenuators, 609–610
unresolved issues with, 612–613, 612t
verification of, 610–612, 611f
Hearing screening programs, 642–643
Heavy metal exposure, ABR in, 242–243
Helmholtz/volume-associated phenomena, 608. *See also* Hearing protection devices (HPD)
Hemizygous, 872
Hepatitis B (HBV) vaccination plan, 866
Herbal supplements, for tinnitus treatment, 653
Hereditary hearing loss
diagnostic evaluation of, 497, 497t
epidemiology of, 477
future prospects of, 498
genes involved in, 484–485, 485t
online resources for, 490t, 498
Heterogeneity, 872
Heteroplasmy, 872
Heterozygous, 872
HFA. *See* High-frequency average (HFA)
HHCIR. *See* Hearing Health Care Intervention Readiness Questionnaire (HHCIR)
HHI. *See* Hearing Handicap Inventory (HHI)
HIA. *See* Hearing Industries Association (HIA)
High-frequency average (HFA), 736
High-level short-duration tone bursts, 742
High-pass filter, 195, 195f
High predictability (HP) words, 69
HINT. *See* Hearing-in-Noise-Test (HINT)
HiRes Harmony processor, 821
HiResolution Bionic Ear System, 821
Hirschsprung disease, 496
Home computers, for people with hearing loss, 695
Homologous (chromosomes), 872
Homozygous, 872
House–Brackmann Facial Nerve Grading Scale, 306
HPD. *See* Hearing protection devices (HPD)
HRQOL. *See* Health-related quality-of-life (HRQOL)
HRTF. *See* Head-related transfer function (HRTF)
HSN. *See* Headshake nystagmus (HSN)
Hughson–Westlake down-up procedure, modified, 36
Human brain
acoustic onsets in
auditory brainstem, 537–538
auditory cortex, 538–539
brainstem–cortex relationships, 539
electrophysiological changes due to training, 539
formant structure in, 533–535
frequency transitions in, 535–537
Human genetics, 477–478
chromosomal abnormalities, 484
chromosome, 478–480, 479f
evaluation and diagnosis
benefits and limitations of genetics testing, 496
genetics professionals, 497–498
genetic testing, 498
genes, 478
genetic mutations, 480, 480t
genotype–phenotype, 480
Mendelian inheritance, 480–481
autosomal dominant inheritance, 481, 481f
autosomal recessive inheritance, 481–482, 482f
sex-linked inheritance, 482
multifactorial inheritance, 483–484
nomenclature, 484
non-Mendelian inheritance, 482
mitochondrial inheritance, 482–483
modifier genes, 483
polygenic inheritance, 483
Human Genome Organization (HUGO) Gene Nomenclature Committee, 484
Hybrid cochlear implant devices, 823
Hyperacusis
annoyance, 654
in childhood, 656
evaluation of, 654
fear, 654
neurophysiological causes, mechanisms, and models of, 653–654
pain, 654
sound therapy for, 655–656
treatments for, 654–656
Hyperbilirubinemia, ABR in, 242
Hyperventilation-induced nystagmus, 405

IA. *See* Interaural attenuation (IA)
ICF. *See* International Classification of Functioning, Disability, and Health (ICF)
ICRA. *See* International Collegium of Rehabilitative Audiology (ICRA)
ICTs. *See* Information and communication technologies (ICTs)
IDEA. *See* Individuals with Disabilities Education Act (IDEA)
IEC. *See* International Electrotechnical Commission (IEC)
IEP. *See* Individualized Education Plan (IEP); Individual Education Program (IEP)
IEP team, 872
IFSP. *See* Individualized Family Service Plans (IFSP)
IHCs. *See* Inner hair cells (IHCs)
IIC. *See* Invisible-in-the-canal (IIC)
ILD. *See* Interaural level difference (ILD)
Immittance equipment, for telehealth service, 667. *See also* Teleaudiology
IMP. *See* Infant Monitor of Vocal Production (IMP)
Impedance audiometry, usage of, 811
Implantable hearing device
bone-anchored hearing devices, 817–818
candidacy for cochlear implant, 823–828
cochlear implants, 819–823
contemporary implantable middle-ear devices, 818–819
external components, 876
future perspectives, 833
hybrid cochlear implant devices, 823
postoperative management, 829–830
rehabilitation, 830–831
speech perception and speech and language results, 831–832
surgery
complications and device failures, 833
procedure, 828–829
Implant ear, determination of, 827–828. *See also* Cochlear implants (CIs)
Independently amplitude-and frequency-modulated (IAFM) stimulus, 286
Individual Education Program (IEP), 501, 504, 836
Individualized Family Service Plans (IFSP), 453–454
Individuals with Disabilities Education Act (IDEA), 447, 835–836
educational audiology services according to, 501. *See also* Educational audiology
Individuals with Disabilities Education Act of 1997 (IDEA 1997), 699
Individuals with Disabilities Education Improvement Act of 2004 (IDEA 2004), 875–876
Induction-based systems, 696
Infant
discharged from NICU, ECochG potentials in, 226–228
hearing loss
conductive, 374
sensory/neural, 373–374
tympanometry in, 148–149, 148t, 149f
Infant Monitor of Vocal Production (IMP), 844
Infection Control for the Audiology Clinic, 867
Infection control, in audiology, 861
implementation of infection control principles, 862–865
relevance of, 861–862, 861t
terms, definition of, 865t
written infection control plan, 865–867
Infection waste, disposal of, 865. *See also* Infection control, in audiology
Inflation–deflation test, 145
Informational masking, 70
Information and communication technologies (ICTs), 659
Infrared assistive device, 692f
Infrared light wave systems, 684–685. *See also* Room acoustics and auditory rehabilitation technology
Inner ear, 382
Inner hair cells (IHCs), 513
selective loss, 357
Insert earphones, 32–33, 32f, 78
in bilateral conductive hearing loss, 99
Insertion, 872

Instructional Communication Access Checklist, 838
Integrated services digital Network (ISDN), 661
Intellectual disability, 588–589
  management considerations, 590
  testing considerations, 589–590
Interaural attenuation (IA), 77–78
  air-conduction measurements, 78–82
  for bone-conducted sound, 82
  calculation of, 78f
  earphone testing and, 78–80
  for speech, 81–82
Interaural level difference (ILD), 516
Interaural phase differences (IPDs), 344–345, 516
Intermediate presbycusis, characteristic of, 634
Internal auditory canal (IAC), 302
International Building Code, 681
International Classification of Functioning, Disability, and Health (ICF), 546
International Classification of Impairment, Disability and Handicap, 849
International Collegium of Rehabilitative Audiology (ICRA), 740, 787
International Electrotechnical Commission (IEC), 9, 689
  standards for audiometers and audiometric testing, 11t
International Organization for Standardization (ISO), 9
  standards for audiometers and audiometric testing, 11t
International Outcomes Inventory for Hearing Aids (IOI-HA), 640t, 669
International Society of Audiology, 3
International Speech Test Signal (ISTS), 735–736
International symbol of access for individuals with hearing loss, 699f
Internet-based rehabilitation methods, 669–670. *See also* Teleaudiology
Interpreting services, 875
Interviews, for case history, 113–114
Interwave intervals (IWIs), 197
In-the-canal (ITC), 705
In-the-ear (ITE), 703, 703f, 704–706, 705f
Intra-axial brainstem disorders, 180–181
Intraoperative neurophysiological monitoring (IOM), 295
  anatomy and physiology related to, 300
    compound muscle action potential, 301
    facial nerve, 302–303, 302f
    motor unit, 300–301, 301f
    motor unit action potential, 300–301
  anesthesia and, 298–300
  of auditory function, 306–309
  cranial motor nerves
    monitoring of, principles of, 304–306
    triggered electrical stimulation of, 303–304
  electromyography and, 300–302
  evolution of, 295
  need of, 296–297
  overview, 295–296
  patient preparation and, 298, 299t
  pharmacologic and physiologic effects on, 300t
  planning scheme for, in selected surgical procedures, 297t
  program, development and management of, 309–312, 311t
  purpose of, 295
  recording sites for, 299t
  standardizing of, 297–298, 297t
  training for, 296
Intratympanic procedures, for ECochG recording, 209–210, 210f
Intron, 872
Inversion, 872
Invisible-in-the-canal (IIC), 705, 705f
IOI-HA. *See* International Outcomes Inventory for Hearing Aids (IOI-HA)
IOM. *See* Intraoperative neurophysiological monitoring (IOM)
Iowa Tinnitus Activities Questionnaire, 891
IPD. *See* Interaural phase difference (IPD)
Iris heterochromia, 873
ISDN. *See* Integrated services digital Network (ISDN)
iSense micro personal frequency modulation system, 684f
ISTS. *See* International Speech Test Signal (ISTS)
ITC. *See* In-the-canal (ITC)
ITE. *See* In-the-ear (ITE)

JCIH. *See* Joint Committee on Infant Hearing (JCIH)
Jervell and Lange-Nielsen syndrome (JLNS), 492
JLNS. *See* Jervell and Lange-Nielsen syndrome (JLNS)
Joint Committee on Infant Hearing (JCIH), 370, 440, 442, 459
  position statement, 120, 836–837

Karyotype, 478, 873
Kinocilium, 383
Klein–Waardenburg syndrome, 495
Koss model HV/1A earphone, 33

Labyrinthine artery, 385
LACE. *See* Listening and Communication Enhancement (LACE)
Language development, in children, 845–846
Laplacian method, 320
Large vestibular aqueducts (LVA), 56, 56f
Lateral ossicular fixation, 139t
Lateral superior olive (LSO), 516
Lateral vestibulospinal tract (LVST), 386
  vestibulospinal reflex and, 392
LDLs. *See* Loudness discomfort levels (LDLs)
LEA. *See* Local education agency (LEA)
Lexical Neighborhood Test (LNT), 474, 826
LIFE. *See* Listening Inventories for Education (LIFE)
LIFE-R. *See* Listening Inventory for Education Revised (LIFE-R)
Light-headedness, 400
Limited liability company (LLC), 808
Limited liability partnership (LLP), 808
Lipreading, 855
LiSN & Learn auditory training software, 576. *See also* Central auditory processing disorder (CAPD)
Listening and Communication Enhancement (LACE), 532, 576, 854, 889–890
Listening Inventories for Education (LIFE), 581
Listening Inventory for Education Revised (LIFE-R), 503
LittlEars Parent Interview, 842
LLC. *See* Limited liability company (LLC)
LLP. *See* Limited liability partnership (LLP)
LNT. *See* Lexical Neighborhood Test (LNT)
Local education agency (LEA), 509
Locus, 478, 873
Long-latency AEP (LLAEP), 328
Long-latency responses (LLRs), 337. *See also* Cortical auditory-evoked potentials (CAEPs)
Long-term average speech spectrum (LTASS), 735, 761
Loudness
  of everyday sounds, 892
  judgments for speech, 802
Loudness discomfort levels (LDLs), 654, 770
Low-pass filter, 194, 195, 195f
Low predictability (LP) words, 69
LSO. *See* Lateral superior olive (LSO)
LTASS. *See* Long-term average speech spectrum (LTASS)
Lyric2, 819, 819f

Magnetic resonance imaging (MRI), 825
Magnetoencephalography (MEG), 339, 530
  role of, 520
Magnitude-squared coherence (MSC), 277
Main cochlear artery, 385
Mainstream Amplification Resource Room Study (MARRS), 686
MAIS. *See* Meaningful Auditory Integration Scale (MAIS)
Marion Downs Hearing Center (MDHCF), 442
MARRS. *See* Mainstream Amplification Resource Room Study (MARRS)
Masking, 77
  acoustic procedures, 92, 103–109
  air-conduction testing, 77–82
  ANSI/ASA definition, 90
  audiogram interpretation, 100–101
  bone-conduction testing, 82
  central, 99–100
  concepts, 90–92
  contralateral, 82
  critical band concept, 90–91
  dilemma, 99
  effective, 91–92
  efficiency, 90
  Hood procedure, 95–99
  ipsilateral, 90
  maximum level, 94–95
  midplateau procedure, 104–109
  minimum level, 92–94
  narrowband noise, 91
  need for, 77–82
  noise selection, 90–91
  optimized masking method, 95–99
  paradigms, 90
  procedures, 92–109
  psychoacoustic procedures, 92, 101–103, 102f
  in puretone air-conduction audiometry, 82–84
  in puretone audiometry, 92–101
  in puretone bone-conduction audiometry, 84–86

recommended procedure, 95–99
in speech audiometry, 86–90, 101–109
speech spectrum noise, 91
Studebaker acoustic procedure, 104
when to use, 82–90
white noise, 91
Masking generator, 20
Maternal and Child Health Bureau (MCHB), 443
Matrilineal inheritance, 873
Maximum-length sequence (MLS) technique, 224
M-CHAT. *See* Modified Checklist for Autism in Toddlers (M-CHAT)
MCHB. *See* Maternal and Child Health Bureau (MCHB)
MCL. *See* Most comfortable level (MCL)
MCT. *See* Motor control test (MCT)
MDHCF. *See* Marion Downs Hearing Center (MDHCF)
Meaningful Auditory Integration Scale (MAIS), 826
MED-EL device, 819, 820f, 821, 823
MED-EL's FLEX^{eas} electrode, 823
Medial geniculate bodies (MGB), 517
Medial geniculate nucleus (MGN), 315
Medial longitudinal fasciculus (MLF), 388
Medial ossicular fixation, 139t
Medial superior olive (MSO), 515
Medial vestibulospinal tract (MVST), 386
vestibulospinal reflex and, 392
Medical comorbidities and hearing loss, 631–632
Medical otolaryngologic evaluation, referral for, 131
MEE. *See* Middle-ear effusion (MEE)
MEG. *See* Magnetoencephalography (MEG)
Meiosis, 873
Melnick–Fraser syndrome. *See* Branchio-oto-renal syndrome (BOR)
Membranous labyrinth, 382f, 385, 387f, 396f
Memory impairment and hearing impairment, 631
MEMS. *See* Microelectrical-mechanical system (MEMS)
Mendelian inheritance, 480–481, 873. *See also* Human genetics
autosomal dominant inheritance, 481, 481f
autosomal recessive inheritance, 481–482, 482f
sex-linked inheritance, 482
Ménière disease, 42–43, 114, 207, 487
ECochG potentials in, 218–219
SP/CAP ratio in, 218
MERRF. *See* Mitochondrial encephalomyopathy with ragged red fibers (MERRF)
Messenger RNA (mRNA), 873
MFT. *See* Multifrequency, multicomponent tympanometry (MFT)
MGB. *See* Medial geniculate bodies (MGB)
MHL. *See* Mixed hearing loss (MHL)
MIC. *See* Microphone-in-concha (MIC)
Microelectrical-mechanical system (MEMS), 706
Microorganisms, in hearing aids and earmolds, 728
Microphone, 12
in hearing aid, 706
and receiver, improper mounting of, 752
Microphone-in-concha (MIC), 705, 705f
Middle ear, 382
Middle-ear dysfunction, TEOAE and, 375f
Middle-ear effusion (MEE), 139t, 143
Middle-ear infection. *See* Otitis media
Middle ear muscle reflexes (MEMR), 165. *See also* Acoustic stapedius reflex (ASR)
Middle-ear pathologies, effects of, 139t
Middle-ear tinnitus, 647, 648
Middle-latency auditory-evoked potentials (MLAEPs), 315
age effects, 324–325
analysis strategy, 320–322
anatomic frame of reference, 315, 316f
bandwidth, 319
clinical use, 327–329
in cochlear implant evaluation, 328–329
gender effects, 324–325
handedness effect, 325
hearing loss on, effects of, 327
in neurologic disease, 324
Pa, 322–323
Pb, 323–324
in psychiatric research, 329–330
recording, 318–320
reference electrode, 320
site-of-lesion testing, 327–328
and speech-evoked ABR, 325
state variables, 325
stimulus considerations, 325–327
subject variables, 324–325
threshold estimation, 327
in tinnitus, 324–325
TP41, 324
waveform, 319, 319f, 320, 321f
Middle latency response (MLR), 189
Mine Safety and Health Administration (MSHA), 600, 887
Minimal Auditory Capabilities (MAC) battery, 71
Minimal response levels (MRLs), 371, 469
Minimal test battery (MTB), 547–548
Mini-Mental Status Evaluation (MMSE), 640
Minimum Speech Test Battery (MSTB), 826
Mismatch negativity (MMN), 189, 339, 346–347, 346f, 539
Missense mutation, 488, 873. *See also* Human genetics
defined, 480
Mitochondria, 873
Mitochondrial DNA (mtDNA), 873
Mitochondrial encephalomyopathy with ragged red fibers (MERRF), 492–493
Mitosis, 873
Mixed hearing loss (MHL), 49
Mixed presbycusis, characteristic of, 634
MLAEPs. *See* Middle-latency auditory-evoked potentials (MLAEPs)
MLNT. *See* Multisyllabic Lexical Neighborhood Test (MLNT)
MLR. *See* Middle latency response (MLR)
MMN. *See* Mismatch negativity (MMN)
MMSE. *See* Mini-Mental Status Evaluation (MMSE)
MoCA. *See* Montreal Cognitive Assessment (MoCA)
Modified Checklist for Autism in Toddlers (M-CHAT), 586
Modified Rhyme Test (MRT), 67
Modifier genes, 873
Moisture effects on hearing aid, 728–729. *See also* Hearing aids
Monaural separation/closure (MSC), 546
Monaural tasks, 72
Monitoring meter, 19–20
Monogenic inheritance, 483, 873
Monomer/ossicular discontinuity, 139t
Monosomy, 873
Monosyllabic words, for speech recognition in quiet, 65–66
CID W-22, 65–66
NU No. 6, 66
PB-50, 65
Montreal Cognitive Assessment (MoCA), 640
Mosaicism, 873
Most comfortable level (MCL), 790
Motor control test (MCT), 420–422
Motor unit, 300–301, 301f
Motor unit action potential (MUAP), 300–301
MPS. *See* Multiple Pulsatile Sampler (MPS)
MRI. *See* Magnetic resonance imaging (MRI)
MRLs. *See* Minimal response levels (MRLs)
MSC. *See* Magnitude-squared coherence (MSC); Monaural separation/closure (MSC)
MSHA. *See* Mine Safety and Health Administration (MSHA)
MSO. *See* Medial superior olive (MSO)
MSTB. *See* Minimum Speech Test Battery (MSTB)
MTB. *See* Minimal test battery (MTB)
MUAP. *See* Motor unit action potential (MUAP)
Multifactorial inheritance, 483–484, 873. *See also* Human genetics
Multifrequency, multicomponent tympanometry (MFT), 149–150, 149f, 150f
high resonant frequency, 153–154
low resonant frequency, 152–153, 153f, 154f
resonant frequency, 151–152, 152f
Vanhuyse model, 150–151, 150f, 151f
Multimeter, 12
Multiple disabilities, individuals with, 583–584, 583t
autism spectrum disorder, 585–586
management considerations, 586–587
testing considerations, 586
customizing technology management, 585
hearing assessment, 584–585
intellectual disability, 588–589
management considerations, 590
testing considerations, 589–590
physical disabilities
management considerations, 588
testing considerations, 587–588
visual impairment, 590–591
management considerations, 591–592
testing considerations, 591
Multiple Pulsatile Sampler (MPS), 821
Multiple sclerosis (MS), ABR in, 241–242
Multisyllabic Lexical Neighborhood Test (MLNT), 474, 826
Multitalker babble, 686
Music use, in tinnitus, 651

Mutation, 873
Myoclonus, defined, 492

N400, 348–349
NAL. *See* The National Acoustic Laboratories (NAL) formula
NAL-NL2, for hearing aid verification, 796–797
Narrowband noise, 91
The National Acoustic Laboratories (NAL) formula, 766
National Center for Hearing Assessment and Management, 442
National Early Childhood Assessment Project, 845
National Health and Nutrition Examination Survey (NHANES), 631
National health insurance program, 826
National Institute for Occupational Safety and Health (NIOSH), 596, 605, 887
National Institutes of health (NIH), 459
National Institutes of Health Consensus Development Panel, 437
NBS 9-A coupler, 17
NCCs. *See* Noise criteria curves (NCCs)
NCHAM. *See* National Center for Hearing Assessment and Management
Near-field magnetic induction (NFMI), 722
  digital wireless hearing instrument system, 722f
Netherlands, screening service in, 666
Neural generators, of ASSR, 268–269
  in humans, 269
Neural response imaging (NRI), 829
Neural response telemetry (NRT), 829
Neural synchrony
  age-related changes in, 633
  importance of
    behavioral evidence, 521
    geographically remote brain regions, 520–521
    neural synchrony, 519–520
    stimulus representation, 518–519
Neuronal age-related atrophy, characteristic of, 634
Newborn hearing screening
  communicating with parents and healthcare providers, 452–453
  data management and tracking, 454–455
  factors contributing to expansion of, 441–443
    federal support, 442
    legislation, 443
    policy initiatives, 442
    professional and advocacy groups, 443
    successful implementation, 442–443
    technologic advances, 443
  federal privacy protection laws, 453–454
  global status of, 439–441
  operating, 450
    ensuring competent screeners, 452
    responsibility and leadership, 451–452
    selecting screening equipment and protocols, 450–451
    standard of care, 450
  otoacoustic emissions and, 370–371
Newborns, tympanometry in, 148–149, 148t, 149f
New York State business law, 809
NFMI. *See* Near-field magnetic induction (NFMI)
NHANES. *See* National Health and Nutrition Examination Survey (NHANES)
NHANES III. *See* Third National Health and Nutrition Examination Survey (NHANES III)
nHL. *See* Normal hearing level (nHL)
NIH. *See* National Institutes of health (NIH)
NIHL. *See* Noise-induced hearing loss (NIHL)
NIOSH. *See* National Institute for Occupational Safety and Health (NIOSH)
NIPTS. *See* Noise-induced permanent threshold shift (NIPTS)
NITTS. *See* Noise-induced temporary threshold shift (NITTS)
Noise
  defined, 595
  historical evidence of auditory damage from, 596
  and its effects on ear, 602–604
  music as, 605–606, 605t
  and reverberation, effects of, 678
Noise criteria curves (NCCs), 676
Noise exposure
  equal energy hypothesis, 599–600
  and hearing loss, 636
  history of, 595
  measurement of, 595–596
  models, historical development of, 598–600, 599t
  noise standards, 886–888
  nonauditory effects, 604–605, 885–888
    cardiovascular effects, 885–886
    on fetal development, 886
    on learning, 886
    on sleep, 885
  reduction of occupational, 601–602, 601f, 602f
  time-weighted average limit for, 886–888
Noise-induced hearing loss (NIHL), 43, 595
  from long-term exposure, 604
  pathophysiology of, 596–597, 597f
Noise-induced permanent threshold shift (NIPTS), 596
Noise-induced temporary threshold shift (NITTS), 596
Noise reduction rating (NRR), 606
Noise standards, 600–602, 886–888
Nonadaptive hearing aid characteristics, acoustic stimuli in assessment, 739–742
Non-Mendelian inheritance, 482. *See also* Human genetics
  mitochondrial inheritance, 482–483
  modifier genes, 483
  polygenic inheritance, 483
Nonorganic hearing loss
  in adults, 617–618
  in children, 618–619
  controversial issues, 628
  counseling nonorganic patients, 627
  indications of
    audiometric configuration, 620
    nontest situation, 619
    shadow curve, 620
    test–retest reliability, 620
    test situation, 619
  obsolete procedures, 626
  qualitative tests for
    acoustic immittance measurements, 621–622
    ascending–descending methods, 623
    low-level speech recognition testing, 624, 624t
    modified Stenger test, 622–623
    pulse-count methods, 624–625
    Stenger test, 622
    swinging story test, 623–624
    yes–no test, 625
  quantitative tests for
    auditory-evoked potentials, 625
    otoacoustic emissions, 625
    puretone delayed auditory feedback, 625–626
  special tests for, 621
  test sequence, 626
  tinnitus, 626–627
Nonsense mutation, 480, 873
Nonsense Syllable Test (NST), 67
Nonsyndromic, 873
Normal hearing level (nHL), 464
Northwestern University Auditory Test Number 6 (NU No. 6), 66
Northwestern University Children's Perception of Speech (NU-CHIPS), 71, 473
Notch filter, 195, 195f
NRI. *See* Neural response imaging (NRI)
NRR. *See* Noise reduction rating (NRR)
NRT. *See* Neural response telemetry (NRT)
NU-CHIPS. *See* Northwestern University-Children's Perception of Speech (NU-CHIPS)
Nucleotide, 873
Nyquist rate, 193
Nystagmus, 407
  headshake, 404
  hyperventilation-induced, 405
  optokinetic, 389–390
  recovery, 395, 395f
  right-beating, 408f
  Valsalva-induced, 405

OAEs. *See* Otoacoustic emissions (OAEs)
Obligate carriers, 873
Occluded ear simulators (OES), 732
Occlusion effect, 41, 50, 51, 57
Occupational Safety and Health Administration (OSHA), 9, 508, 600, 861, 886–888
Ocular motility, 402–403
Ocular motility test, 408–409
  gaze stability test, 408
  optokinetic nystagmus test, 409
  saccade test, 409
  smooth pursuit test, 408–409, 408f
Oculomotor control systems, 389–390
Oddball paradigm, for study of ERPs, 189
OES. *See* Occluded ear simulators (OES)
Off-vertical axis rotations (OVAR), 417
OHCs. *See* Outer hair cells (OHCs)
OKN. *See* Optokinetic nystagmus (OKN)
Older adults, hearing loss and medical comorbidities in, 631
Older population, demographies of, 631

OME. *See* Otitis media with effusion (OME)
Ontario Infant Hearing Program (OIHP), 260
Ootoacoustic emissions (OAE), 633, 666
Open-fit behind-the-ear hearing aids, 751
Open-fit hearing aids, 732
Open-jaw impressions, 750
Optokinetic nystagmus (OKN), 389–390
Optokinetic nystagmus (OKN) test, 409
Organ of Corti, histopathologic changes in, 633
Oscilloscope, 13
OSHA. *See* Occupational Safety and Health Administration (OSHA)
OSPL90. *See* Output sound pressure level with a 90-dB input (OSPL90)
Otitis media, 43
Otitis media with effusion (OME), 137–138
Otoacoustic emissions (OAEs), 119, 120, 121t, 439, 464, 485, 590
  case study, 372
  classification, 359
  click-evoked
    example, 360f
    growth function, 362f
    levels, 362
    waveform, 360–361
  clinical applications, 364–372
  as clinical tests, 364–368
  defined, 357
  devices, 25
  differential diagnosis of, 370
  distortion product
    characteristics, 363–364
    clinical applications, 367–368
    cumulative distributions, 368f
    diagnose hearing loss, 371–372
    frequency separation, 363
    graph, 363f
    hearing status prediction, 372
    input/output function, 372
    level as function of frequency, 371f
    levels, 363–364, 367, 367f, 368–369, 369f, 371
    measurement, 362–363, 375f
    patient characteristics, 364
    "primaries," 362
    ROC curves, 367–368
    spectrum, 362f
    TEOAEs *vs.,* 364
  evoked, 359
    levels, 365
  generation
    hypotheses, 357–359
    mechanisms, 358–359
  in hearing loss identification, 368–370
  high-frequency stimuli, 372
  interpretation of, 368–371
  measurement, 359
  newborn hearing screening, 370–371
  outer hair cells and, 357–358
  range for normally hearing ears and, 370
  role in cochlear amplifier, 357–358
  spontaneous, 359–360
    example, 359f
    measurement, 359–360, 359f
    TEOAEs and, 364
    tinnitus and, 360
  stimulus-frequency, 360
  templates, 368–369
  toneburst-evoked
    examples, 361f
    frequency, 361
  transient-evoked, 360–361
    audiogram, 373f, 374f
    broadband, 365f, 366, 366f
    case study, 373
    characteristics, 361–362
    clinical applications, 366–367
    defined, 360
    DPOAEs *vs.,* 364
    evaluation, 361
    "frequency dispersion," 361
    frequency distributions, 365f
    level, 362, 365, 365f
    measurement, 375f
    middle-ear dysfunction and, 375f
    parameters, 366
    patient characteristics, 364
    ROC curves, 365–366, 365f, 366f
    SNR criteria, 369
    SOAEs and, 364
    spectra, 361–362
  types of, 359–364
OTOF gene mutations, 223
Otolith function test, 417–418
Otolith membrane, 385
Otolith organs, 381–383, 383f
Otologic disorders, 173
  auditory neuropathy spectrum disorder, 179–180
  central nervous system disorders, 177–181
  conductive hearing loss, 176–177
  demyelinating and cortical disorders, 181
  extra-axial brainstem disorders, 180
  facial nerve paralysis, 175–176, 176f
  functional hearing loss, 183
  hearing loss of cochlear origin, 173–175, 174f, 175f
  intra-axial brainstem disorders, 180–181
  retrocochlear and brainstem disorders, 177–181
  superior canal dehiscence, 181–182
  vestibular schwannoma, 177–179
Otosclerosis (OTSC), 489
  and bone-conduction thresholds, 40–41
Ototoxicity, 43
OTSC. *See* Otosclerosis (OTSC)
Outer ear, 382
Outer hair cells (OHCs)
  motility, 357
  OAEs and, 357–358
Output sound pressure level with a 90-dB input (OSPL90), 736
  curve, open vent on, 738f
OVAR. *See* Off-vertical axis rotations (OVAR)

P300, 347–348, 347f
P600, 348–349
Pain hyperacusis, 654
Paramedian pontine reticular formation (PPRF), 289
PARC. *See* Placement and Readiness Checklists (PARC)
p arm, 873
PASC. *See* Pediatric Audiology Specialty Certification (PASC)
Pathognomonic, 873
Patient characteristics, in audiology, 662–663
Patient Health Questionnaire (PHQ), 640
Patient Protection and Affordable Care Act, 849
Patrilineal inheritance, 873
Patulous ET, 145
PDD. *See* Pervasive developmental disorder (PDD)
PDDST-II. *See* Pervasive Developmental Disorder Screening Test II (PDDST-II)
PEA. *See* Phonemic error analysis (PEA)
Pediatric amplification
  clinical pathway for, 759f
  guideline, 759
Pediatric audiology
  considerations for, 662
  otoscopic inspection, 463
  patient history, 463
Pediatric Audiology Specialty Certification (PASC), 447, 510
Pediatric cochlear implant users, speech perception skills evaluation, 825–827, 825t
Pediatric earhooks, design of, 762
Pediatric *vs.* adult hearing aid fitting, 759–762. *See also* Hearing aids
Pedigree, 873
Pendred syndrome, 493
Penetrance, 873
Perilymph, 381
Periodicity, defined, 530
Peripheral hypothesis, for speech recognition difficulty, 637
Peripheral vestibular system, 381–386
  ampulla, 384
  arterial blood supply, 385–386
  bony labyrinth, 381, 382f
  cranial nerve VIII, 385
  cristae ampullaris, 383f, 384–385
  cupula, 384–385
  endolymph, 381, 384–385
  hair cell structure, 383–384, 384f
  maculae, 383, 384–385, 386f
  membranous labyrinth, 382f, 385, 387f
  otolith organs, 381–383, 383f
  perilymph, 381
  semicircular canals, 381, 383f
  vestibular receptors, 386
Periphery, age-related changes in, 632
Permanent hearing loss, 598
Permanent threshold shifts (PTS), 43
Permissible exposure limit (PEL) for noise, 886
Personal frequency modulation amplification, 682–684, 682–684f. *See also* Room acoustics and auditory rehabilitation technology
Personal sound amplification products (PSAPs), 705
Pervasive developmental disorder (PDD), 585
Pervasive Developmental Disorder Screening Test II (PDDST-II), 586
Phase coherence
  defined, 276
  examples, 278
  measures, 276–277

PHCP. *See* Primary healthcare provider (PHCP)
Phenocopy, 873
Phenotype, 480, 873
Phonak iSense, 684
Phoneme recognition tests, 67
Phonemic error analysis (PEA), 562
Phonemic synthesis (PS), 550, 564–566. *See also* Central auditory processing disorder (CAPD)
  benefits and disadvantages of recorded, 564–565
  branching strategies, 565, 566f
  decoding therapy outcomes, 566, 566f
  program and basic approach, 565
Phonemic Training Program (PTP), 562f
  branching strategies, 564
  principles, 562–563
  purpose of, 562
  steps, 563–564
Phonetically Balanced 50 (PB-50), 65
PHQ. *See* Patient Health Questionnaire (PHQ)
Physical disabilities
  management considerations, 588
  testing considerations, 587–588
Pistonphones, 13
Placement and Readiness Checklists (PARC), 838
Plateau masking procedure, 95–99
Platinum sound body-worn processor, 821
Pleiotropy, 483, 873
P1-N1-P2 complex, 340–343
Point mutation, 480, 873
Point-optimized variance ratio (POVR), 257
Polygenic inheritance, 483, 873
Polymorphism, 873
Polyploidy, 484, 874
Portable assistive listening device, 690f
Positional nystagmus, 410–411
Postauricular hearing aids, 732
Posterior vestibular artery, 385
Postlingually deafened adults, rehabilitation of, 830
PPRF. *See* Paramedian pontine reticular formation (PPRF)
Prelingually deafened adults, rehabilitation of, 830
Presbycusis, 42, 632
Pre-syncope, 400
Primary healthcare provider (PHCP), 444
Proband, 874
Probe effect, 175
Protein, 874
PS. *See* Phonemic Synthesis (PS)
PSAPs. *See* Personal sound amplification products (PSAPs)
Pseudohypacusis, 41
PseudoSNHL, 53
Psychologic evaluation, for pediatric patients, 826. *See also* Cochlear implants (CIs)
Psychosocial consequences, of hearing loss, 638–639
PTA. *See* Puretone average (PTA)
PTP. *See* Phonemic Training Program (PTP)
Public health screening programs, 438–439
Puretone (PT), 29
  amplitude, 29
  EM level for, 91
  evaluation, 29–47
  frequency, 29
Puretone air- and bone-conduction testing, 641
Puretone audiometers, 14–16, 32
  basic signal, 14
  biologic check, 14–16, 15f
  frequency check, 16
  harmonic distortion check, 16
  linearity check, 16
  rise–fall time, 16
Puretone audiometry, 29, 31
  acoustic trauma and, 43
  acoustic tumors and, 42
  aging and, 42, 42f
  audiometric interpretation, 37–39
  auditory neuropathy and, 41
  automated audiometry, 44–45
  calibration, 45
  cross-checks for, 120
  equipment
    audiometers, 32
    transducers, 32–34
  Ménière's disease and, 42–43
  noise-induced hearing loss and, 43
  otitis media and, 43
  ototoxicity and, 43
  pseudohypacusis and, 41
  special populations, 41–44
  test environment, 34–35, 35t
  testing limitations
    bone-conduction thresholds, 40–41
    test–retest reliability, 39–40
    vibrotactile thresholds, 40, 40f
  tinnitus and, 41
  tympanic membrane perforations and, 43–44, 44f
Puretone audiometry screening, 667. *See also* Teleaudiology
Puretone average (PTA), 120, 121t, 371
  and speech recognition threshold, 120
Puretone hearing sensitivity, decline of, 636
Puretone thresholds, 29
  and audiologic test battery, 45
  in dB sound pressure level and dB hearing level, 29, 30f
  measurements, 35
    age 5 years to adult, 35–37
    audiometric procedure for, 36–37
    children younger than age 5, 37
    persons with special needs, 37
    psychophysical procedure in, 35
  and speech measures, relation between, 44
  tuning fork tests, 30–31
  use of, reasons for, 30

q arm, 874
Quantization, 193
Quantization error, 193
Questionnaires, hearing, 116
Quick Speech-in-Noise Test (QuickSIN), 69, 70, 131

Race, influence on hearing status, 636–637
RCC. *See* Relay Conference Captioning (RCC)
Read My Quips, 855
Real ear aided response (REAR), 770
Real ear insertion gain (REIG), 770, 783
Real-ear measures (REMs), 711
Real ear method, 16–17
Real-ear-to-coupler difference (RECD), 590, 736, 768
Real ear to dial difference (REDD), 768, 781
Real ear unaided gain (REUG), 768
Real ear–unaided response (REUR), 598
Real-time telehealth, 660. *See also* Information and communication technologies (ICTs)
  technologies facilitating, 660t
REAR. *See* Real ear aided response (REAR)
RECD. *See* Real-ear-to-coupler difference (RECD)
Receiver-in-the-canal (RIC), 703
Receiver-in-the-ear (RIE), 703, 704, 704f
Recommendations
  and counseling, 889–890
  vestibular dysfunction and, 431
Recommended exposure limit (REL), 600
Recovery nystagmus, 395, 395f
REDD. *See* Real ear to dial difference (REDD)
Reference equivalent threshold sound pressure level (RETSPL), 767
Reference-free derivations, 320
Rehabilitation
  audiologic, 849–851, 850t
    candidacy types, 852t
    counseling, 851–853
    future perspectives of, 858–859
    options for therapeutic AR intervention, 854–858
  aural, 696
  internet-based, 669–670. *See also* Teleaudiology
  postlingually deafened adults, 830
  prelingually deafened adults, 830
  room acoustics and auditory. *See* Room acoustics and auditory rehabilitation technology
REIG. *See* Real ear insertion gain (REIG)
REL. *See* Recommended exposure limit (REL)
Related services, 875
Relative operating characteristic (ROC) curves
  DPOAEs, 367–368
  TEOAEs, 365–366, 365f, 366f
Relay Conference Captioning (RCC), 695
Remote audiometry test transmisson, 661f
Remote technology, 310
Remote wireless microphone technology, 764–765
REMs. *See* Real-ear measures (REMs)
Response to Intervention (RTI), 506
Retinitis pigmentosa, 874
Retrocochlear and brainstem disorders, and ASR threshold, 177–181
RETSPL. *See* Reference equivalent threshold sound pressure level (RETSPL)
Rett's syndrome, 585
REUG. *See* Real ear unaided gain (REUG)
REUR. *See* Real ear–unaided response (REUR)
Reverberation, 677–678
Reverberation time (RT), 680
Rhode Island Hearing Assessment Project, 442
Ribonucleic acid (RNA), 874
Ribosomal RNA (rRNA), 874

Ribosome, 874
RIC. *See* Receiver-in-the-canal (RIC)
RIE. *See* Receiver-in-the-ear (RIE)
Right-beating nystagmus, 408f
Rinne tuning fork test, 31, 50
ROC curves. *See* Relative operating characteristic (ROC) curves
Rollover effect, 72
Romberg test, 404
Room acoustics and auditory rehabilitation technology
  acoustic modifications of room, 680
  alerting systems, 696
  classroom acoustic standard, status of, 680–681
  communication deficits and needs, assessment of, 696–698
  communication in room settings, improvment of
    background room noise, 675–677
    distance, 678–680
    noise and reverberation, effects of, 678
    reverberation, 677–678
  communication strategies, 691
  deaf and hard of hearing, technologies with application, 694–696
  future research issues, 700
  hearing aid technology, limitations of
    advances in digital transmission technology, 690
    classroom audio distribution systems, 685–688
    electromagnetic induction loop systems, 688–690
    hardwired systems, 690
    infrared light wave systems, 684–685
    personal frequency modulation amplification, 682–684
  hearing assistance technology
    legislation for, 699
    research for, 699–700
  individuals with hearing loss/auditory processing deficits, technologies, 691–694
  outcomes measures, 698
  personal and group amplification systems, 681
  rehabilitation technology in audiology setting, 698–699
Room amplification systems, goals of, 681
Rotational chair test, 413–414
  generic, 414f
  normal, 415f
RT. *See* Reverberation time (RT)
RTI. *See* Response to Intervention (RTI)

SAC-A. *See* Self-Assessment of Communication—Adolescents (SAC-A)
Saccades, 389
Saccade test, 409
Saccular evaluation, 418–419
Saccule. *See* Otolith organs
SAT. *See* Speech awareness threshold (SAT)
SBA. *See* Small Business Association (SBA)
SCM muscles. *See* Sternocleidomastoid (SCM) muscles
Scores on the Hearing Handicap Inventory for the Elderly, 636
Screening for Otologic Functional Impairments (SOFI), 640t
Screening Instrument for Targeting Educational Risk (SIFTER), 553, 581
SDT. *See* Speech detection threshold (SDT)
Seasonal and climate problems, of hearing aids, 747, 747t
Section 504 of the Rehabilitation Act of 1973, 836
Segregation, 874
Self-advocacy competency checklist, 504, 882–884
  for adult, 884
  for elementary school, 882
  for high school, 883
  for middle school, 882–883
Self-Assessment of Communication—Adolescents (SAC-A), 504
Self-assessment questionnaires, for hearing aid benefit, 783
Semicircular canal dehiscence (SCD), 159f
Semicircular canals (SCC), 381, 383f
  anterior, 381, 385
  cupula in, 385
  endolymph in, 381
  extraocular muscle and, 388t
  horizontal VOR and, 390
  lateral, 381
  orientation of, 383f
  posterior, 381, 385
Sennheiser model HDA200 earphone, 33
Sensory/neural acuity level (SAL) technique, 284
Sensory/neural hearing loss (SNHL), 49, 675. *See also* Bone conduction evaluation
  in infant, 373–374
Sensory/neural tinnitus, 647
Sensory organization test (SOT), 419–420
  abnormalities of, 421t
  conditions for, 420f
  quantitative equilibrium score and, 420
Sentence-level tests, 66–67
Service animals, for people with hearing loss, 696
Sex-linked inheritance, 874
SFOAEs. *See* Stimulus-frequency otoacoustic emissions (SFOAEs)
SHARP. *See* Situational Hearing Aid Response Profile (SHARP)
Shell-related occlusion, 748–749
Short term auditory memory (STAM), 561
Short-term auditory memory program (STAMP), 568–570
  procedures of, 569–570, 570f
SIFTER. *See* Screening Instrument for Targeting Educational Risk (SIFTER)
Signal-to-noise ratio (SNR), 61, 196, 639, 676
  improvement of, 715–716
  TEOAE, 369
Significant Other Assessment of Communication—Adolescents (SOAC-A), 504
SII. *See* Speech Intelligibility Index (SII)
Simultaneous communication approach, 840
SIN. *See* Speech-in-Noise (SIN)
Single-sided deafness (SSD), 817
Sinusoidal harmonic acceleration (SHA) test, 414–416, 415f
  gain, 415–416
  parameters, 415f
  phase, 416
  symmetry, 416
Sinusoidally amplitude modulated (SAM) waveform, 278
SIRS. *See* Speech Intelligibility Rating Scale (SIRS)
Situational Hearing Aid Response Profile (SHARP), 761
Sixty-cycle interference, 298
Sleep therapy, for tinnitus, 652
SLM. *See* Sound level meter (SLM)
Slow-component eye velocity (SCV), 408f
Slow vertex potential (SVP), 189
Small Business Association (SBA), 808
Smoking and hearing loss, 631
Smooth pursuit test, 408–409, 408f
Snellen Visual Acuity Eye Chart, 640
SNHL. *See* Sensory/neural hearing loss (SNHL)
SNR. *See* Signal-to-noise ratio (SNR)
SOAC-A. *See* Significant Other Assessment of Communication—Adolescents (SOAC-A)
SOAEs. *See* Spontaneous otoacoustic emissions (SOAEs)
SOAP format, 116–117
Social-emotional development, in children, 846
Social isolation
  and hearing impairment, 638, 639
  and hearing loss, 631–632
SOFI. *See* Screening for Otologic Functional Impairments (SOFI)
SOT. *See* Sensory organization test (SOT)
Sound-activated systems, 696
Sound-field frequency modulation amplification system, cost effectiveness of, 687t, 688
Sound field testing, 20
Sound-field thresholds, 33–34
Sound level meter (SLM), 12–13, 505, 675
Sound pressure level (SPL), 29, 465, 649, 676, 730
  reference level for dB SPL, 29
Sound therapy
  for hyperacusis, 655–656
  for tinnitus, 651–652
SP. *See* Summating potential (SP)
SPA. *See* Special-purpose average (SPA)
Spatial hearing, 521–523, 523f
Speakers, 33–34
Spearman–Kärber equation, 64
Special-purpose average (SPA), 736
Spectral analysis, for ASSR detection, 277–278
Spectrum analyzer, 13–14
Speech, acoustic features of
  acoustic onsets, 537–539
  formant structure, 532–535
  frequency transitions, 535–537
  periodicity, 530–532
    in human brain, 530–532, 531f
    perception of speech, 530
  speech envelope, 539–540
Speech audiometers, 19

Speech audiometry, 61, 667–668. *See also* Teleaudiology
calibration, 61
clinical functions of speech recognition measures, 71–72
considerations for
in children, 70–71
in nonverbal patients, 71
in profoundly hearing-impaired adults, 71
general considerations, 61–63
masking during, 86–90, 101–109
presentation methods, 62
psychometric function and performance, 62–63, 62f, 63f
recorded speech for SRSs, 62
response mode for, 63
speech recognition in noise, 67–70
informational masking, 70
materials, 69–70, 70f
speech recognition in quiet, 65–67
materials for, 65–67
monosyllabic words for, 65–66
nonsense syllables for, 67
purpose of, 65
sentences for, 66–67
speech recognition threshold, 63
clinical functions of, 64–65
stimuli, 63, 64t
testing protocol, 63–64
terminology related to, 61
Speech awareness threshold (SAT), 61, 371. *See also* Speech detection threshold (SDT)
Speech detection threshold (SDT), 61, 81, 472, 825. *See also* Speech audiometry
Speech-evoked ABR (sABR), MLAEPs and, 325
Speech-in-noise (SIN), 715
Speech-in-noise test, 120, 121t
Speech Intelligibility Index (SII), 779
Speech Intelligibility Rating Scale (SIRS), 845
Speech-language evaluations, for pediatric patients, 826. *See also* Cochlear implants (CIs)
Speech Pattern Contrast (SPAC) test, 67
Speech perception, in adults, 678
Speech Perception in Noise (SPIN) test, 69
Speech perception skills evaluation, 825–826, 825t
Speech perception testing and hearing aid evaluation, 825–827. *See also* Cochlear implants (CIs)
Speechreading, 855
Speech reception threshold (SRT), 44, 472, 618
and puretone average disagreement, 620–621
Speech recognition
in noise, 67–70
informational masking, 70
materials, 69–70, 70f
in quiet, 65–67
materials for, 65–67
monosyllabic words for, 65–66
nonsense syllables for, 67
purpose of, 65
sentences for, 66–67
tests for children, 71
threshold. *See* Speech recognition threshold (SRT)
Speech recognition score (SRS), 61. *See also* Speech audiometry
Speech recognition threshold (SRT), 61, 63, 120, 121t, 825. *See also* Speech audiometry
clinical functions of, 64–65
interaural attenuation and, 81
measurement, 63
calculation of threshold, 64
determination of threshold, 64
familiarization, 64
instructions, 64
spondaic words, use of, 63, 64t
stimuli, 63, 64t
testing protocol, 63–64
use of, reasons for, 64–65
Speech signal
formant structure, 528–529
fundamental frequency, 528, 529f
harmonic structure, 528
Speech, Spatial, and Qualities of Hearing Scale (SSQ), 640t
Speech spectrum noise, 91
Speech thresholds and recognition, testing, 472–474
Speech understanding difficulty, in elderly, 637–638
SPL. *See* Sound pressure level (SPL)
SPLogram, for unaided conversation-level speech, 761, 761f
Spoken language skills, for D/HH children, 841
Spondaic words, 63, 64t
Spontaneous otoacoustic emissions (SOAEs), 359–360
example, 359f
measurement, 359–360, 359f
TEOAEs and, 364
tinnitus and, 360
SRS. *See* Speech recognition score (SRS)
SRT. *See* Speech reception threshold (SRT); Speech recognition threshold (SRT)
SSD. *See* Single-sided deafness (SSD)
SSQ. *See* Speech, Spatial, and Qualities of Hearing Scale (SSQ)
SSW test. *See* Staggered Spondaic Word (SSW) test
Staggered Spondaic Word (SSW) test, 72, 550
STAM. *See* Short term auditory memory (STAM)
STAMP. *See* Short-term auditory memory program (STAMP)
Stapedius muscle, 165. *See also* Acoustic stapedius reflex (ASR)
Staphylococcus aureus, 753
Start codon, 874
Static admittance (tympanometric measurement), 137, 141f, 142–143
Static positional test, 410–411
Stenger test, 622. *See also* Nonorganic hearing loss
Stereocilia displacement, 384f
Sternocleidomastoid (SCM) muscles, 418
Stickler syndrome, 493–494
Stimulus
auditory brainstem response, 198–204, 252–255
auditory-evoked potentials, 197–198
ECochG recording, 210–211
middle-latency auditory-evoked potentials, 325–327
Stimulus-frequency otoacoustic emissions (SFOAEs), 360
Stop codon, 874
Stopping rules, for ASSR tests, 290
Store-and-forward telehealth, 660. *See also* Information and communication technologies (ICTs)
technologies facilitating, 660t
Strial degeneration, age-related, 633–634
Striola, 385
STS. *See* Superior temporal sulcus (STS)
Subjective visual vertical (SVV), 419
Summating potential (SP), 188, 207
Superior canal dehiscence (SCD), 181–182
Superior temporal sulcus (STS), 534
Supra-aural earphones, 32, 32f, 78
Suprathreshold perceptual testing, in children, 772–773
Suprathreshold word-recognition scores (WRSs), 44
SVV. *See* Subjective visual vertical (SVV)
Synchronous telehealth. *See* Real-time telehealth
Syndromic, 874

TACL-3. *See* Test of Auditory Comprehension of Language (TACL-3)
Tangible-reinforcement operant conditioning audiometry (TROCA), 586
TAPS-R. *See* Test of Auditory Perceptual Skills—Revised (TAPS-R)
TBOAEs. *See* Toneburst-evoked otoacoustic emissions (TBOAEs)
TDD. *See* Telecommunications device for the deaf (TDD)
TEACH. *See* Teachers' Evaluation of Aural/Oral Performance of Children (TEACH)
Teachers' Evaluation of Aural/Oral Performance of Children (TEACH), 841
Teleaudiology, 659. *See also* Room acoustics and auditory rehabilitation technology
applications of
audiologic intervention, 668–670
continued professional education, 670
diagnosis, 666–668
screening, 666
clinician and patient perceptions of, 670–671
future perspectives of, 671
service, considerations for
equipment for telehealth purposes, 661–662
facilitators for, 662
information and communication technologies for, 660–661
patient and environmental considerations, 662–663, 662t, 663t
standards and guidelines for, 663–664
telehealth service
for audiology, 664–666
delivery models, 660
telemedicine, telehealth, and ehealth, 659–660
Telecoils
hearing aids with, 708–709, 754
magnetic interference and, 754–755
real-ear measures for, 693

Telecommunications device for the deaf (TDD), 693f
Telehealth service, 659. *See also* Teleaudiology
for audiology, 664–666
delivery models, 660
illustration of, 660f
equipment for purposes, 661–662
facilitators for, 662
information and communication technologies for, 660–661
patient and environmental considerations, 662–663, 662t, 663t
reimbursement and insurance coverage for, 664
Tele-intervention methods, 670. *See also* Teleaudiology
Telemedicine, 659
Telephone communication, improvement of, 693–694
Telephone relay services (TRSs), 695
Telephones
coin-operated, 754
hearing aid compatible, 694
and hearing aids, 754–755
and hearing aids, 708, 754
modifications to, 755
voice carry over, 755
Teletypewriters (TTYs), 693
Television Decoder Circuitry Act of 1990, 692
Telomeres, 478, 874. *See also* Human genetics
Temporary threshold shift (TTS), 43
Temporomandibular joint (TMJ), 750
dysfunction, 653
Tensor tympani, 165, 652
TEOAE. *See* Transient-evoked otoacoustic emissions (TEOAE)
Test battery approach
audiologic, 119, 465. *See also* Diagnostic audiology
for birth to 6 months of age infants, 463–465
for infants 6 months of age and older, 465–466
puretone thresholds and, 45
central auditory processing and, 546
in children with hearing loss, 462
Test of Auditory Comprehension of Language (TACL-3), 846
Test of Auditory Perceptual Skills-Revised (TAPS-R), 555
Test-retest reliability, 39–40
Text telephones (TTs), 693
TFM. *See* Tolerance-fading memory (TFM)
TFOE. *See* Transfer function of the open ear (TFOE)
Third National Health and Nutrition Examination Survey (NHANES III), 508
Three Interval Forced Choice Test of speech pattern contrast perception (THRIFT), 67
Threshold limit value (TLV), for noise, 886
Thymine, 874
Time-domain signal averaging, 190, 196
Time weighted averages (TWA), 596
Tinnitus, 41
auditory hallucinations and, 648
auditory hallucinations as, 648
in childhood, 653
cognitive behavior therapy, 650
electricity for, 653
evaluation of, 648–649
habituation therapy, 649–650
impact, domains of, 648f
management, hearing aids in, 721–722
middle-ear, 647, 648
middle-latency auditory-evoked potentials, 324–325
music use in, 651
neurophysiological causes, mechanisms, and models, 647
nonorganic hearing loss, 626–627
puretone audiometry and, 41
relative handicap of, 893
retraining therapy, 650
sensory/neural, 647
sleep therapy for, 652
sound therapy for, 651–652
spontaneous otoacoustic emissions and, 360
treatment for, 649–653
Tinnitus Handicap Questionnaire, 649
Tinnitus Primary Activities Questionnaire, 649
TMJ. *See* Temporomandibular joint (TMJ)
Tolerance-fading memory (TFM), 550, 551
Tonal duration, in puretone test, 36
Toneburst-evoked otoacoustic emissions (TBOAEs)
examples, 361f
frequency, 361
Toynbee test, 145
TP41, 324
TPP. *See* Tympanometric peak pressure (TPP)
Traditional BTE hearing aids, 704, 704f
Transcription, 874
Transducers
bone vibrators, 34, 34f
earphones, 32–33, 32f, 33f
speakers, 33–34
Transfer function of the open ear (TFOE), 598
Transfer RNA (tRNA), 874
Transient artifacts, 298
Transient-evoked otoacoustic emissions (TEOAEs), 25, 360–361, 463
audiogram, 373f, 374f
broadband, 365f, 366, 366f
case study, 373
characteristics, 361–362
clinical applications, 366–367
defined, 360
DPOAEs *vs.,* 364
evaluation, 361
"frequency dispersion," 361
frequency distributions, 365f
high-frequency stimuli, 372
level, 362, 365, 365f
measurement, 375f
middle-ear dysfunction and, 375f
parameters, 366
patient characteristics, 364
ROC curves, 365–366, 365f, 366f
SNR criteria, 369
SOAEs and, 364
spectra, 361–362
Translation, 874
Translocation, 874
Traumatic brain injury (TBI), 349
Treacher Collins syndrome, 494
Trisomy, 874
TROCA. *See* Tangible-reinforcement operant conditioning audiometry (TROCA)
TRSs. *See* Telephone relay services (TRSs)
TTs. *See* Text telephones (TTs)
TTYs. *See* Teletypewriters (TTYs)
Tuning fork tests, 30–31
Turner syndrome, 494
causes of, 484
TWA. *See* Time weighted averages (TWA)
Tympanic membrane (TM)
perforations, 43–44, 44f
thinning, 138
Tympanogram, 137
asymmetric, 141
Lidén–Jerger classification scheme, 138f, 140
normal, 137, 138f
problems in middle ear and shape of, 137–138, 138f
Tympanometric compensation, 140
Tympanometric gradient, 143, 144f
Tympanometric peak pressure (TPP), 143–144
Tympanometric width (TW), 143
Tympanometry, 119, 121t, 137–138. *See also* Wideband acoustic immittance (WAI)
ANSI standard, 146–147
development and aging, affects of, 144
equivalent ear canal volume, 140–141, 142f
eustachian tube function tests, 144–145
history of, 138–140
innovations in, 139, 149
middle-ear pathologies and, 139t
multifrequency, multicomponent, 149–150, 149f, 150f
high resonant frequency, 153–154
low resonant frequency, 152–153, 153f, 154f
resonant frequency, 151–152, 152f
Vanhuyse model, 150–151, 150f, 151f
in newborns and infants, 148–149, 148t, 149f
normative values and cutoff criteria, 142t
patulous ET, 145
screening, 147–148
sensitivity and specificity, 146
static-compensated acoustic admittance, 141–142
tympanometric peak pressure, 143–144
tympanometric shape, 140
tympanometric width and gradient, 143, 143f
wideband, 156–157, 156f, 157f
Tympanosclerosis, 139t

UHL. *See* Unilateral hearing loss (UHL)
ULC. *See* Upper limits of comfort (ULC)
Ullrich–Turner syndrome. *See* Turner syndrome
UNHS. *See* Universal newborn hearing screening (UNHS)
Unilateral hearing loss (UHL), 39
children with, 440
screening method, 622
Unilateral peripheral vestibular lesions
acute phase effects, 392
compensation after, 392–395
static compensation after, 392–395, 393f
Unilateral weakness (UW), 411–412

United States
early hearing detection and intervention in, 441–443
newborns percentage screened for hearing loss in, 441f
United States Government Printing Office, 10
United States Preventive Services Task Force, 443
Universal newborn hearing screening (UNHS), 439, 444, 836
University of Oklahoma Closed Response Speech Test, 67
Upper limits of comfort (ULC), 761
Uracil, 874
USAF. *See* US Air Force (USAF)
US Air Force (USAF), 600
Usher syndrome, 494–495, 590–591
classical hearing, vision, and vestibular phenotypes in, 495t
US Preventive Services Task Force, 437
Utricle. *See* Otolith organs
UW. *See* Unilateral weakness (UW)

Valsalva-induced nystagmus, 405
Valsalva test, 145
Variable expressivity, 874
Varying Intensity Story Test (VIST), 623, 624t
Vascular loop syndrome, ABR findings in, 238–239
VCN. *See* Ventral cochlear nucleus (VCN)
VCO. *See* Voice carry over (VCO)
Velocity step test, 416–417
VEMP. *See* Vestibular-evoked myogenic potential (VEMP)
Ventral cochlear nucleus (VCN), 515
Vents, hearing aid, 729–730
Vertigo, 400
Vertigo Symptom Scale (VSS), 402
Vestibular assessment, telehealth for, 668. *See also* Teleaudiology
Vestibular dysfunction
case study, 431–432
clinical findings, 431
diagnosis-based strategies, 429–431, 429t
adaptation, 429–430, 430f
habituation, 430
substitution, 430–431, 431f
history and symptoms, 431
overview, 425
recommendations, 431
status, 425–427
compensated *vs.* noncompensated, 426–427
stabilized *vs.* nonstabilized, 426
treatment, 432
unilateral, 425t
Vestibular evaluation, referral for, 132
Vestibular-evoked myogenic potential (VEMP), 418, 418f
cervical, 396
ocular, 395–396, 418–419
Vestibular function
clinical assessment, 427, 428t
patient reports, 427
subjective handicap
instruments, 429t
scales, 427
Vestibular nerve, 385
Vestibular nuclei (VN), 385, 386
cerebellum and, 386–387
medial, 390–391
vestibulospinal reflex and, 392
Vestibular pathways
central, 390–391
compensation, 392–395
excitatory and inhibitory, 389f
as velocity storage mechanism, 390–391
vestibulocollic, 387
vestibulo-ocular, 387
Vestibular receptors, 386
Vestibular rehabilitative therapy (VRT), 425–432
identification for, 427
for labyrinthine events, 426
outcomes, 432
physiological basis, 427–429
Vestibular schwannomas, 177–179, 233
ABR findings in, patterns of, 234–237
absolute latency delay, 234, 234f
amplitude comparison of waves, 235
interaural latency difference, 235
interear latency comparison, 235
interwave latency delays, 234–235
laterality, 236, 236f
repetition rate shifts, 235–236
Stacked ABR, 236–237
threshold measures, 236
waveform morphology and absence of waves, 235
ASR threshold, 177–179
Vestibular symptoms, 487
Vestibular system
anatomy, 381–387
central, 386–387
cerebellum, 386–387
vestibular nuclei, 386
vestibulocerebellum, 387
lesions of, 392–395
compensation after, 393f
unilateral peripheral, 392–395
overview, 381
peripheral, 381–386
ampulla, 384
arterial blood supply, 385–386
bony labyrinth, 381, 382f
cranial nerve VIII, 385
cristae ampullaris, 383f, 384–385
cupula, 384–385
endolymph, 381, 384–385
hair cell structure, 383–384, 384f
maculae, 383, 384–385, 386f
membranous labyrinth, 382f, 385, 387f
otolith organs, 381–383, 383f
perilymph, 381
semicircular canals, 381, 383f
vestibular receptors, 386
physiology, 381–387
role of, 387–392
extraocular muscles, 388–390, 388t
neural integrator, 390–391
ocular motility, 388–390
oculomotor control systems, 389–390
velocity storage, 390–391
vestibulocollic reflex, 392, 392t
vestibulo-ocular reflex, 390, 390t
vestibulospinal reflex, 391–392
Vestibulocerebellum, 387
Vestibulocollic reflex (VCR), 387f, 392, 392t
vestibular disorders and, 425
Vestibulo-ocular reflex (VOR), 387, 387f, 390
active head rotation and, 413
horizontal, 390, 390t
oculomotor control systems and, 389–390
suppression test, 417
unilateral impairment, 392
vestibular disorders and, 425
Vestibulospinal reflex (VSR), 388, 391–392
vestibular disorders and, 425
Vibrant Soundbridge, 819, 819f
Vibrating ossicular prosthesis (VORP), 819
Vibrotactile responses, 56–57
Vibrotactile thresholds, 40, 40f
Video conferencing, communication through, 694–696
Video conferencing technology, 694. *See also* Room acoustics and auditory rehabilitation technology
Videonystagmography (VNG), 406–413, 811
Video-otoscopy, for telehealth service, 667. *See also* Teleaudiology
VIST. *See* Varying Intensity Story Test (VIST)
Visual-Aural Digit Span Test, 555
Visual communication support, for D/HH children, 840–841
Visual impairment, 590–591
environmental auditory cues, 592
etiologies, 590–591
management considerations, 591–592
testing considerations, 591
Visual inspection, of hearing aids, 727–732
Visually enhanced vestibulo-ocular reflex (VVOR) test, 417
Visually reinforced infant speech discrimination (VRISD), 468
Visual perception training, 856
Visual reinforcement audiometry (VRA), 120, 121t, 465, 829
age considerations and, 468–469
algorithm used in, 470f
in children with down syndrome, 468–469
conditioning in, 469–470
control trials, 471–472
false-positive responses, 589
as function of developmental age, 468
head turn response coupled with, 587, 589
novelty of, 467, 470
procedural guidelines, 470–471
reinforcement duration, 471
reinforcement schedule, 470–471
response shaping, 469
second examiner, 467, 467f
signal trials, 470
single examiner approach, 467
success of, 468, 469
test room arrangement, 467, 467f
thresholds, 471
training completion, 469
trial duration, 470
valid outcomes, ensuring, 471
Visual signal, 855
Visual speech perception, 855–856, 855t
Visual–vestibular interaction, 417

VNG. *See* Videonystagmography (VNG)
VNG goggles, 405, 406f
Vocal development, in children, 844–845
Voice carry over (VCO), 755
Voice onset time (VOT), 537
Voice over Internet protocol (VoIP), 661
VoIP. *See* Voice over Internet protocol (VoIP)
VORP. *See* Vibrating ossicular prosthesis (VORP)
VOT. *See* Voice onset time (VOT)
Vowels
  in children with HL, 845
  distinction of, 533
  formant structure in, 532–534
VRA. *See* Visual reinforcement audiometry (VRA)
VRISD. *See* Visually reinforced infant speech discrimination (VRISD)
VSS. *See* Vertigo Symptom Scale (VSS)

Waardenburg–Shah syndrome, 496
WAI. *See* Wideband acoustic immittance (WAI)
Walking difficulty and hearing impairment, relation of, 632, 636
Walsh-Healey Public Contracts Act, 600, 886
Warbled tones, 34
Washington University questionnaire, 778f, 783
Wax guards, use of, 754
WBN. *See* Well-baby nursery (WBN)
WDRC. *See* Wide dynamic range compression (WDRC)
Wearable ear-level devices, use of, 651
Weber tuning fork test, 31, 50
Weighted averaging, 276
Weighting filters, application of, 597–598, 597f
Welch Allyn Audioscope™, 642
Well-baby nursery (WBN), 370
Western Electric 4 A, 61
White noise, 91
WHO. *See* World Health Organization (WHO)
Whole-nerve action potential (WNAP), 189
Wideband acoustic immittance (WAI), 154–155. *See also* Tympanometry
  ASR threshold measurement by, 183–184
  conductive hearing loss, prediction of, 159
  ear disorders on, effects of, 157–159, 158f
  maturation and aging, effects of, 159–160
  measurements, 155–156
  principles and calibration, 155
  tympanometric, 156–157
Wide dynamic range compression (WDRC), 712
  feedback in circuits of, 751
  signal processing technologies to reduce, 761
Wild type, 874
Wind noise reduction, 718–719
WINT. *See* Words-in-noise training (WINT)
WIN test. *See* Words-in-Noise (WIN) test
WIPI test. *See* Word Intelligibility by Picture Identification (WIPI) test
Within normal limits (WNL), 684
WNAP. *See* Whole-nerve action potential (WNAP)
WNL. *See* Within normal limits (WNL)
Wolfram syndrome, 487
Women's Health Initiative, 638
Word charts, 565, 566f
Word Intelligibility by Picture Identification (WIPI) test, 71, 473
Word recognition
  age-appropriate, 772
  assessment of, 474
  audiologic evaluation, 825
  and degree of hearing loss, 486
  by pediatric cochlear implant users, 474
  scores and acoustic reflex thresholds, 487
  scores for normal hearing individuals, 624t
Words-in-noise (WIN) test, 68, 68f, 70
Words-in-noise training (WINT), 566–568. *See also* Central auditory processing disorder (CAPD)
  description of, 567–568, 568f, 569f
  overview of, 566
Worker training program, 612
Working memory
  assessment of, 573, 574
  auditory, 578
  CAPD therapy for, 575
  deficits in, 638
  definition of, 569
World Health Assembly, 439
World Health Organization (WHO), 438, 659
  hearing screening options recommended by, 439f

Yes–no test, 625

Zephry® Dry & Store, 778
Zinc–air batteries, for hearing aids, 708
Zwislocki coupler, 732